MD GERIATRIC MEDICINE

Consolidated Examination Answer Bank

Gastroenterology · Hepatology · Nutrition · Haematology · Oncology

41 questions in strict mark-wise format

15 marks = 10 textbook points + 2 latest updates | 7 marks = 6 + 1 | 6 marks = 5 + 1

Primary sources: Pathy's Principles and Practice of Geriatric Medicine, 6th ed.

and Essentials of Clinical Geriatrics, 7th ed.

Supplemented with current guidelines and trial evidence, 2023–2026

Prepared for Sudarsan • August 2026

Table of Contents

PART I — GASTROENTEROLOGY — LUMINAL DISEASE

1.	Management of gastro-oesophageal reflux disease	p.4 · 15 Marks
2.	*Helicobacter pylori* — diagnosis and eradication	p.5 · 15 Marks
3.	Upper gastrointestinal bleeding — risk scores and management	p.6 · 15 Marks
4.	Malabsorption — etiopathogenesis, clinical features, diagnostic algorithm and management	p.7 · 15 Marks
5.	Constipation and faecal impaction in the elderly	p.8 · 15 Marks
6.	Bristol Stool Chart; classes of laxatives and their modes of action	p.9 · 7 Marks
7.	Colorectal carcinoma — presentation and staging	p.10 · 15 Marks
8.	Palliative management of malignant bowel obstruction	p.11 · 7 Marks

PART II — HEPATOLOGY

9.	Causes of jaundice in an older person and their management	p.12 · 7 Marks
10.	Drug-induced jaundice	p.12 · 7 Marks
11.	Chronic active hepatitis	p.13 · 6 Marks
12.	Alcoholic hepatitis	p.14 · 7 Marks
13.	Management of newly diagnosed hepatitis B in the elderly	p.14 · 7 Marks
14.	Decompensated chronic liver disease in the elderly, and the OPD approach to chronic liver disease	p.15 · 15 Marks
15.	Management of hepatic encephalopathy	p.16 · 7 Marks
16.	Ascites and refractory ascites	p.17 · 7 Marks
17.	Hepatorenal syndrome	p.18 · 7 Marks

PART III — CLINICAL NUTRITION

18.	Parenteral nutrition	p.19 · 7 Marks
19.	Refeeding syndrome	p.19 · 7 Marks
20.	Inanition	p.20 · 7 Marks

PART IV — ANAEMIA AND TRANSFUSION

21.	Anaemia in the elderly and the evaluation of anaemia	p.22 · 7 Marks
22.	Iron deficiency anaemia — red cell indices and investigation	p.22 · 7 Marks
23.	Megaloblastic anaemia and B_{12} deficiency	p.23 · 15 Marks
24.	Anaemia of chronic disease	p.24 · 7 Marks
25.	Blood transfusion in the older adult — thresholds and hazards	p.25 · 7 Marks

PART V — HAEMATOLOGICAL DISORDERS AND MALIGNANCY

26.	Thrombocytopenia	p.27 · 7 Marks

27.	Disseminated intravascular coagulation	p.27 · 7 Marks
28.	Myelodysplasia	p.28 · 7 Marks
29.	Chronic myeloid leukaemia — clinical features and management	p.29 · 7 Marks
30.	Chronic lymphocytic leukaemia	p.30 · 15 Marks
31.	Myelofibrosis and the myeloproliferative neoplasms	p.31 · 15 Marks
32.	Plasma cell dyscrasias — types, presentation, diagnostic criteria, course and treatment	p.32 · 15 Marks
33.	Multiple myeloma — aetiology, diagnosis and management	p.33 · 15 Marks
34.	Bone marrow transplantation in older patients	p.34 · 7 Marks
PART VI — ONCOLOGY		
35.	Prostatic cancer	p.36 · 7 Marks
36.	Vertebral secondaries with unknown primary — evaluation and treatment	p.37 · 7 Marks
37.	Paraneoplastic syndromes	p.37 · 7 Marks
38.	Tumour lysis syndrome	p.38 · 7 Marks
PART VII — CANCER SCREENING AND IMAGING		
39.	Indications and rationale for cancer screening in the elderly	p.40 · 7 Marks
40.	Screening for breast, colorectal and cervical cancer — population, tests, performance and guidelines	p.40 · 7 Marks
41.	Positron emission tomography	p.41 · 6 Marks
APPENDIX		
★	Rapid Revision Grid — examinable numbers, named trials and guidelines	p.43

Point counts within each answer are exact and must be reproduced as such in the examination. Purple panels contain evidence from 2023 onwards; amber panels flag the specific places where the textbook position has been superseded by current practice.

PART I — GASTROENTEROLOGY — LUMINAL DISEASE

Question 1. Management of gastro-oesophageal reflux disease 15 MARKS

Answer — Textbook Core (10 points)

1. **Definition and mechanism.** GORD exists when reflux of gastric contents causes troublesome symptoms or complications. Mechanisms include **transient lower oesophageal sphincter relaxations (the dominant mechanism)**, a hypotensive LOS, **hiatus hernia**, delayed gastric emptying and impaired oesophageal acid clearance.
2. **Presentation in the elderly — the key examination point.** **Symptoms are milder or atypical but mucosal injury is more severe**: erosive oesophagitis, peptic stricture and Barrett's oesophagus are all commoner, while heartburn is reported less often because of **diminished oesophageal sensitivity**. Presentations include **dysphagia, vomiting, weight loss, anaemia, chest pain, chronic cough, hoarseness and aspiration pneumonia**.
3. **Aggravating factors.** Obesity, hiatus hernia, smoking, alcohol, large or late meals and recumbency; and drugs that reduce LOS pressure or injure the mucosa — **calcium channel blockers, nitrates, anticholinergics, theophylline, benzodiazepines, bisphosphonates, potassium chloride, doxycycline and NSAIDs**. **Reviewing the drug chart is a therapeutic act in the elderly.**
4. **Alarm features requiring endoscopy.** **Dysphagia, odynophagia, weight loss, persistent vomiting, anaemia, gastrointestinal bleeding, and new-onset symptoms in a patient over 60. The threshold for endoscopy must be lower than in the young**, because empirical acid suppression may mask malignancy.
5. **Investigations.** Upper GI endoscopy graded by the **Los Angeles classification (A–D)** with biopsy for Barrett's oesophagus; **ambulatory 24-hour pH or pH-impedance monitoring performed off PPI is the gold standard** for confirming pathological acid exposure in non-erosive disease; **high-resolution manometry** before anti-reflux surgery and to exclude achalasia; and barium swallow for strictures and large hernias.
6. **Lifestyle measures.** Weight reduction, **elevation of the head end of the bed by 6–8 inches**, avoiding meals within 3 hours of lying down, left lateral sleeping position, smoking and alcohol cessation, and avoidance of individual trigger foods. **These carry no drug risk and are especially valuable in the elderly.**
7. **Pharmacological therapy.** **Proton pump inhibitors are the drugs of choice**, given **once daily 30–60 minutes before breakfast for 8 weeks**; they are clearly superior to H_2 receptor antagonists for healing oesophagitis. **No dose reduction is required for age**, but step down to the lowest effective dose or on-demand therapy after healing. Antacids and alginates provide symptomatic relief, an H_2 blocker may be added at night for nocturnal breakthrough, and **prokinetics have no established role**.
8. **Long-term PPI safety in older adults.** A causal relationship is likely for **acute interstitial nephritis, benign fundic gland polyps, enteric infections including *C. difficile*, vitamin B_{12} deficiency and hypomagnesaemia**. Associations with **fracture, pneumonia, dementia and progression of CKD are not supported by strong evidence** and are heavily confounded. Review the indication periodically and deprescribe where possible; the clopidogrel interaction is clinically meaningful essentially only for **omeprazole**.
9. **Complications and their management.** Erosive oesophagitis; **peptic stricture — endoscopic dilatation plus long-term PPI**; bleeding; **Barrett's oesophagus** requiring surveillance endoscopy, with endoscopic eradication therapy for high-grade dysplasia; oesophageal adenocarcinoma; and extra-oesophageal disease (asthma, chronic cough, laryngitis, dental erosion, aspiration).
10. **Refractory disease and surgery.** First confirm adherence and correct dosing timing, then optimise to twice-daily PPI, and re-evaluate with pH-impedance testing to identify **functional heartburn or reflux hypersensitivity** (treated with low-dose tricyclics or SSRIs, used cautiously in the elderly). **Laparoscopic fundoplication gives outcomes and complication rates in patients over 70 comparable to the young**, but long-term medical therapy is usually more cost-effective given remaining life expectancy; **patients with ineffective oesophageal motility are at risk of post-operative dysphagia**. Endoscopic anti-reflux procedures remain investigational.

Latest Updates (2023–2026) — 2 points

11. **AGA Clinical Practice Update on potassium-competitive acid blockers** (*Gastroenterology* 2024;167:1228) — **vonoprazan is superior to PPIs for healing and maintaining healing of severe (LA grade C/D) erosive oesophagitis** and for *H. pylori* eradication, and is now FDA-approved for both erosive and non-erosive GERD. However, the update advises that **P-CABs should not be used first-line for mild (LA grade A/B) oesophagitis, nor for peptic ulcer treatment or prophylaxis**, so their role is targeted rather than universal.
12. **ASGE Clinical Practice Guideline on GERD (2025)** (*Gastrointest Endosc*) — the first ASGE update since 2014. It endorses **PPI as initial therapy with endoscopy reserved for alarm features or refractory symptoms**, supports vonoprazan for severe erosive disease, and positions **endoscopic and surgical anti-reflux therapy only for objectively confirmed, PPI-refractory GERD** proven on pH-impedance testing rather than for symptom-based referral.

Current Practice Update

Textbook: PPIs are the most potent acid suppressants available and are first-line for all grades of GORD.
Current recommendation: Potassium-competitive acid blockers (vonoprazan) are superior for LA grade C/D oesophagitis and for *H. pylori* eradication; PPIs remain first-line for mild disease.
Exam approach: Keep PPI as the answer to "drug of choice", then add one line on P-CABs for severe erosive disease.

Question 2. *Helicobacter pylori* — diagnosis and eradication 15 MARKS

Answer — Textbook Core (10 points)

1. **Definition.** *H. pylori* is a **Gram-negative, spiral, urease-producing** bacterium that colonises the gastric mucus layer. It is a **WHO Class I carcinogen**.
2. **Why it matters in the elderly.** Prevalence rises with age (over 60–70% in Indian elderly). It causes **chronic gastritis, peptic ulcer, gastric MALT lymphoma and gastric adenocarcinoma**, and worsens NSAID/aspirin ulcer bleeding.
3. **Indications to test ("test and treat").** Active or past peptic ulcer, gastric MALT lymphoma, after resection of early gastric cancer, unexplained **iron deficiency anaemia**, ITP, long-term NSAID/aspirin or PPI use, and a first-degree relative with gastric cancer.
4. **Alarm features → endoscopy first.** New dyspepsia at age ≥60, weight loss, dysphagia, persistent vomiting, anaemia, GI bleeding or abdominal mass. **Empirical "test and treat" is not appropriate in the elderly with new dyspepsia.**
5. **Non-invasive tests.** **Urea breath test (^{13}C-UBT)** and **stool antigen test** are the tests of choice for both diagnosis and test-of-cure (sensitivity and specificity >90%). **Serology (IgG) cannot distinguish current from past infection** and must never be used for test-of-cure.
6. **Invasive (endoscopic) tests.** **Rapid urease test** on antral + body biopsies, **histology** (Giemsa stain), **culture with susceptibility testing**, and PCR for clarithromycin resistance.
7. **Causes of false-negative tests.** PPI within **2 weeks**, antibiotics or bismuth within **4 weeks**, active bleeding, and extensive atrophic gastritis/intestinal metaplasia. Stop the PPI two weeks before UBT or stool antigen testing.
8. **First-line eradication (textbook).** **Standard triple therapy** — PPI + **clarithromycin 500 mg** + **amoxicillin 1 g**, all twice daily for **14 days**. Metronidazole 400 mg BD replaces amoxicillin in penicillin allergy.
9. **Bismuth quadruple therapy.** PPI BD + bismuth subcitrate + **tetracycline 500 mg QID + metronidazole 400 mg TID for 14 days**. Used when clarithromycin resistance is high, after prior macrolide exposure, or after treatment failure. Salvage options are levofloxacin-based or rifabutin-based triple therapy.
10. **Geriatric considerations and confirmation of cure.** Check interactions (clarithromycin with **statins, digoxin, warfarin and QT-prolonging drugs**), adjust amoxicillin/levofloxacin for renal function, warn about ***C. difficile*** and use adherence aids. **Confirm eradication at least 4 weeks after therapy by UBT or stool antigen — never by serology.**

Latest Updates (2023–2026) — 2 points

11. **ACG Clinical Guideline 2024** (*Am J Gastroenterol* 2024;119:1730) — **optimized bismuth quadruple therapy for 14 days is now first-line** when susceptibility is unknown. **Clarithromycin triple therapy and concomitant therapy are no longer recommended empirically** because clarithromycin resistance is 22–32%. Rifabutin triple therapy and vonoprazan-amoxicillin dual therapy are alternatives, and **test-of-cure is now mandatory in every treated patient**.
12. **Potassium-competitive acid blockers (vonoprazan)** — vonoprazan-amoxicillin dual and vonoprazan-clarithromycin-amoxicillin triple regimens are FDA-approved. Vonoprazan dual therapy was **superior to lansoprazole-clarithromycin triple therapy in clarithromycin-resistant strains (70% vs 32% eradication)**, offering a new option for resistant infection.

Current Practice Update

Textbook: PPI + clarithromycin + amoxicillin triple therapy is first-line.
Current recommendation: Optimized bismuth quadruple therapy for 14 days is first-line; clarithromycin triple therapy only if susceptibility is documented.
Exam approach: Write the textbook regimen first, then add "however, ACG 2024 now recommends optimized bismuth quadruple therapy first-line owing to clarithromycin resistance >15%."

Question 3. Upper gastrointestinal bleeding — risk scores and management 15 MARKS

Answer — Textbook Core (10 points)

1. **Definition.** Bleeding from a source **proximal to the ligament of Treitz**, presenting with haematemesis, coffee-ground vomiting or melaena. Mortality in the elderly is **10–15%**.
2. **Causes in the elderly.** **Peptic ulcer is the commonest**, followed by erosive gastritis/duodenitis, oesophagitis, varices, Mallory-Weiss tear, **angiodysplasia**, malignancy and Dieulafoy lesion. Key drugs: **NSAIDs, aspirin, anticoagulants, SSRIs and corticosteroids**.
3. **Atypical presentation in old age.** May present as **syncope, falls, confusion, angina or unexplained anaemia** rather than overt bleeding. Beta-blockers mask tachycardia, and comorbidity determines mortality more than the volume of blood lost.
4. **Resuscitation.** ABC approach, two wide-bore cannulae, crystalloid, group-and-crossmatch, correct coagulopathy (PCC for warfarin, andexanet/idarucizumab where available). **Restrictive transfusion at Hb <7 g/dL with a target of 7–9 g/dL**; a higher threshold of 8 g/dL is used in cardiovascular disease.
5. **Glasgow-Blatchford Score (GBS) — the pre-endoscopy score of choice.** Uses **urea, haemoglobin, systolic BP, pulse, melaena, syncope, hepatic disease and cardiac failure**. **GBS 0–1 identifies very low-risk patients who can be managed as outpatients** without early endoscopy.
6. **Other risk scores.** **Rockall score** (age, shock, comorbidity, diagnosis, endoscopic stigmata) predicts rebleeding and mortality; **AIMS65** (Albumin <3 g/dL, INR >1.5, altered Mental status, SBP <90, age >65) predicts in-hospital mortality; the **ABC score** predicts 30-day mortality.
7. **Forrest classification of ulcer stigmata.** **Ia** spurting, **Ib** oozing, **IIa** non-bleeding visible vessel, **IIb** adherent clot, **IIc** flat pigmented spot, **III** clean base. **Forrest Ia, Ib and IIa require endoscopic therapy** (rebleed risk approximately 55%, 43% and 22%); IIc and III need no therapy.
8. **Pharmacotherapy.** **High-dose IV PPI — 80 mg bolus then 8 mg/hour for 72 hours** after successful endoscopic haemostasis. **IV erythromycin 250 mg 30–120 minutes before endoscopy** improves visualisation. If varices are suspected, add **terlipressin and prophylactic ceftriaxone**.
9. **Endoscopy.** Perform **within 24 hours** of presentation after resuscitation. For actively bleeding ulcers use **combination therapy — adrenaline injection plus a second modality** (clip or thermal coagulation); adrenaline alone is inadequate. Haemostatic powder (TC-325) is useful for diffuse or malignant bleeding. Routine second-look endoscopy is not recommended.
10. **Failure and secondary prevention.** Rebleeding → repeat endoscopy → **transcatheter arterial embolisation** (preferred in the frail elderly) → surgery if embolisation fails or is unavailable. Then **test and eradicate *H. pylori***, stop NSAIDs, resume aspirin early (within 3–5 days) when needed for secondary prevention, and continue PPI cover.

Latest Updates (2023–2026) — 2 points

11. **AABB 2025 international guideline** (*Ann Intern Med* 2025;178:1469) — based on the **MINT trial (NEJM 2023)**, a **liberal transfusion threshold of Hb <10 g/dL is now suggested in acute myocardial infarction with anaemia**. This is the key exception to the universal restrictive rule and is directly relevant when an older patient bleeds while having myocardial ischaemia.
12. **Canadian Association of Gastroenterology 2025 guideline** (*Gastroenterology* 2025) provides the first dedicated guidance for **non-variceal, non-peptic-ulcer bleeding** (angiodysplasia, GAVE, Dieulafoy lesion, Mallory-Weiss). A 2025 individual-patient-data meta-analysis also confirmed **TC-325 hemostatic powder is superior to conventional therapy for malignant GI bleeding**.

Cross-reference: The transfusion thresholds referred to here, including the acute myocardial infarction exception, are covered in full in Question 25.

Question 4. Malabsorption — etiopathogenesis, clinical features, diagnostic algorithm and management 15 MARKS

Answer — Textbook Core (10 points)

1. **Definitions.** **Maldigestion** is impaired breakdown of nutrients within the lumen or at the brush border; **malabsorption** is impaired mucosal uptake and transport of digested nutrients. Both produce the clinical **malabsorption syndrome**, and the two frequently coexist.
2. **Luminal (maldigestive) causes.** **Chronic pancreatitis and pancreatic exocrine insufficiency** (also pancreatic carcinoma and post-pancreatectomy), **bile salt deficiency** from cholestasis or ileal resection, **small intestinal bacterial overgrowth** with bile salt deconjugation, post-gastrectomy states, and Zollinger-Ellison syndrome (acid inactivation of enzymes).
3. **Mucosal causes.** **Coeliac disease — increasingly diagnosed in the elderly and often presenting with isolated anaemia or osteoporosis rather than diarrhoea**; **tropical sprue (important in India)**; Whipple's disease; Crohn's disease; radiation enteritis; amyloidosis; intestinal lymphoma; giardiasis; HIV enteropathy; and drugs — **metformin, olmesartan, colchicine, NSAIDs, orlistat and neomycin**.
4. **Structural, lymphatic and vascular causes.** Short bowel after resection, blind loops, fistulae and strictures; **intestinal lymphangiectasia** with protein-losing enteropathy; and **chronic mesenteric ischaemia**, which is particularly important in older vasculopaths and presents with **postprandial abdominal pain, food fear and weight loss**.
5. **Pathogenesis.** Reduced absorptive surface area, defective luminal digestion, impaired brush-border enzyme activity, bacterial deconjugation of bile salts and lymphatic obstruction lead to **osmotic diarrhoea and steatorrhoea**, with secondary deficiencies of fat, protein, carbohydrate, vitamins and minerals. Unabsorbed fatty acids and bile acids in the colon further stimulate secretion.
6. **Clinical features.** Chronic diarrhoea with **pale, bulky, offensive, greasy stools that float and are difficult to flush (steatorrhoea)**, flatulence, borborygmi, abdominal distension, and **weight loss despite an adequate appetite and intake**. **In the elderly the presentation is often subtle** — unexplained weight loss, refractory anaemia, oedema, osteoporotic fracture or falls.
7. **Deficiency syndromes (very high yield).** **Iron, folate and B_{12} deficiency anaemia** (often dimorphic); **osteomalacia and osteoporosis from calcium and vitamin D deficiency — bone pain, proximal myopathy and fractures**; bleeding and bruising from **vitamin K deficiency**; **hypoproteinaemic oedema**; night blindness from vitamin A deficiency; glossitis, cheilosis and dermatitis; **tetany from hypocalcaemia and hypomagnesaemia**; and peripheral neuropathy.
8. **Diagnostic algorithm — Step 1: confirm malabsorption and screen for consequences.** Complete blood count with indices and film, iron studies, B_{12} and folate, calcium, phosphate, magnesium, vitamin D, albumin, PT/INR, and thyroid function. Confirm fat malabsorption with **faecal fat estimation — qualitative Sudan III stain or quantitative 72-hour faecal fat >7 g/day**; measure **faecal calprotectin** to separate inflammatory from non-inflammatory causes.
9. **Diagnostic algorithm — Step 2: identify the specific cause.** **IgA anti-tissue transglutaminase with total IgA for coeliac disease, confirmed by duodenal biopsy showing villous atrophy (Marsh grading)**; **faecal elastase-1 <200 µg/g for pancreatic exocrine insufficiency**, with CT or MRCP of the pancreas; **glucose or lactulose hydrogen breath test for bacterial overgrowth** (or an empirical antibiotic trial); stool for ova, parasites and *Giardia* antigen; CT or MR enterography and capsule endoscopy for small bowel disease; and CT angiography if mesenteric ischaemia is suspected. **Remember that coeliac serology is falsely negative in IgA deficiency and**

may be lower-titre in the elderly.

10. **Management. Treat the specific cause** — a lifelong strict **gluten-free diet with dietitian supervision** for coeliac disease; **pancreatic enzyme replacement of 40,000–50,000 units of lipase with each main meal plus a PPI** for exocrine insufficiency; rotating antibiotics (rifaximin, norfloxacin) for bacterial overgrowth; **tetracycline with folic acid for tropical sprue**; and revascularisation for mesenteric ischaemia. **Replace deficiencies** — iron, B_{12}, folate, calcium with vitamin D, fat-soluble vitamins A, D, E and K, zinc and magnesium; use **medium-chain triglycerides** in severe steatorrhoea since they are absorbed without bile salts or lymphatics; monitor bone mineral density; and address weight, sarcopenia and quality of life.

Latest Updates (2023–2026) — 2 points

11. **ACG Clinical Guideline on Coeliac Disease 2023** (*Am J Gastroenterol* 2023;118:59) — **IgA anti-tTG with a total IgA level remains the single best initial test**; **duodenal biopsy (one from the bulb and four from the distal duodenum) is still required in adults** for diagnosis; **HLA-DQ2/DQ8 typing is useful only to exclude the disease**, not to confirm it. The guideline emphasises follow-up biopsy or serology for **persistent villous atrophy**, and routine screening for nutritional deficiency and metabolic bone disease — particularly relevant in older patients diagnosed late.
12. **AGA Clinical Practice Update on exocrine pancreatic insufficiency (2023)** — highlights that PEI is **substantially under-diagnosed and, when diagnosed, systematically under-dosed**. It occurs not only in chronic pancreatitis but also after gastric and pancreatic surgery, and in diabetes, coeliac disease and advanced age. **Faecal elastase-1 should be checked in any patient with unexplained weight loss or steatorrhoea**, and enzyme doses should be titrated upward against symptoms and nutritional markers rather than left at a fixed low dose.

Question 5. Constipation and faecal impaction in the elderly 15 MARKS

Answer — Textbook Core (10 points)

1. **Definition (Rome IV).** Two or more of — straining, lumpy or hard stools, sensation of incomplete evacuation, sensation of anorectal obstruction, need for manual manoeuvres, and **fewer than three spontaneous bowel movements per week** — present for 3 months with onset at least 6 months earlier.
2. **Prevalence.** Affects **30–40% of community-dwelling elderly and more than 50% of nursing home residents**; laxative use is even more common than the symptom itself.
3. **Age-related mechanisms.** Reduced fibre and fluid intake, **immobility**, **reduced rectal sensation and compliance**, and impaired pelvic floor and anal sphincter function. **Colonic transit itself is largely preserved in healthy ageing** — constipation is not a normal part of ageing.
4. **Classification. Primary** — normal-transit, slow-transit, and defecatory (dyssynergic) disorder. **Secondary** — drug-induced, metabolic, neurological or obstructive.
5. **Drug causes (very high yield). Opioids, anticholinergics, tricyclic antidepressants, calcium channel blockers (verapamil), oral iron, calcium and aluminium antacids, diuretics, antiparkinsonian drugs, ondansetron and clozapine.**
6. **Secondary causes to exclude. Hypothyroidism, hypercalcaemia, hypokalaemia, diabetes, dehydration, Parkinson's disease, stroke, spinal cord lesions, depression, colorectal carcinoma and strictures.**
7. **Assessment.** History with the **Bristol Stool Chart**, full drug review, abdominal examination, and a **digital rectal examination in every patient** (to detect impaction, tone, masses and dyssynergia). Alarm features — weight loss, bleeding, anaemia, recent change in bowel habit — require **colonoscopy**. Refractory cases need **anorectal manometry and balloon expulsion testing**.
8. **Faecal impaction — clinical features.** A hard faecal mass in the rectum (or higher in the colon), **overflow (paradoxical) diarrhoea and faecal incontinence**, abdominal pain, anorexia, urinary retention and **delirium**. Complications include **stercoral ulceration and perforation**, bleeding, sepsis and sigmoid volvulus.
9. **Management of impaction. Manual disimpaction with adequate analgesia** for a low, hard impaction, followed by a **phosphate or arachis oil retention enema**. High impaction is treated with **high-dose oral polyethylene glycol (macrogol)** — contraindicated if obstruction is suspected. Follow with maintenance laxatives and bowel retraining to prevent recurrence.
10. **Stepwise treatment of constipation.** Mobility, fluids, gradual fibre increase (**avoid bulking agents in immobile, impacted or opioid-treated patients**), and toileting after meals to use the **gastrocolic reflex → osmotic laxative — polyethylene glycol is first-line** (better evidence than lactulose) → stimulant (senna, bisacodyl) → secretagogues

(lubiprostone, linaclotide) or the prokinetic **prucalopride → PAMORAs (methylnaltrexone, naloxegol, naldemedine) for opioid-induced constipation**. **Biofeedback is the treatment of choice for dyssynergic defecation.** Avoid magnesium salts in CKD; docusate is ineffective.

Latest Updates (2023–2026) — 2 points

11. **AGA/ACG joint clinical guideline 2023** on chronic idiopathic constipation — **strong recommendation for polyethylene glycol and senna**; conditional recommendations for linaclotide, plecanatide, prucalopride and lubiprostone; and a **recommendation against routine use of docusate and other stool softeners**, which are ineffective.
12. **WGO Global Guideline on Constipation 2025** and the **ASCRS 2024 guideline** reaffirm the Rome IV framework, recommend **early anorectal physiology testing in refractory cases** rather than escalating laxatives blindly, and confirm that **bulk-forming laxatives must be avoided in opioid-induced constipation** because of the risk of impaction and obstruction.

Cross-reference: The Bristol Stool Chart and the mode of action of each laxative class are set out in Question 6.

Question 6. Bristol Stool Chart; classes of laxatives and their modes of action 7 MARKS

Answer — Textbook Core (6 points)

1. **The Bristol Stool Form Scale — purpose and diagram.** A validated seven-point pictorial scale developed at Bristol Royal Infirmary that classifies stool by form, which **correlates with whole-gut transit time**. It gives an objective, reproducible way for patients and nurses to describe stool, and is used to diagnose and monitor constipation, diarrhoea and faecal incontinence.

Type	Appearance	Description	Interpretation
Type 1	● ● ● ●	Separate hard lumps, like nuts, hard to pass	**Constipation** (slow transit)
Type 2	▮▮▮▮▮	Sausage-shaped but lumpy	
Type 3	▮▮▮▮	Sausage-shaped with cracks on the surface	**Normal** (Type 4 is ideal)
Type 4	▮▮▮▮	Smooth and soft, like a sausage or snake	
Type 5	◉ ◉ ◉	Soft blobs with clear-cut edges, passed easily	Tending to **lack of fibre**
Type 6	▒▒▒▒	Fluffy pieces with ragged edges, mushy stool	**Diarrhoea** (rapid transit, urgency)
Type 7	≈≈≈≈	Entirely liquid, watery, no solid pieces	

2. **Clinical use of the chart.** **Types 1–2 indicate constipation, 3–4 are normal (Type 4 ideal), 5 suggests inadequate fibre, and 6–7 indicate diarrhoea.** It is used to satisfy the Rome IV criterion of "lumpy or hard stools", to **titrate laxatives (target Type 3–4)**, to monitor bowel preparation, to distinguish true diarrhoea from **overflow around faecal impaction**, and as a nursing chart in institutional and palliative care.
3. **Bulk-forming laxatives.** **Ispaghula/psyllium, methylcellulose and sterculia.** Mode of action — they are **non-absorbable hydrophilic polysaccharides that increase faecal mass and retain water, distending the colon and stimulating peristalsis**. Onset 2–3 days; they require an adequate fluid intake. **Avoid in immobile, dehydrated, opioid-treated or impacted patients because of the risk of obstruction and faecal loading.**
4. **Osmotic laxatives.** **Polyethylene glycol (macrogol) — first-line, with the best evidence**, and **lactulose**, a non-absorbable disaccharide that is fermented by colonic bacteria to organic acids, drawing water osmotically (and useful in hepatic encephalopathy). **Magnesium salts and sodium phosphate** act more rapidly. Cautions: **avoid magnesium salts in renal impairment (hypermagnesaemia) and phosphate enemas in the frail elderly (hyperphosphataemia, hypocalcaemia and acute kidney injury)**; lactulose causes bloating and flatulence.
5. **Stimulant (irritant) laxatives.** **Senna, bisacodyl and sodium picosulfate.** Mode of action — they **stimulate the myenteric plexus to increase colonic motility and also reduce water and electrolyte absorption**. Onset 6–12 hours orally (bisacodyl suppository within 1 hour). They cause abdominal cramps and hypokalaemia with prolonged use, and anthraquinones produce **melanosis coli** (benign and reversible). They are the most useful class alongside opioids.
6. **Softeners, lubricants and newer agents.** **Docusate sodium** is a surfactant that lowers stool surface tension but is **largely ineffective and no longer recommended**; **liquid paraffin must be avoided in the elderly because of**

lipoid aspiration pneumonia; glycerine suppositories act as rectal lubricants and stimulants. Newer classes: **secretagogues** — lubiprostone (a chloride channel-2 activator) and linaclotide/plecanatide (guanylate cyclase-C agonists) which increase intestinal chloride and water secretion; the **prokinetic prucalopride**, a selective 5-HT_4 agonist that accelerates colonic transit; and **PAMORAs — methylnaltrexone, naloxegol and naldemedine**, peripherally acting mu-opioid receptor antagonists that reverse opioid-induced constipation without reversing analgesia.

Latest Update (2023–2026) — 1 point

7. **AGA/ACG joint guideline on chronic idiopathic constipation (2023)**, reinforced by the **WGO Global Guideline 2025 — polyethylene glycol and senna carry strong recommendations**; linaclotide, plecanatide, prucalopride and lubiprostone are conditional recommendations for refractory cases; there is an explicit **recommendation against docusate and other stool softeners**; and **PAMORAs are the agents of choice for opioid-induced constipation, in which bulk-forming laxatives should be avoided altogether**.

Cross-reference: The full stepwise management of constipation, including faecal impaction, is given in Question 5; this answer covers the chart and the pharmacology of each laxative class.

Question 7. Colorectal carcinoma — presentation and staging 15 MARKS

Answer — Textbook Core (10 points)

1. **Epidemiology and risk factors.** Median age at diagnosis is about 70 years. Risk factors are **age, adenomatous polyps, inflammatory bowel disease, family history, Lynch syndrome and FAP**, red meat, obesity, smoking and diabetes. India shows rising incidence with a higher proportion of rectal and younger-onset disease.
2. **Molecular pathways.** The **adenoma-carcinoma sequence** (APC → KRAS → p53), the serrated pathway (BRAF, CIMP), and **mismatch repair deficiency (dMMR/MSI-high) in about 15%** of tumours.
3. **Right-sided presentation.** **Iron deficiency anaemia**, fatigue, weight loss, vague right iliac fossa pain and a palpable mass. Obstruction occurs late because stool is fluid and the caecum is capacious. **Unexplained iron deficiency anaemia in an older adult is right-sided colon cancer until proved otherwise.**
4. **Left-sided and rectal presentation.** **Change in bowel habit**, blood mixed with stool, colicky pain, and obstruction. Rectal cancer causes **bleeding, tenesmus, mucus discharge and a sense of incomplete evacuation**.
5. **Emergency presentation.** Up to 20% of elderly patients present as an emergency with **large bowel obstruction, perforation with peritonitis, or acute lower GI bleeding**, all carrying high operative mortality.
6. **Investigations.** **Colonoscopy with biopsy is the gold standard**; CT colonography if colonoscopy is incomplete or unsafe. **CEA is for prognosis and follow-up, not diagnosis.**
7. **Staging investigations.** **CT chest, abdomen and pelvis** for distant disease; **MRI pelvis for rectal cancer** to assess T stage and the circumferential resection margin; endorectal ultrasound for early rectal lesions; PET-CT for equivocal or recurrent disease.
8. **TNM staging (AJCC 8th edition).** **T1** submucosa, **T2** muscularis propria, **T3** through into pericolorectal tissue, **T4a** visceral peritoneum, **T4b** adjacent organ. **N1** = 1–3 nodes, **N2** = 4 or more nodes. **M1a** one organ, **M1b** more than one organ, **M1c** peritoneal. **At least 12 lymph nodes must be examined** for adequate staging.
9. **Stage groups and prognosis.** Stage I about **90%** 5-year survival, Stage II **70–80%**, Stage III **50–65%**, Stage IV about **15%**. Older **Dukes** classification: A confined to bowel wall, B through the wall, C nodal involvement, D distant metastasis.
10. **Geriatric principles.** **Age alone is not a contraindication to curative surgery**; decisions should follow **comprehensive geriatric assessment and frailty screening (G8, CARG or CRASH scores)**. Adjuvant oxaliplatin benefit is less certain over 70 years (single-agent capecitabine is often preferred), and screening should stop when life expectancy is under 10 years (usually age 75–85).

Latest Updates (2023–2026) — 2 points

11. **Non-operative management of dMMR tumours** — neoadjuvant **dostarlimab** (anti-PD-1) produced a **100% clinical complete response in 49 patients with dMMR locally advanced rectal cancer** with 92% disease-free at two years (Cercek, *NEJM* 2025), avoiding surgery and radiotherapy entirely. **Every colorectal cancer must now be tested for MMR/MSI status.**
12. **ctDNA-guided adjuvant therapy** — the **DYNAMIC trial** showed post-operative circulating tumour DNA identifies which stage II patients truly need chemotherapy, **halving chemotherapy use without worsening recurrence-free survival**. At ASCO 2025 the **ATOMIC trial** established that adding atezolizumab to adjuvant mFOLFOX improves disease-free survival in stage III dMMR colon cancer.

Cross-reference: Screening for colorectal cancer is dealt with separately in Question 40; this answer covers presentation and staging only.

Question 8. Palliative management of malignant bowel obstruction 7 MARKS

Answer — Textbook Core (6 points)

1. **Definition and context.** Mechanical or functional obstruction **distal to the ligament of Treitz** in a patient with known intra-abdominal or pelvic malignancy. It is commonest in **ovarian and colorectal cancer** (10–28% of colorectal cancers), is frequently **multi-level due to peritoneal carcinomatosis**, and carries a median survival of only weeks to a few months when inoperable.
2. **Clinical features and assessment.** Colicky and continuous abdominal pain, vomiting (early and bilious if proximal, late and faeculent if distal), distension and absolute constipation. **Contrast CT abdomen is the investigation of choice** to define the level, cause, whether there are single or multiple sites, and whether ischaemia is present.
3. **Decision-making.** Multidisciplinary assessment of performance status, prognosis, nutrition, disease extent and the patient's own wishes. **Surgery or stenting is appropriate only for a single-level obstruction in a patient with good performance status, without ascites or carcinomatosis.** A **self-expanding metallic stent** is preferred for a single left-sided colonic or gastroduodenal obstruction.
4. **Drug therapy — analgesia and antiemesis.** All drugs must be given **subcutaneously or parenterally** because oral absorption is unreliable. Use a **subcutaneous morphine infusion** (renally adjusted, or fentanyl in renal impairment) with **hyoscine butylbromide 60–120 mg/24 h for colic**. For antiemesis, **haloperidol 1.5–3 mg/24 h in complete obstruction**; **metoclopramide 30–60 mg/24 h only in incomplete or functional obstruction — it is contraindicated in complete obstruction because it precipitates colic and perforation**.
5. **Antisecretory and anti-inflammatory therapy.** **Octreotide 300–600 µg/24 h subcutaneously** reduces gastrointestinal secretions and vomiting within 24–48 hours and is more effective than hyoscine butylbromide. **Dexamethasone 6–16 mg/24 h** may resolve the obstruction by reducing peritumoural and bowel wall oedema — **stop it if there is no response within 5 days**. Add a proton pump inhibitor or H_2 blocker to reduce gastric secretion volume.
6. **Supportive and non-drug measures.** A **venting nasogastric tube for short-term relief only**; a **venting gastrostomy (PEG)** for irreversible obstruction so the patient may eat and drink for pleasure; **judicious hydration of 500–1000 mL/24 h subcutaneously** (over-hydration worsens secretions and vomiting); meticulous mouth care for thirst; parenteral nutrition only in selected patients with a prognosis beyond 2–3 months; and **early palliative care referral with advance care planning and family communication**.

Latest Update (2023–2026) — 1 point

7. **ASCO Palliative Care Guideline Update 2024** (*J Clin Oncol* 2024;42:2336) together with the **S1316 trial** confirm that **"triple therapy" — dexamethasone plus octreotide plus an antiemetic — is the standard non-surgical medical regimen**, and that although surgery gives better symptom scores on the MDASI-GI instrument, it offers **no survival advantage and causes more treatment-related complications (15% vs 4%)**. Surgery should therefore be reserved for carefully selected patients after shared decision-making.

PART II — HEPATOLOGY

Question 9. Causes of jaundice in an older person and their management 7 MARKS

Answer — Textbook Core (6 points)

1. **Definition and framework.** Jaundice is yellow discoloration of skin and sclerae when serum bilirubin exceeds about **2.5–3 mg/dL**. Classify as **pre-hepatic, hepatocellular or post-hepatic (obstructive)**. In older adults, **obstructive and malignant causes predominate, and drugs must always be excluded first**.
2. **Pre-hepatic causes.** Haemolysis (autoimmune, drug-induced, transfusion reaction), **ineffective erythropoiesis** (megaloblastic anaemia, myelodysplastic syndrome) and resorption of a large haematoma. Features: **unconjugated hyperbilirubinaemia with normal liver enzymes, raised LDH, low haptoglobin and reticulocytosis**.
3. **Hepatocellular causes.** **Drug-induced liver injury (the commonest cause in the elderly)**, viral hepatitis (A and E are more severe in old age; B and C), **alcoholic hepatitis**, autoimmune hepatitis, **ischaemic hepatitis or "shock liver"**, cirrhosis with decompensation, and sepsis-associated cholestasis. The **transaminases rise disproportionately**.
4. **Post-hepatic (obstructive) causes.** **Choledocholithiasis**, and malignancy — **carcinoma of the head of the pancreas, cholangiocarcinoma and periampullary carcinoma** giving painless progressive jaundice with a palpable gallbladder (**Courvoisier's law**); also benign strictures, primary sclerosing cholangitis, primary biliary cholangitis and drug-induced cholestasis. Features: **conjugated hyperbilirubinaemia with raised ALP and GGT, pale stools, dark urine and pruritus**.
5. **Evaluation.** History of **drugs including ayurvedic and herbal preparations**, alcohol, transfusion and travel; examination for stigmata of chronic liver disease, palpable gallbladder, nodes and masses. Investigations: LFT pattern with **R value**, **PT/INR (the best marker of severity)**, albumin, viral serology, autoantibodies and ferritin. **Ultrasound abdomen is the first imaging test** — if ducts are dilated proceed to MRCP, EUS or ERCP; if not dilated, complete the hepatocellular workup ± biopsy.
6. **Management.** **Treat the cause** — withdraw the offending drug, antivirals for hepatitis B and C, corticosteroids for autoimmune or severe alcoholic hepatitis, and **ERCP with stone extraction or stenting** for obstruction (percutaneous transhepatic drainage if ERCP fails). Supportive care: **vitamin K**, correction of dehydration and acute kidney injury, nutritional support, cholestyramine or rifampicin for pruritus, and avoidance of hepatotoxic drugs and sedatives. **Ascending cholangitis (Charcot's triad of fever, jaundice and right upper quadrant pain) requires urgent antibiotics plus biliary drainage.**

> **Latest Update (2023–2026) — 1 point**
>
> 7. **New nomenclature for steatotic liver disease** (multi-society Delphi consensus, *Hepatology* 2023) — NAFLD is replaced by **MASLD (metabolic dysfunction-associated steatotic liver disease)**, NASH by **MASH**, and a new category **MetALD** covers patients with both metabolic risk factors and significant alcohol intake, which is exactly the overlap commonly seen in older Indian patients. MASLD is now the commonest chronic liver disease worldwide and an increasingly frequent background cause of jaundice in the elderly, and **resmetirom became the first drug approved for MASH with F2–F3 fibrosis (2024)**.

Cross-reference: The MASLD/MetALD nomenclature introduced here is expanded, with the new drug therapy, in Question 14.

Question 10. Drug-induced jaundice 7 MARKS

Answer — Textbook Core (6 points)

1. **Definition and importance.** Drug-induced liver injury (DILI) is liver damage caused by drugs, herbs or supplements. It is the **commonest cause of acute liver failure in the West** and an important cause of jaundice in the elderly because of **polypharmacy, reduced hepatic mass and blood flow, and altered cytochrome P450 metabolism**.
2. **Types and biochemical pattern.** **Intrinsic** (dose-dependent and predictable — paracetamol) versus **idiosyncratic** (dose-independent, unpredictable — most drugs). Pattern is defined by the **R value = (ALT/ULN) ÷ (ALP/ULN)**: **R ≥5 hepatocellular, R ≤2 cholestatic, R 2–5 mixed**.
3. **Common culprit drugs.** **Amoxicillin-clavulanate (the commonest single idiosyncratic cause)**, **anti-tubercular drugs — isoniazid, rifampicin, pyrazinamide (highly relevant in India)**, NSAIDs (diclofenac), statins, methotrexate, amiodarone, nitrofurantoin, azathioprine, phenytoin and carbamazepine, allopurinol, anabolic

steroids, **herbal and ayurvedic preparations**, and **immune checkpoint inhibitors**.

4. **Clinical phenotypes.** Acute hepatitis; cholestatic hepatitis (co-amoxiclav, erythromycin); pure cholestasis (anabolic steroids, oestrogens); **hypersensitivity/DRESS with fever, rash and eosinophilia**; **drug-induced autoimmune hepatitis (nitrofurantoin, minocycline, statins, diclofenac)**; **vanishing bile duct syndrome**; steatohepatitis (amiodarone, tamoxifen); and sinusoidal obstruction syndrome.
5. **Diagnosis.** DILI is a **diagnosis of exclusion**. Establish a temporal relationship (latency of days to months), demonstrate improvement after withdrawal (**dechallenge**), and exclude viral, autoimmune and biliary disease by serology and ultrasound. Use the **RUCAM causality score**; liver biopsy only when the diagnosis is unclear or there is no recovery. **Deliberate rechallenge is contraindicated.**
6. **Management and prognostic rule. Immediate withdrawal of the offending drug is the definitive treatment.** Give supportive care and avoid other hepatotoxins; **N-acetylcysteine for paracetamol toxicity and for non-paracetamol acute liver failure**; corticosteroids for hypersensitivity reactions and drug-induced autoimmune hepatitis; ursodeoxycholic acid and cholestyramine for cholestatic pruritus. **Hy's Law — ALT >3× ULN with bilirubin >2× ULN in the absence of cholestasis predicts about 10% mortality**; refer urgently to a transplant centre if the INR rises or encephalopathy develops. Report to pharmacovigilance (PvPI in India).

Latest Update (2023–2026) — 1 point

7. **Two current developments.** First, the **RECAM (Revised Electronic Causality Assessment Method)** is being adopted in place of RUCAM as a more reproducible, computerised causality tool, supported by the **LiverTox** database for drug-specific likelihood scores. Second, **immune checkpoint inhibitor hepatitis** is now a distinct and increasingly common form of DILI in older patients with cancer — it is graded by CTCAE, managed by withholding the drug with **corticosteroids for grade 2–3 and mycophenolate for steroid-refractory cases**, and notably does **not** follow the usual DILI rule that rechallenge is always forbidden.

Question 11. Chronic active hepatitis 6 MARKS

Answer — Textbook Core (5 points)

1. **Definition and modern terminology.** Chronic hepatitis is hepatic necroinflammation persisting for **more than 6 months**. The old histological labels "chronic active hepatitis" (**interface hepatitis/piecemeal necrosis extending beyond the limiting plate, progressing to cirrhosis**) and "chronic persistent hepatitis" have been replaced by classification according to **aetiology + grade of activity + stage of fibrosis**.
2. **Causes. Chronic hepatitis B ± D, chronic hepatitis C, autoimmune hepatitis**, drugs (nitrofurantoin, minocycline, methyldopa, isoniazid), Wilson disease, alpha-1 antitrypsin deficiency and MASH.
3. **Autoimmune hepatitis in the elderly.** Often presents **acutely or as already established cirrhosis**, or is found incidentally; features are fatigue, arthralgia, jaundice and amenorrhoea (not applicable in old age), with **associated autoimmune disease such as thyroiditis and rheumatoid arthritis**. **Type 1 — ANA and anti-smooth muscle antibody with raised IgG**; Type 2 — anti-LKM1 (a disease of the young).
4. **Diagnosis.** Raised transaminases with **hypergammaglobulinaemia (raised IgG)**, autoantibodies, and liver biopsy showing **interface hepatitis with a lymphoplasmacytic infiltrate, hepatocyte rosettes and emperipolesis**. The **IAIHG simplified criteria** (autoantibody titre, IgG, histology, exclusion of viral hepatitis) are used for diagnosis; grade by METAVIR or Ishak and stage with transient elastography.
5. **Treatment. Prednisolone 0.5–1 mg/kg induction with azathioprine 1–2 mg/kg as steroid-sparing maintenance**; **budesonide** may be used in non-cirrhotic patients but **must be avoided in cirrhosis because portosystemic shunting bypasses first-pass metabolism**. Check **TPMT activity before azathioprine**. Older patients respond well biochemically but tolerate steroids poorly — give **osteoporosis prophylaxis, monitor glucose and blood pressure, and consider PCP prophylaxis**. Treat hepatitis B with tenofovir or entecavir, and hepatitis C with direct-acting antivirals (SVR >95%, well tolerated in the elderly).

Latest Update (2023–2026) — 1 point

6. **CAMARO trial** (*J Hepatol* 2024;80:576) — in treatment-naive autoimmune hepatitis, **mycophenolate mofetil with prednisolone achieved biochemical remission more often and with fewer adverse events than azathioprine with prednisolone**, challenging azathioprine's position as standard first-line maintenance. In parallel, the accepted treatment endpoint is now **complete biochemical response — normalisation of both ALT and IgG — ideally within 6 months**, rather than transaminase normalisation alone.

Current Practice Update

Textbook: Prednisolone plus azathioprine is the standard first-line regimen for autoimmune hepatitis.
Current recommendation: Mycophenolate mofetil plus prednisolone is at least as effective and better tolerated (CAMARO 2024); target is complete biochemical response (ALT and IgG normal).
Exam approach: Write the classical regimen, then add the CAMARO line — it converts a routine answer into an updated one.

Question 12. Alcoholic hepatitis 7 MARKS

Answer — Textbook Core (6 points)

1. **Definition.** An acute clinical syndrome of **rapid onset or worsening of jaundice** in a patient with sustained heavy alcohol use, with underlying histological steatohepatitis. Severe disease may progress to **acute-on-chronic liver failure with a 1-month mortality of 20–50%**.
2. **Diagnostic criteria (NIAAA).** Onset of jaundice within **8 weeks**; ongoing heavy alcohol use (**>40 g/day in women, >60 g/day in men**) for ≥6 months with fewer than 60 days abstinence before onset; **AST >50 IU/L with AST:ALT ratio >1.5 and both below 400 IU/L**; bilirubin >3 mg/dL; and exclusion of other liver disease. **Biopsy is not needed unless there are confounding factors.**
3. **Clinical features.** Tender hepatomegaly, fever, anorexia, ascites, encephalopathy and neutrophil leucocytosis. **In older patients alcohol misuse is frequently unrecognised**, and presentation may be with confusion, falls, malnutrition or unexplained jaundice.
4. **Severity scores.** **Maddrey Discriminant Function ≥32** defines severe disease; **MELD >20 is the most accurate score**; also the Glasgow Alcoholic Hepatitis Score and the ABIC score. **The Lille score at day 7 — a value >0.45 indicates steroid non-response and steroids should be stopped.**
5. **General management.** **Abstinence is the single most important determinant of survival.** Manage alcohol withdrawal (benzodiazepines cautiously in the elderly — prefer **lorazepam**, which has no active metabolites), give **thiamine before any glucose**, provide aggressive nutrition (**35 kcal/kg and 1.2–1.5 g/kg protein**) preferably enterally, actively look for and treat infection, avoid nephrotoxins, and manage acute kidney injury, electrolytes and encephalopathy.
6. **Specific therapy.** **Prednisolone 40 mg daily for 28 days** in severe disease (DF ≥32 or MELD >20), after excluding active infection, gastrointestinal bleeding and renal failure; reassess with the Lille score at day 7. **Pentoxifylline is no longer recommended.** Long-term care involves treating alcohol use disorder (**baclofen is preferred in cirrhosis**; acamprosate and naltrexone are limited by hepatotoxicity), vaccination, and consideration for transplantation.

Latest Update (2023–2026) — 1 point

7. **ACG Clinical Guideline 2024** (*Am J Gastroenterol* 2024;119:30) — adopts the term **"alcohol-associated liver disease"**; recommends corticosteroids **with adjuvant intravenous N-acetylcysteine** for severe alcohol-associated hepatitis; recommends **against pentoxifylline and against routine prophylactic antibiotics**; and gives a stronger recommendation for **early liver transplantation** in steroid non-responders at low risk of relapse, explicitly stating that **candidacy must not be based on an arbitrary 6-month sobriety rule** but on structured psychosocial assessment (SALT score, Stanford Integrated Psychosocial Assessment Tool).

Question 13. Management of newly diagnosed hepatitis B in the elderly 7 MARKS

Answer — Textbook Core (6 points)

1. **Confirm and characterise the infection.** Repeat **HBsAg at 6 months to confirm chronicity**; check **HBeAg/anti-HBe, quantitative HBV DNA, ALT**, and screen for **anti-HDV, anti-HCV and HIV**. Assess fibrosis with **APRI, FIB-4 and transient elastography**, and perform an ultrasound of the liver.
2. **Determine the phase of infection.** HBeAg-positive chronic infection (immune-tolerant), HBeAg-positive chronic hepatitis, HBeAg-negative chronic infection (inactive carrier), HBeAg-negative chronic hepatitis, and resolved/occult infection. **Older patients are usually HBeAg-negative with fluctuating DNA and ALT, so a single normal ALT does not exclude active disease.**
3. **Who to treat.** **All patients with cirrhosis and any detectable HBV DNA, regardless of ALT**; those with **HBV DNA >2000 IU/mL and ALT above the upper limit of normal**; significant fibrosis (≥F2) on elastography or APRI; extrahepatic manifestations (glomerulonephritis, vasculitis); and a family history of hepatocellular carcinoma.

4. **Antiviral therapy.** An oral nucleos(t)ide analogue with a **high genetic barrier to resistance — tenofovir disoproxil fumarate, tenofovir alafenamide or entecavir** — once daily and usually lifelong. **In older adults prefer tenofovir alafenamide or entecavir**, because TDF causes **renal tubular dysfunction and reduced bone mineral density**; entecavir requires dose adjustment for creatinine clearance. **Pegylated interferon is generally unsuitable in the elderly.**
5. **Prophylaxis and prevention of flares.** Give **antiviral prophylaxis before immunosuppression or chemotherapy** — especially **rituximab and high-dose corticosteroids** — in HBsAg-positive and anti-HBc-positive patients, continuing for 6–12 months afterwards, because reactivation can be fatal. **Never stop antivirals abruptly.** Vaccinate household and sexual contacts, and give hepatitis A vaccine.
6. **Monitoring and surveillance.** ALT and HBV DNA every 3–6 months; renal function, phosphate and bone density on tenofovir. **Six-monthly ultrasound ± alpha-fetoprotein for hepatocellular carcinoma in all cirrhotics, and also in non-cirrhotic HBsAg-positive Asian men over 40 and women over 50**; endoscopy for varices if cirrhotic. Counsel on alcohol avoidance, drug interactions and transmission.

Latest Update (2023–2026) — 1 point

7. **WHO Guidelines for chronic hepatitis B, March 2024** — treatment eligibility has been **substantially expanded and simplified**. Treat if there is **significant fibrosis or cirrhosis (APRI >0.5 or elastography)**; or **HBV DNA >2000 IU/mL with raised ALT**; or the presence of **coinfection (HIV, HDV, HCV), a family history of cirrhosis or liver cancer, immunosuppression, comorbidity such as diabetes or MASLD, or extrahepatic manifestations — regardless of DNA and ALT levels**; and, where DNA testing is unavailable, on persistently abnormal ALT alone. The guideline also recommends **point-of-care HBV DNA and reflex viral load testing**, and **testing every HBsAg-positive person for hepatitis D**.

Current Practice Update

Textbook: Treat only cirrhotics or those with persistently raised transaminases and high HBV DNA.
Current recommendation: WHO 2024 has widened eligibility to include comorbidity, family history, immunosuppression and coinfection irrespective of DNA/ALT — substantially more patients now qualify.
Exam approach: Give the classical AASLD/EASL criteria, then add the WHO 2024 expansion in one line.

Question 14. Decompensated chronic liver disease in the elderly, and the OPD approach to chronic liver disease 15 MARKS

Answer — Textbook Core (10 points)

1. **Definition.** Cirrhosis is diffuse hepatic fibrosis with regenerative nodules. **Decompensation is defined by the appearance of ascites, variceal bleeding, hepatic encephalopathy or jaundice** (Baveno VII). Median survival is **more than 12 years in compensated cirrhosis but about 2 years once decompensated**.
2. **Aetiology in the elderly.** **MASLD/MASH (now the commonest)**, alcohol-related liver disease, chronic hepatitis B and C, drug-induced injury, autoimmune hepatitis, primary biliary cholangitis, haemochromatosis, and **cardiac (congestive) hepatopathy from right heart failure — a frequently missed cause in older patients**.
3. **Pathogenesis.** Chronic injury activates **hepatic stellate cells** → fibrosis and nodules → **increased intrahepatic resistance plus splanchnic vasodilatation** → portal hypertension (**HVPG >10 mmHg = clinically significant portal hypertension**) → hyperdynamic circulation with effective arterial underfilling → activation of the renin-angiotensin-aldosterone and sympathetic systems → sodium and water retention producing **ascites, dilutional hyponatraemia and hepatorenal syndrome**. Systemic inflammation and bacterial translocation drive acute-on-chronic liver failure.
4. **Precipitants of decompensation.** **Infection, especially spontaneous bacterial peritonitis**; gastrointestinal bleeding; alcohol binge; dehydration or excessive diuresis; constipation; sedatives, opioids and NSAIDs; hepatotoxic drugs; portal vein thrombosis; hepatocellular carcinoma; and surgery.
5. **Clinical features.** Jaundice, ascites, oedema, spider naevi, palmar erythema, gynaecomastia, testicular atrophy, Dupuytren's contracture, caput medusae, splenomegaly, asterixis, and **muscle wasting with sarcopenia**. **In the elderly, decompensation commonly presents as delirium, falls or functional decline rather than classical signs.**
6. **Approach to the OPD patient with chronic liver disease.** History — quantified alcohol intake, all drugs including **ayurvedic and over-the-counter preparations**, transfusion, risk factors and family history. Examination for stigmata plus **nutritional and frailty assessment**. Baseline tests: LFT, PT/INR, albumin, complete blood count (**thrombocytopenia suggests portal hypertension**), renal function and electrolytes. **Aetiological screen**: HBsAg, anti-HCV, autoantibodies with IgG, ferritin and transferrin saturation, ceruloplasmin if under 55, alpha-1 antitrypsin,

and lipid and glucose profile.

7. **Staging and prognostic scoring.** **Child-Turcotte-Pugh score** using bilirubin, albumin, INR, ascites and encephalopathy (**A 5–6, B 7–9, C 10–15**) and **MELD, MELD-Na and MELD 3.0** for transplant prioritisation. Non-invasive fibrosis assessment with **FIB-4, APRI and transient elastography** — liver stiffness **>15 kPa with platelets <150 × 10^9/L suggests clinically significant portal hypertension**.
8. **Management of the stable cirrhotic outpatient.** **Treat the cause** — abstinence, antivirals, weight loss and metabolic control. **Six-monthly ultrasound ± AFP for hepatocellular carcinoma surveillance.** Screening endoscopy for varices (may be deferred if liver stiffness <20 kPa and platelets >150,000 — the Baveno "rule of five"); **carvedilol as the preferred non-selective beta-blocker**. Vaccinate against hepatitis A and B, influenza, pneumococcus and COVID-19; avoid NSAIDs, aminoglycosides and sedatives; salt restriction 5–6 g/day; and treat sarcopenia with **1.2–1.5 g/kg protein and a late evening snack**.
9. **Management of decompensation.** **Ascites** — salt restriction, spironolactone with furosemide, large-volume paracentesis with albumin. **Variceal bleeding** — terlipressin, prophylactic antibiotics, band ligation, and early TIPS in high-risk patients. **Encephalopathy** — treat precipitants, lactulose plus rifaximin. **Spontaneous bacterial peritonitis** — cefotaxime plus **albumin 1.5 g/kg on day 1 and 1 g/kg on day 3**, followed by lifelong norfloxacin prophylaxis. **Hepatorenal syndrome** — terlipressin with albumin.
10. **Complications and transplantation.** Ascites, SBP, variceal bleeding, hepatic encephalopathy, hepatorenal and hepatopulmonary syndromes, portopulmonary hypertension, **hepatocellular carcinoma**, coagulopathy, malnutrition and sarcopenia, osteoporosis and cirrhotic cardiomyopathy. **Refer for transplantation when MELD exceeds 15 or after the first decompensating event**; carefully selected patients over 65–70 with good performance status and social support achieve about **80% five-year survival**, so age alone is not a contraindication.

Latest Updates (2023–2026) — 2 points

11. **Baveno VII consensus** (*J Hepatol* 2022;76:959) — introduced the concept of **"recompensation"** (resolution of ascites, encephalopathy and bleeding off diuretics and lactulose with sustained improvement in liver function after the cause is controlled), the **"rule of five" for liver stiffness**, and the principle that **non-selective beta-blockers, preferably carvedilol, should be given to all patients with clinically significant portal hypertension to prevent the first decompensation (PREDESCI trial)** — not merely to prevent variceal bleeding.
12. **MASLD nomenclature and the first approved drug therapy.** The 2023 multi-society Delphi consensus replaced NAFLD with **MASLD**, NASH with **MASH**, and introduced **MetALD** for combined metabolic and alcohol-related disease. **Resmetirom (a thyroid hormone receptor-β agonist) received approval in 2024 as the first drug for MASH with F2–F3 fibrosis**, and GLP-1 receptor agonists have shown histological benefit in MASH — a major shift from purely lifestyle-based management.

Question 15. Management of hepatic encephalopathy 7 MARKS

Answer — Textbook Core (6 points)

1. **Definition and classification.** Brain dysfunction caused by liver failure and/or portosystemic shunting, ranging from subclinical impairment to coma. **Type A** — acute liver failure; **Type B** — portosystemic bypass without intrinsic liver disease; **Type C** — cirrhosis. Severity is graded by the **West Haven criteria I–IV**; **covert HE** comprises minimal HE plus grade I.
2. **Identify and treat precipitants — the mainstay of management.** **Infection (SBP, urinary and chest), gastrointestinal bleeding, constipation, dehydration and excessive diuresis, hypokalaemia and hyponatraemia, sedatives and opioids, alcohol binge, portal vein thrombosis, TIPS and hepatocellular carcinoma.** In an older patient always exclude other causes of delirium, subdural haematoma, hypoglycaemia and drug toxicity.
3. **Diagnosis.** Essentially clinical — **asterixis, reversal of the sleep-wake cycle, disorientation, constructional apraxia and fetor hepaticus**. A raised venous ammonia supports the diagnosis but does not confirm it; a **normal ammonia makes HE unlikely**, and levels should not be used to monitor treatment. Covert HE is detected with psychometric tests — the **Animal Naming Test, PHES battery or Stroop smartphone app**. Image the brain when the picture is atypical.
4. **First-line treatment.** **Lactulose 30 mL two or three times daily, titrated to produce 2–3 soft stools per day**; in comatose patients give as a retention enema (300 mL lactulose in 700 mL water). It acidifies the colon, converting absorbable ammonia to non-absorbable ammonium, and acts as an osmotic laxative. **Monitor for dehydration, hypernatraemia and aspiration — over-treatment with lactulose can itself precipitate HE.**

5. **Second-line agents and adjuncts.** **Rifaximin 550 mg twice daily added to lactulose** reduces recurrence and hospitalisation and is the agent of choice for **secondary prophylaxis after a second episode of overt HE**. Alternatives: **L-ornithine L-aspartate**, oral branched-chain amino acids, polyethylene glycol for rapid clearance, and zinc if deficient. Neomycin and metronidazole are effective but should be avoided long term in the elderly (ototoxicity, nephrotoxicity, neuropathy).
6. **Nutrition and general care.** **Protein restriction is obsolete and harmful.** Give **1.2–1.5 g/kg/day protein with 35 kcal/kg**, favouring vegetable and dairy protein, in small frequent meals with a **late evening snack** to avoid overnight catabolism. Protect the airway and manage in ICU for grades III–IV; **avoid benzodiazepines** (flumazenil only for proven benzodiazepine ingestion); advise about driving and falls; consider **TIPS reduction or occlusion or embolisation of large spontaneous shunts** for recurrent HE; and refer for liver transplantation.

Latest Update (2023–2026) — 1 point

7. **AASLD/EASL Practice Guidance (2022) and subsequent evidence** — the treatment goal has shifted from clearing the acute episode to **preventing recurrence**, with **lactulose plus rifaximin as standard secondary prophylaxis**. Routine screening for **covert HE** is now recommended because it independently predicts falls, road traffic accidents, poor quality of life and progression to overt HE; and **sarcopenia is recognised as an independent driver of HE** (muscle is the main extra-hepatic site of ammonia detoxification), making nutritional therapy a specific treatment rather than merely supportive care. Faecal microbiota transplantation is promising but remains investigational.

Current Practice Update

Textbook: Restrict dietary protein and use lactulose; monitor response with serum ammonia.
Current recommendation: Give 1.2–1.5 g/kg protein (restriction is harmful), add rifaximin for secondary prophylaxis, and do not use serial ammonia levels to monitor treatment.
Exam approach: Explicitly state "protein restriction is no longer recommended" — examiners look for this single line.

Question 16. Ascites and refractory ascites 7 MARKS

Answer — Textbook Core (6 points)

1. **Causes and initial assessment.** **Cirrhosis with portal hypertension accounts for about 80%**; other causes are malignancy (peritoneal carcinomatosis), heart failure, tuberculosis, nephrotic syndrome and pancreatitis. **Perform a diagnostic paracentesis in every patient with new-onset ascites** — send cell count and differential, albumin, total protein, **culture inoculated into blood culture bottles at the bedside**, and cytology and adenosine deaminase where indicated. **Neutrophils ≥250/mm³ diagnoses spontaneous bacterial peritonitis.**
2. **Serum-ascites albumin gradient (SAAG).** **SAAG ≥1.1 g/dL indicates portal hypertension** (cirrhosis, cardiac ascites — cardiac ascites has an ascitic protein >2.5 g/dL, cirrhotic <2.5). **SAAG <1.1 g/dL indicates non-portal causes** — malignancy, tuberculosis, pancreatic or nephrotic ascites.
3. **Grading.** **Grade 1** detectable only on ultrasound; **Grade 2** moderate, with symmetrical abdominal distension; **Grade 3** large or tense ascites.
4. **First-line management.** **Sodium restriction to 5–6 g of salt (about 2 g sodium) per day**; fluid restriction only if serum sodium is below 125 mmol/L. **Spironolactone 100 mg with furosemide 40 mg daily, maintaining the 100:40 ratio**, increased stepwise to maxima of 400 mg and 160 mg. Target weight loss of **0.5 kg/day without oedema and up to 1 kg/day with oedema**. Monitor weight, sodium, potassium and creatinine, and **stop NSAIDs, ACE inhibitors and ARBs**.
5. **Definition of refractory ascites.** Ascites that cannot be mobilised or that recurs early and cannot be prevented, and is either **diuretic-resistant** (no response to maximal diuretic doses with salt restriction) or **diuretic-intractable** (diuretic-induced complications — encephalopathy, creatinine rise above 2 mg/dL, sodium below 125 mmol/L, or potassium disturbance — preventing the use of effective doses). It occurs in about 10% and carries a **1-year survival of only 50%**.
6. **Management of refractory ascites.** **Serial large-volume paracentesis with albumin 8 g per litre removed** whenever more than 5 litres is drained (to prevent post-paracentesis circulatory dysfunction). **TIPS** for patients needing repeated paracenteses — contraindicated with recurrent encephalopathy, age over 70, Child-Pugh >13 or cardiac dysfunction. **Discontinue beta-blockers if systolic BP falls below 90 mmHg, or with hyponatraemia or AKI.** Consider indwelling tunnelled peritoneal drains or an alfapump for palliation, and **refer for liver transplantation**. Watch for SBP, hepatorenal syndrome, hyponatraemia, umbilical hernia and hepatic hydrothorax.

Latest Update (2023–2026) — 1 point

7. **Baveno VII plus recent trial evidence — TIPS with covered stents should now be considered earlier, in patients requiring three or more large-volume paracenteses per year, irrespective of the presence of varices or previous variceal bleeding**, as this improves transplant-free survival compared with repeated paracentesis. In addition, the **ANSWER trial (Lancet 2018) and subsequent data support long-term weekly albumin infusion** in uncomplicated cirrhotic ascites, which improved survival and reduced complications; this is now standard in some countries, although confirmatory evidence remains debated and cost limits its use in Indian practice.

Question 17. Hepatorenal syndrome 7 MARKS

Answer — Textbook Core (6 points)

1. **Definition and mechanism.** A **functional, potentially reversible** renal failure occurring in advanced cirrhosis with ascites, caused by **intense renal vasoconstriction secondary to splanchnic arterial vasodilatation and effective arterial underfilling**. **The kidneys are structurally normal** — transplanted HRS kidneys function normally in the recipient.
2. **Current nomenclature.** **HRS-AKI** (formerly type 1) — a rise in serum creatinine of **≥0.3 mg/dL within 48 hours or ≥50% from baseline within 7 days**, with rapid deterioration. **HRS-NAKI** (formerly type 2) covers HRS-AKD and HRS-CKD — a slower creatinine rise typically accompanied by **refractory ascites**.
3. **Diagnostic criteria (International Club of Ascites).** Cirrhosis with ascites; AKI by ICA criteria; **no improvement after 2 days of diuretic withdrawal and volume expansion with albumin 1 g/kg/day (maximum 100 g)**; absence of shock; no current or recent nephrotoxic drugs; and **no evidence of structural kidney injury — proteinuria <500 mg/day, no haematuria (<50 RBC/hpf) and a normal renal ultrasound**. Supportive findings: **urine sodium <10 mmol/L, FENa <0.2%** and oliguria.
4. **Precipitants.** **Spontaneous bacterial peritonitis is the classic trigger**; also other sepsis, gastrointestinal bleeding, **large-volume paracentesis without albumin cover**, over-diuresis, diarrhoea from excess lactulose, and NSAIDs, aminoglycosides or contrast media.
5. **Treatment.** **Terlipressin plus albumin is first-line** — terlipressin 1 mg IV every 4–6 hours (or 2–12 mg/day by continuous infusion, which is better tolerated) with **albumin 20–40 g/day**; continue for up to 14 days or until creatinine returns to within 0.3 mg/dL of baseline. Alternatives are **noradrenaline with albumin in an ICU setting**, or midodrine plus octreotide with albumin where terlipressin is unavailable. Watch for **ischaemic complications and, most importantly, respiratory failure and volume overload — the principal safety concern, particularly in the elderly and in acute-on-chronic liver failure**.
6. **Prevention, prognosis and definitive therapy.** Prevent by giving **albumin 8 g per litre of ascites removed during large-volume paracentesis**, albumin in SBP, and antibiotic prophylaxis in high-risk patients; avoid nephrotoxins and NSAIDs. **Liver transplantation is the only definitive treatment**; renal replacement therapy is a bridge to transplantation rather than a treatment in its own right. Untreated HRS-AKI has a median survival of only a few weeks.

Latest Update (2023–2026) — 1 point

7. **ADQI 29 / International Club of Ascites joint consensus 2024** (*J Hepatol* 2024;81:163) — revises the traditional approach by recommending **crystalloids rather than albumin for first-line volume resuscitation in clearly hypovolaemic patients**, and **shortening the diagnostic albumin challenge from 48 to 24 hours** so that terlipressin is not delayed and fluid overload is avoided (a point still actively debated, as up to a third of responses occur between 24 and 48 hours). It also stresses distinguishing HRS-AKI from **acute tubular necrosis using biomarkers such as urinary NGAL**. Terlipressin is now FDA-approved on the basis of the **CONFIRM trial**, carrying a boxed warning for respiratory failure.

PART III — CLINICAL NUTRITION

Question 18. Parenteral nutrition 7 MARKS

Answer — Textbook Core (6 points)

1. **Definition and indications.** Intravenous delivery of nutrients when the gastrointestinal tract cannot be used. Indications are a **non-functioning, inaccessible or obstructed gut** — short bowel syndrome, high-output enterocutaneous fistula, prolonged ileus, complete intestinal obstruction, severe malabsorption, severe mucositis, and severe pancreatitis not tolerating enteral feeding. **The governing principle is "if the gut works, use it" — enteral nutrition is always preferred.**
2. **Routes.** **Central parenteral nutrition** through a subclavian, internal jugular or PICC line with the tip in the superior vena cava is required for hyperosmolar complete formulations. **Peripheral parenteral nutrition** is limited to short-term use (under 10–14 days) with osmolarity below about 850 mOsm/L. Total PN supplies all requirements; supplemental PN tops up inadequate enteral intake.
3. **Composition.** Energy **20–30 kcal/kg/day** (use the lower end in the elderly and critically ill), protein as amino acids **1.0–1.5 g/kg/day**, glucose not exceeding **4–5 mg/kg/min**, and lipid emulsions providing 25–30% of non-protein calories. Add **electrolytes, trace elements (zinc, selenium, copper, chromium) and both water- and fat-soluble vitamins, including thiamine**. Modern practice uses all-in-one three-chamber bags.
4. **Timing and geriatric considerations.** Start only when inadequate oral or enteral intake is expected for **more than 7 days** (earlier if the patient is already malnourished), **begin at about 50% of the target and build up over 2–3 days**. Older patients have less physiological reserve, higher infection risk and reduced cardiac and renal tolerance — **restrict volume, monitor for fluid overload, and always define the goal of therapy before starting**.
5. **Catheter-related complications.** **Catheter-related bloodstream infection is the commonest serious complication**; also pneumothorax, arterial puncture, air embolism, catheter tip malposition, central venous thrombosis and catheter occlusion. Prevention requires **strict aseptic insertion and handling with a dedicated lumen used only for nutrition**, and ultrasound-guided insertion.
6. **Metabolic complications and monitoring.** **Hyperglycaemia (and rebound hypoglycaemia on abrupt withdrawal), refeeding syndrome, electrolyte disturbance, hypertriglyceridaemia, fluid overload, parenteral nutrition-associated liver disease (steatosis and cholestasis), metabolic bone disease and gut mucosal atrophy with bacterial translocation.** Monitor **glucose, electrolytes, phosphate and magnesium daily at first**, then weekly liver function, triglycerides, trace elements and nitrogen balance, with regular weight and fluid balance.

> **Latest Update (2023–2026) — 1 point**
>
> 7. **ESPEN practical guideline on clinical nutrition and hydration in geriatrics** — recommends that **every older patient be screened with a validated tool such as the MNA-SF**, that nutritional goals be individualised through shared decision-making, and, critically, that **artificial nutrition (enteral or parenteral) should not be started in advanced dementia or in the terminal phase of illness**, where **careful hand-feeding and comfort feeding** give better outcomes and quality of life. Parenteral nutrition is justified only when it can achieve an outcome the patient actually values — a point examiners increasingly expect to be linked to advance care planning.

Question 19. Refeeding syndrome 7 MARKS

Answer — Textbook Core (6 points)

1. **Definition.** A potentially fatal shift of fluid and electrolytes that occurs when nutrition is reintroduced to a **malnourished or starved patient**, characterised by **hypophosphataemia (the hallmark), hypokalaemia, hypomagnesaemia, thiamine deficiency and sodium and water retention**.
2. **Pathophysiology.** Prolonged starvation shifts metabolism to fat and protein catabolism and **depletes intracellular phosphate, potassium and magnesium despite normal serum levels**. **Reintroduction of carbohydrate triggers insulin release**, which drives glucose, phosphate, potassium and magnesium intracellularly, consumes phosphate for ATP and 2,3-DPG synthesis, and depletes **thiamine** as a cofactor in carbohydrate metabolism; insulin also causes **renal sodium and water retention**.
3. **Patients at risk (NICE criteria).** **Any one of** — BMI <16 kg/m², unintentional weight loss >15% in 3–6 months,

little or no intake for >10 days, or low potassium, phosphate or magnesium before feeding. **Or any two of** — BMI <18.5, weight loss >10%, negligible intake >5 days, alcohol misuse, or drugs such as insulin, chemotherapy, antacids or diuretics. Relevant geriatric groups: **anorexia of ageing, post-stroke dysphagia, cancer cachexia, prolonged hospitalisation, post-operative starvation and alcohol misuse**.

4. **Clinical features.** Mostly attributable to hypophosphataemia — **muscle weakness, rhabdomyolysis, respiratory failure and failure to wean from ventilation, cardiac failure and arrhythmias, delirium, seizures, haemolysis and thrombocytopenia**; thiamine deficiency causes **Wernicke's encephalopathy and wet beriberi**; fluid retention causes oedema and pulmonary congestion. **Onset is typically within 72 hours of restarting feeding.**
5. **Prevention.** Identify risk **before** feeding begins. Give **thiamine 200–300 mg daily orally (or intravenous Pabrinex) together with a vitamin B complex and multivitamins, starting 30 minutes before feeding and continuing for the first 10 days**. **Start feeding at 10 kcal/kg/day — or 5 kcal/kg/day in extreme risk (BMI <14 or negligible intake for >15 days) — and increase slowly to full requirements over 4–7 days.** Restrict sodium and fluid, and avoid rapid volume expansion.
6. **Monitoring and treatment.** Check **potassium, phosphate, magnesium, calcium and glucose before feeding, then daily for the first 3 days and on alternate days for the first week**. **Correct electrolytes concurrently but do not delay feeding to do so** — replace phosphate (intravenously if <0.6 mmol/L), potassium approximately 2–4 mmol/kg/day and magnesium 0.2–0.4 mmol/kg/day. Monitor fluid balance, daily weight and ECG; reduce or temporarily hold the feed if severe biochemical disturbance or cardiac failure develops.

Latest Update (2023–2026) — 1 point

7. **ASPEN consensus recommendations for refeeding syndrome** (*Nutr Clin Pract/JPEN* 2020) — provided the first standardised **definition and severity grading**: refeeding syndrome is a fall in serum phosphate, potassium and/or magnesium of **10–20% (mild), 20–30% (moderate), or >30% with organ dysfunction (severe)**, occurring within **5 days** of restarting or substantially increasing nutrition. It recommends starting with **100–150 g of dextrose or 10–20 kcal/kg in the first 24 hours, advancing by about 33% of the goal every 1–2 days**, with **thiamine given before feeding** and continued for 5–7 days or longer in high-risk patients.

Question 20. Inanition 7 MARKS

Answer — Textbook Core (6 points)

1. **Definition.** Inanition is a state of **profound exhaustion and wasting resulting from prolonged inadequate intake, absorption or utilisation of food** — the advanced end-stage of starvation, with loss of both fat and muscle mass, weakness and multi-organ dysfunction. In geriatric practice it overlaps closely with the **"failure to thrive" syndrome of the elderly**.
2. **Distinction from related states (very high yield).** **Inanition/starvation** — pure nutrient deficiency, largely **reversible with feeding**. **Cachexia** — inflammation-driven, cytokine-mediated (**TNF-α, IL-1, IL-6**) loss of muscle in cancer, heart failure, COPD or CKD, **not fully reversible by nutrition alone**. **Sarcopenia** — age-related loss of muscle mass and strength. **Anorexia of ageing** — the physiological decline in appetite with early satiety, delayed gastric emptying and altered gut hormones.
3. **Causes in the elderly — the "9 Ds".** **Dementia, Depression, Dysphagia, Dentition, Drugs, Disease (malignancy, infection, hyperthyroidism, heart failure), Diarrhoea/malabsorption, Dysgeusia and Destitution (poverty and social isolation)**. Also immobility, alcohol misuse, restrictive therapeutic diets, and **elder abuse or neglect**.
4. **Clinical features and consequences.** Severe weight loss with **temporal and interosseous muscle wasting**, hypothermia, bradycardia and hypotension, **sarcopenia with falls and fractures**, **pressure ulcers**, impaired cell-mediated immunity with infection, delayed wound healing, **oedema from hypoalbuminaemia**, apathy, depression and cognitive slowing, and greatly increased mortality and rates of institutionalisation.
5. **Assessment.** **Screen with the MNA-SF (or MUST)**; document weight, BMI, percentage unintentional weight loss and calf circumference. Apply the **GLIM criteria — at least one phenotypic criterion (weight loss, low BMI, reduced muscle mass) plus one aetiologic criterion (reduced intake or absorption, or disease burden with inflammation)**. Measure **hand-grip strength**. Investigate with haemoglobin, electrolytes, calcium, vitamin D, B_{12}, thyroid function, CRP and albumin, and screen for malignancy, depression, dysphagia and oral disease. **A comprehensive geriatric assessment with medication review is mandatory.**
6. **Management.** **Treat reversible causes first** — dental repair, dysphagia therapy with texture modification and speech therapist input, treatment of depression (**mirtazapine is useful where weight gain is desirable**), and

withdrawal of appetite-suppressing drugs (digoxin, SSRIs, metformin, opioids, anticholinergics). Then apply a **"food-first" approach** — food fortification, small frequent energy-dense meals, **protein 1.2–1.5 g/kg/day**, feeding assistance, improved mealtime environment and social eating; add **oral nutritional supplements between (not with) meals**. Combine with **resistance exercise** to convert intake into muscle, correct vitamin D and micronutrient deficiency, and consider tube feeding only after explicit goal-setting — **it should be avoided in advanced dementia**.

Latest Update (2023–2026) — 1 point

7. **Two changes that reverse older teaching.** First, **megestrol acetate and other appetite stimulants are now to be avoided** in older adults (**AGS Beers Criteria 2023**) — they produce minimal weight gain with **no benefit on function or survival**, and carry risks of thrombosis, adrenal suppression, fluid retention and death. Second, under the **GLIM framework and current ESPEN geriatric guidance, serum albumin is no longer accepted as a nutritional marker** and has been reclassified as an **inflammatory marker**; the diagnosis of malnutrition must be made on phenotypic and aetiologic criteria with objective assessment of muscle mass.

Current Practice Update

Textbook: Low serum albumin indicates malnutrition; megestrol acetate may be used as an orexigenic agent.
Current recommendation: Albumin is an inflammatory marker and is excluded from GLIM criteria; megestrol is listed as potentially inappropriate in the AGS Beers Criteria 2023.
Exam approach: These are two of the highest-yield "textbook is outdated" lines in the whole nutrition syllabus — state both explicitly.

PART IV — ANAEMIA AND TRANSFUSION

Question 21. Anaemia in the elderly and the evaluation of anaemia 7 MARKS

Answer — Textbook Core (6 points)

1. **Definition and magnitude.** Anaemia is defined by WHO as **haemoglobin below 13 g/dL in men and below 12 g/dL in women**. Prevalence rises steeply with age — about **10% over 65 years and over 20–25% beyond 85 years**, and higher still in nursing homes and hospital inpatients. **Anaemia is never a normal consequence of ageing and must always be investigated.**
2. **Why it matters.** Even mild anaemia in older people is independently associated with **fatigue and reduced quality of life, falls, frailty and sarcopenia, cognitive decline, prolonged hospital stay, poorer surgical outcomes and increased mortality**. It also decompensates existing coronary artery disease, heart failure and cerebrovascular disease.
3. **The classical "rule of thirds" (NHANES).** Approximately one third is due to **nutritional deficiency (iron, B_{12}, folate)**; one third to **anaemia of chronic disease and chronic kidney disease**; and one third is **"unexplained anaemia of ageing"** — attributed to blunted erythropoietin response, low-grade inflammation, reduced marrow reserve, testosterone decline and occult clonal haematopoiesis or early myelodysplasia.
4. **Morphological approach by MCV.** **Microcytic (MCV <80 fL)** — iron deficiency, thalassaemia trait, sideroblastic anaemia, some anaemia of chronic disease. **Normocytic (80–100 fL)** — anaemia of chronic disease, CKD, acute blood loss, haemolysis, marrow infiltration, endocrine disease. **Macrocytic (>100 fL)** — B_{12} and folate deficiency, alcohol, hypothyroidism, liver disease, myelodysplasia, drugs (methotrexate, hydroxyurea, zidovudine).
5. **First-line evaluation.** History (bleeding, diet, alcohol, drugs including NSAIDs and aspirin, weight loss, comorbidity) and examination for lymphadenopathy, organomegaly and per-rectal examination. Tests: **complete blood count with indices and RDW, peripheral smear, reticulocyte count, ferritin with transferrin saturation, B_{12} and folate, renal function, LFT, thyroid function, CRP/ESR**. The **reticulocyte count is the key branch point — high suggests haemolysis or blood loss, low suggests production failure**.
6. **Second-line evaluation and treatment principles.** **Bidirectional endoscopy in every older adult with unexplained iron deficiency**; serum protein electrophoresis with free light chains and urine Bence-Jones protein if there is a raised ESR, renal impairment, bone pain or rouleaux; haemolytic screen (LDH, bilirubin, haptoglobin, direct antiglobulin test) if reticulocytes are high; and **bone marrow aspiration and biopsy if there is pancytopenia, blasts, dysplasia or a persistently unexplained anaemia**. **Treat the cause rather than transfusing** — replace iron, B_{12} or folate; use erythropoiesis-stimulating agents only in CKD or MDS with a target Hb of 10–11.5 g/dL (never normalise); and reserve transfusion for symptomatic anaemia at Hb <7 g/dL (<8 g/dL with cardiovascular disease).

Latest Update (2023–2026) — 1 point

7. **WHO revised haemoglobin thresholds (2024)** — a technical review reaffirmed the diagnostic cut-offs but recommended **adjustment for smoking and altitude**, and emphasised that **ferritin below 30 µg/L (rather than 15) should be used to identify iron deficiency in the presence of inflammation**. In parallel, "unexplained anaemia of ageing" is increasingly attributed to **clonal haematopoiesis of indeterminate potential (CHIP)** and early clonal cytopenia, which reframes it as a pre-malignant rather than purely physiological state and justifies bone marrow evaluation in persistent unexplained anaemia.

Question 22. Iron deficiency anaemia — red cell indices and investigation 7 MARKS

Answer — Textbook Core (6 points)

1. **Definition.** Anaemia (**WHO: Hb <13 g/dL in men, <12 g/dL in women**) resulting from depleted body iron stores. It is the commonest anaemia worldwide and the commonest nutritional anaemia in Indian elderly. **In an older adult, iron deficiency means blood loss until proved otherwise.**
2. **Causes in the elderly.** **Chronic gastrointestinal blood loss** — peptic ulcer, NSAID/aspirin use, **colonic carcinoma**, angiodysplasia, oesophagitis and hookworm infestation (important in India); **malabsorption** — coeliac disease, atrophic gastritis, post-gastrectomy and long-term PPI use; poor dietary intake; and genitourinary blood loss.
3. **Red cell indices.** **Low MCV (<80 fL) and low MCH (<27 pg)** giving a microcytic hypochromic picture, with a **raised RDW (>15%) — the earliest index to change**. This distinguishes it from **thalassaemia trait**, which has a

normal RDW, a disproportionately low MCV and a high red cell count (**Mentzer index MCV/RBC >13 favours iron deficiency**). **Reticulocyte haemoglobin content (CHr/Ret-He) is an early marker.** The film shows microcytes, hypochromia, **pencil cells**, target cells and mild thrombocytosis.

4. **Confirmatory biochemistry.** **Serum ferritin is the single best test — below 30 ng/mL is diagnostic**, but because ferritin is an acute-phase reactant a higher cut-off of **45–100 ng/mL** is used in the elderly with inflammation. Also: low serum iron, **raised TIBC**, **transferrin saturation <16%**, raised soluble transferrin receptor and low hepcidin. Bone marrow iron staining is needed only when the diagnosis remains unclear.
5. **Distinction from anaemia of chronic disease.** Anaemia of chronic disease shows a **normal or high ferritin, low TIBC, normal soluble transferrin receptor and raised CRP**. **Combined states are common in the elderly** — use the sTfR/log-ferritin index to separate them.
6. **Investigation of the source.** **Bidirectional endoscopy (upper GI endoscopy plus colonoscopy) is mandatory in every older adult with unexplained iron deficiency anaemia**, with duodenal biopsy and coeliac serology. Also check urine for blood and stool for occult blood and ova. If both are negative and anaemia persists or recurs, proceed to **capsule endoscopy or CT enterography** for small bowel angiodysplasia or tumour.

Latest Update (2023–2026) — 1 point

7. **Modern iron replacement.** Hepcidin studies have shown that **oral iron given on alternate days as a single morning dose achieves better fractional absorption with fewer side-effects** than conventional twice or thrice daily dosing. New-generation intravenous preparations — **ferric carboxymaltose and ferric derisomaltose** — allow total-dose correction in a single sitting and are preferred in malabsorption, intolerance, CKD, heart failure and ongoing blood loss; watch for **hypophosphataemia with ferric carboxymaltose**.

Cross-reference: Anaemia of chronic disease, with which iron deficiency frequently coexists, is covered in Question 24.

Question 23. Megaloblastic anaemia and B_{12} deficiency 15 MARKS

Answer — Textbook Core (10 points)

1. **Definition.** A **macrocytic anaemia caused by impaired DNA synthesis** from vitamin B_{12} (cobalamin) or folate deficiency, producing **nuclear-cytoplasmic asynchrony** and ineffective erythropoiesis.
2. **Physiology of B_{12} absorption.** Dietary B_{12} binds R-protein, is released by pancreatic proteases, binds **intrinsic factor from gastric parietal cells**, and is absorbed in the **terminal ileum**. **Body stores last 3–5 years, whereas folate stores last only 3–4 months.**
3. **Causes of B_{12} deficiency in the elderly.** **Food-cobalamin malabsorption from atrophic gastritis and hypochlorhydria is the commonest cause in old age**; **pernicious anaemia** (autoimmune, with anti-intrinsic factor and anti-parietal cell antibodies); gastrectomy or bariatric surgery; terminal ileal disease or resection; bacterial overgrowth; tropical sprue; strict vegetarian diet (very relevant in India); and drugs — **metformin, proton pump inhibitors, H_2 blockers, colchicine and nitrous oxide**.
4. **Causes of folate deficiency.** Poor intake and alcoholism, malabsorption (coeliac disease, tropical sprue), increased demand (haemolysis, malignancy, pregnancy), and drugs — **methotrexate, trimethoprim, phenytoin and sulfasalazine**.
5. **General clinical features.** Insidious anaemia with **lemon-yellow pallor** and mild jaundice, **glossitis (beefy red smooth tongue)**, angular stomatitis, anorexia and weight loss; frail patients may present with heart failure.
6. **Neurological features (B_{12} only, and may occur without anaemia).** **Subacute combined degeneration of the cord** — symmetrical paraesthesiae, loss of vibration and joint position sense, sensory ataxia, and **spastic paraparesis with extensor plantars but absent ankle jerks**. Also peripheral neuropathy, optic atrophy, dementia, depression and psychosis ("megaloblastic madness"). **Folate deficiency does not cause subacute combined degeneration.**
7. **Laboratory findings.** **Raised MCV (often >110 fL)**, **oval macrocytes** and **hypersegmented neutrophils** (5% with five lobes or any six-lobed cell), pancytopenia when severe, and a low reticulocyte count. Ineffective erythropoiesis gives a **raised LDH and unconjugated bilirubin with low haptoglobin**, mimicking haemolysis or TTP (**pseudo-thrombotic microangiopathy**). Marrow shows megaloblasts and **giant metamyelocytes**.
8. **Confirming the deficiency.** **Serum B_{12} <200 pg/mL (148 pmol/L)** is diagnostic; for indeterminate values of 200–350 pg/mL measure **serum methylmalonic acid (raised in B_{12} deficiency only)** and **homocysteine (raised in both B_{12} and folate deficiency)**. **Holotranscobalamin (active B_{12})** is an alternative first-line test. Add serum and red cell folate, **anti-intrinsic factor antibody (highly specific, poorly sensitive)** and anti-parietal cell antibody,

and gastroscopy for atrophic gastritis (risk of gastric cancer and carcinoid).

9. **Treatment.** **Hydroxocobalamin 1000 µg intramuscularly on alternate days for 1–2 weeks, then 1000 µg every 2–3 months for life.** If there are neurological features, continue alternate-day injections until no further improvement occurs, then maintain 2-monthly. **Oral cyanocobalamin 1000–2000 µg daily** is effective for dietary deficiency and food-cobalamin malabsorption. Folate deficiency is treated with **folic acid 5 mg daily**.
10. **Cautions and monitoring.** **Never give folate alone when B_{12} deficiency is possible — it precipitates or worsens subacute combined degeneration.** Expect a **reticulocytosis at day 5–7** and a rising haemoglobin within 1–2 weeks. Watch for **hypokalaemia** during the response, and for **unmasked iron deficiency** (falling MCV with a dimorphic film). Screen for thyroid disease and other autoimmune conditions in pernicious anaemia.

Latest Updates (2023–2026) — 2 points

11. **NICE NG239 (March 2024) — Vitamin B_{12} deficiency in over-16s.** Test using either total B_{12} or **active B_{12} (holotranscobalamin)**; use **serum methylmalonic acid for indeterminate results**; **do not delay replacement while awaiting results if there are neurological symptoms or megaloblastic anaemia**; and offer **lifelong intramuscular replacement** after total gastrectomy, complete terminal ileal resection, or when autoimmune gastritis is the cause.
12. **Metformin-associated B_{12} deficiency** is now formally classified as a **common adverse effect** by regulators, with periodic B_{12} testing advised in long-term metformin users, particularly those with anaemia or neuropathy. Evidence also now supports **high-dose oral B_{12} as non-inferior to intramuscular therapy** in non-neurological deficiency without severe malabsorption.

Question 24. Anaemia of chronic disease 7 MARKS

Answer — Textbook Core (6 points)

1. **Definition.** Also called **anaemia of inflammation** — a mild to moderate, usually **normocytic normochromic** anaemia occurring in chronic infection, inflammation or malignancy. It is the **second commonest anaemia worldwide and the commonest anaemia in hospitalised older patients**.
2. **Causes.** **Chronic infections** (tuberculosis, osteomyelitis, endocarditis, HIV), **chronic inflammatory disease** (rheumatoid arthritis, polymyalgia rheumatica, giant cell arteritis, inflammatory bowel disease), **malignancy**, chronic kidney disease, heart failure, COPD and diabetes.
3. **Pathogenesis — three mechanisms.** Inflammatory cytokines (**IL-6, TNF-α, IL-1, interferon-γ**) drive: (a) **IL-6-induced hepcidin release from the liver, which degrades ferroportin and traps iron in macrophages and enterocytes — producing functional iron deficiency**; (b) **blunted erythropoietin production and reduced marrow responsiveness to EPO**; and (c) **shortened red cell survival**. Hepcidin is the central molecule.
4. **Laboratory features.** Hb usually **8–11 g/dL**, normocytic (microcytic in 25–30% if long-standing), **low reticulocyte count**, **low serum iron with low TIBC/transferrin**, transferrin saturation low or normal, **normal or high ferritin (an acute-phase reactant)**, raised CRP and ESR, and **normal soluble transferrin receptor**.
5. **Distinction from iron deficiency (very high yield).**

Parameter	Iron deficiency	Anaemia of chronic disease
Serum ferritin	**Low** (<30 ng/mL)	**Normal or high**
TIBC / transferrin	**High**	**Low**
Transferrin saturation	<16%	Low or normal
Soluble transferrin receptor	**High**	**Normal**
sTfR / log ferritin index	>2	<1
Serum hepcidin	Low	**High**

The two frequently coexist in the elderly — use the sTfR/log ferritin index, or a ferritin cut-off of 100 ng/mL in the presence of inflammation.

6. **Management.** **Treat the underlying disease — this is the definitive therapy and the anaemia resolves with disease control.** Do not give oral iron unless true deficiency coexists, because **high hepcidin blocks its absorption**; if iron is needed, use **intravenous iron, which bypasses the hepcidin-ferroportin block**. Erythropoiesis-stimulating agents are indicated in **CKD and chemotherapy-induced anaemia**, targeting Hb 10–

11.5 g/dL (never normalisation, because of thrombosis, hypertension and tumour progression risk), with iron repletion first. Transfuse only for symptomatic anaemia.

Latest Update (2023–2026) — 1 point

7. **Hepcidin-targeted and HIF-PHI therapy. Hypoxia-inducible factor prolyl-hydroxylase inhibitors (roxadustat, daprodustat, vadadustat)** are now approved for anaemia of CKD; they are **orally active, raise endogenous erythropoietin and simultaneously suppress hepcidin**, thereby improving iron mobilisation — a mechanistic advantage over conventional ESAs in inflammatory states, although cardiovascular safety signals mean current guidance restricts them to selected patients. Anti-hepcidin and anti-IL-6 agents are in trials for anaemia of inflammation.

Current Practice Update

Textbook: Ferritin below 15–30 ng/mL defines iron deficiency; oral iron is first-line replacement.
Current recommendation: In inflammation use a ferritin threshold of 100 ng/mL (or sTfR index), and give **intravenous** iron, since high hepcidin blocks oral absorption.
Exam approach: State the hepcidin mechanism explicitly — it explains both the diagnostic difficulty and why IV iron is preferred.

Cross-reference: The general evaluation of anaemia in an older patient is given in Question 21, and iron deficiency in Question 22.

Question 25. Blood transfusion in the older adult — thresholds and hazards 7 MARKS

Answer — Textbook Core (6 points)

1. **Guiding principle.** Transfuse for **symptoms and physiology, not for a number**. Give a **single unit and then reassess** ("why give two when one will do"), and always consider treating the cause — iron, B_{12}, folate or erythropoietin — instead of transfusing.
2. **Thresholds (AABB 2023 international guidelines).** **Restrictive threshold of Hb <7 g/dL** for haemodynamically stable hospitalised adults, including critical care patients and those with gastrointestinal bleeding. **Hb <8 g/dL** for patients undergoing orthopaedic or cardiac surgery and those with pre-existing cardiovascular disease.
3. **Special situations.** **Chronic asymptomatic anaemia should not be transfused** — correct the cause. In active bleeding, transfuse by clinical judgement rather than haemoglobin. In heart failure and frailty, transfuse **slowly over 3–4 hours per unit with diuretic cover**.
4. **Acute hazards.** **Acute haemolytic reaction from ABO incompatibility (commonest cause is a clerical/identification error)**, febrile non-haemolytic reaction, allergic and anaphylactic reactions, bacterial contamination and sepsis, **TACO — transfusion-associated circulatory overload, the commonest cause of transfusion-related death and the commonest serious hazard in the elderly** — and **TRALI** (non-cardiogenic pulmonary oedema within 6 hours).
5. **Delayed hazards.** Delayed haemolytic reaction, red cell alloimmunisation, **transfusion-transmitted infection (HIV, hepatitis B and C, malaria, syphilis — relevant to Indian practice)**, iron overload with repeated transfusion, **transfusion-associated graft-versus-host disease** (prevent with irradiated components after purine analogues or stem cell transplant), and post-transfusion purpura.
6. **Safe practice.** **Positive patient identification at the bedside**, baseline and 15-minute observations, transfusion during daytime hours wherever possible, documented indication and consent, strict fluid balance monitoring, and a **formal TACO risk assessment before every transfusion in an older patient**.

Latest Update (2023–2026) — 1 point

7. **AABB 2025 international guideline for acute myocardial infarction** (*Ann Intern Med* 2025;178:1469) — in hospitalised patients with **acute myocardial infarction and anaemia, a liberal strategy transfusing at Hb <10 g/dL is now suggested**. This follows the **MINT trial (NEJM 2023, n=3504, mean age 72)** and a patient-level meta-analysis showing lower cardiac death at 30 days and lower all-cause mortality at 6 months with the liberal strategy. **It is the first major departure from universal restrictive transfusion.**

Current Practice Update

Textbook: A restrictive threshold of 7–8 g/dL applies to all stable hospitalised patients, including those with cardiac disease.
Current recommendation: Restrictive 7 g/dL remains standard, but **acute myocardial infarction is now an exception — transfuse to keep Hb ≥10 g/dL**.
Exam approach: State the restrictive rule, then add the AABB 2025/MINT exception for acute MI — this single line distinguishes an updated answer.

Cross-reference: For transfusion practice in the specific setting of acute upper gastrointestinal bleeding, see also Question 3.

PART V — HAEMATOLOGICAL DISORDERS AND MALIGNANCY

Question 26. Thrombocytopenia 7 MARKS

Answer — Textbook Core (6 points)

1. **Definition and bleeding risk.** Platelet count below **150 × 10^9/L**. **Spontaneous bleeding is unusual above 50, purpura and mucosal bleeding appear below 30, and the risk of spontaneous serious or intracranial bleeding rises below 10–20 × 10^9/L.** Always **exclude pseudothrombocytopenia from EDTA-induced platelet clumping** by repeating the count in a citrate tube and examining the film.
2. **Classification by mechanism.** **Decreased production** — marrow failure or infiltration (leukaemia, myeloma, metastases, myelofibrosis), **myelodysplasia**, aplastic anaemia, B_{12}/folate deficiency, alcohol, chemotherapy and radiation. **Increased destruction** — immune (ITP, drug-induced, HIT, post-transfusion purpura, secondary to CLL, SLE, HIV, hepatitis C) or non-immune (DIC, TTP/HUS, mechanical valves, sepsis). **Sequestration** — hypersplenism from portal hypertension. **Dilutional** — massive transfusion.
3. **Drug-induced thrombocytopenia (high yield in the elderly). Heparin (HIT), quinine and quinidine, sulphonamides and co-trimoxazole, vancomycin, linezolid, rifampicin, valproate, carbamazepine, phenytoin, thiazides, glycoprotein IIb/IIIa inhibitors and chemotherapy.** Onset is typically 1–2 weeks after starting a new drug, and recovery follows within 5–7 days of withdrawal.
4. **Immune thrombocytopenia (ITP) in older adults.** A diagnosis of exclusion — **isolated thrombocytopenia with a normal blood film and no other cause**. In the elderly it is more often **secondary (to CLL, lymphoma, myelodysplasia, HIV, hepatitis C, *H. pylori*, SLE or drugs)** and carries a **higher bleeding risk**, so a **bone marrow examination should be performed in most patients over 60 to exclude myelodysplasia**. Antiplatelet antibody testing is not required.
5. **Emergency causes that must not be missed. Heparin-induced thrombocytopenia** — a fall of >50% at 5–10 days with **thrombosis, not bleeding**; assess with the **4Ts score**, stop all heparin (including flushes) and start a non-heparin anticoagulant (argatroban, fondaparinux); **never give platelet transfusions or warfarin alone**. **TTP** — microangiopathic haemolytic anaemia with fragmented red cells, fever, renal impairment and neurological signs from **ADAMTS13 deficiency**, requiring urgent **plasma exchange**. Also **DIC** and sepsis.
6. **Management. Treat the cause and stop the offending drug.** In ITP, treat only if the platelet count is **below 20–30 × 10^9/L or there is bleeding**: first-line is **corticosteroids (prednisolone 1 mg/kg, or dexamethasone 40 mg for 4 days — preferred in the elderly for shorter steroid exposure)** with **IVIG 1 g/kg for urgent rises**. Second-line: **thrombopoietin receptor agonists (eltrombopag, romiplostim, avatrombopag)**, rituximab, or splenectomy (less favoured in the elderly). **Platelet transfusion only for active bleeding or before invasive procedures** — it is contraindicated in HIT and TTP. Prophylactic transfusion threshold is 10 × 10^9/L in stable patients.

Latest Update (2023–2026) — 1 point

7. **Newer targeted therapy in ITP.** The **ADVANCE trials of efgartigimod** (a neonatal Fc receptor blocker that accelerates IgG degradation, *Lancet* 2023) and the **Bruton tyrosine kinase inhibitor rilzabrutinib** have both demonstrated durable platelet responses in chronic ITP, offering **steroid-sparing options that are particularly valuable in older patients**, in whom prolonged corticosteroids cause diabetes, osteoporosis, delirium and infection. Current practice increasingly moves to a **TPO receptor agonist as early second-line therapy rather than prolonged steroids**.

Question 27. Disseminated intravascular coagulation 7 MARKS

Answer — Textbook Core (6 points)

1. **Definition.** An **acquired syndrome of widespread intravascular activation of coagulation** with loss of localisation, causing **microvascular thrombosis with organ dysfunction and simultaneous consumption of platelets and clotting factors leading to bleeding**. It is **always secondary to an underlying disorder** — never a primary diagnosis.
2. **Causes. Sepsis (the commonest, especially Gram-negative)**; **malignancy** — adenocarcinoma of pancreas, prostate and stomach (chronic, thrombotic DIC) and **acute promyelocytic leukaemia** (fulminant bleeding); **trauma, burns, major surgery, head injury**; obstetric causes (abruption, amniotic fluid embolism); **transfusion reactions, snake envenomation, heat stroke, severe pancreatitis, aortic aneurysm and giant haemangioma**.

3. **Pathogenesis.** **Tissue factor exposure** and cytokine release trigger excess thrombin generation, while the natural anticoagulants (**antithrombin, protein C, tissue factor pathway inhibitor**) are consumed and **fibrinolysis is suppressed by raised PAI-1**. Fibrin deposition in the microvasculature causes ischaemic organ failure and **microangiopathic haemolysis**; ongoing consumption then produces the bleeding phase.
4. **Clinical features.** **Bleeding** — oozing from venepuncture and cannula sites, petechiae, ecchymoses, mucosal and gastrointestinal bleeding. **Thrombosis** — acral cyanosis, digital gangrene, purpura fulminans, and organ failure (renal, respiratory, hepatic, cerebral). Chronic compensated DIC in malignancy may present only as **migratory thrombophlebitis or venous thromboembolism**.
5. **Laboratory diagnosis and the ISTH score.** **Thrombocytopenia with a falling trend, prolonged PT and aPTT, low fibrinogen, and markedly raised D-dimer/fibrin degradation products**; the film shows **schistocytes**. The **ISTH overt DIC score** (in a patient with a predisposing condition) awards points for platelet count (<100 = 1, <50 = 2), fibrin marker elevation (moderate 2, strong 3), PT prolongation (≥3 s = 1, ≥6 s = 2) and fibrinogen <1 g/L (=1); **a score of 5 or more is compatible with overt DIC**. **Serial trends matter more than a single value**, and fibrinogen may be spuriously normal because it is an acute-phase reactant.
6. **Management.** **Vigorous treatment of the precipitating cause is the cornerstone** — antibiotics and source control in sepsis, delivery in obstetric DIC, ATRA in acute promyelocytic leukaemia. Give blood products only **if there is bleeding or a planned procedure, not to correct numbers**: **platelets if <50 × 10^9/L with bleeding (<20 if not bleeding but high risk), fresh frozen plasma 15 mL/kg for prolonged PT/aPTT, and cryoprecipitate or fibrinogen concentrate if fibrinogen <1.5 g/L**. **Therapeutic heparin is reserved for the predominantly thrombotic phenotype** (purpura fulminans, digital ischaemia, chronic DIC of malignancy); **prophylactic-dose heparin for VTE prevention is appropriate in non-bleeding patients**. Tranexamic acid is generally contraindicated except in hyperfibrinolytic states such as APL.

Latest Update (2023–2026) — 1 point

7. **Sepsis-induced coagulopathy (SIC) and earlier recognition.** Current ISTH guidance recommends using the **simpler two-step SIC score (platelet count, INR and SOFA score)** to detect coagulopathy **before overt DIC develops**, since by the time the ISTH overt DIC score reaches 5 the mortality is already very high. Practice has also moved towards **viscoelastic testing (ROTEM/TEG) and fibrinogen concentrate rather than empirical fresh frozen plasma** for targeted correction, and away from routine antithrombin or recombinant thrombomodulin, which have not shown consistent survival benefit.

Question 28. Myelodysplasia 7 MARKS

Answer — Textbook Core (6 points)

1. **Definition.** Myelodysplastic syndromes are **clonal haematopoietic stem cell disorders characterised by ineffective haematopoiesis — a hypercellular or normocellular marrow with peripheral cytopenias — morphological dysplasia, and a tendency to transform into acute myeloid leukaemia**. It is **predominantly a disease of old age**, with a median age of 70–75 and incidence rising to over 30 per 100,000 beyond 70 years.
2. **Aetiology.** Mostly **primary/de novo**, arising from age-related clonal haematopoiesis. **Secondary (therapy-related) MDS** follows **alkylating agents (5–7 years later, with del(5q)/del(7q)) and topoisomerase II inhibitors**, radiotherapy, and exposure to benzene, organic solvents and tobacco. Common somatic mutations: **SF3B1 (favourable, ring sideroblasts), TET2, ASXL1, DNMT3A, RUNX1, TP53 (adverse)**.
3. **Clinical features.** Often **discovered incidentally on a routine blood count**. Otherwise, symptoms of **anaemia (fatigue, dyspnoea, angina, falls, worsening heart failure)**, **recurrent infection from neutropenia and impaired neutrophil function**, and **bruising and bleeding from thrombocytopenia**. Splenomegaly is unusual except in CMML. Autoimmune phenomena (vasculitis, arthritis, pericarditis) occur in about 10%.
4. **Diagnosis.** Persistent unexplained cytopenia with a blood film showing **oval macrocytes, hypogranular neutrophils and the Pelger-Huët anomaly (bilobed nuclei), giant or hypogranular platelets, and circulating blasts**. **Bone marrow aspirate and trephine are essential** — dysplasia in ≥10% of cells in one or more lineages, **blasts <20% (20% or more defines AML)**, **ring sideroblasts on Perls stain**, and cytogenetics (**del(5q), monosomy 7, trisomy 8, complex karyotype**). **Always exclude the mimics first — B_{12} and folate deficiency, copper deficiency, alcohol, HIV, drugs and recent chemotherapy.**
5. **Classification and prognosis.** Classified by the WHO 5th edition (2022)/ICC by **blast percentage, lineage dysplasia, ring sideroblasts and defining genetic lesions — MDS with low blasts, MDS with SF3B1 mutation, MDS with del(5q), MDS with increased blasts, MDS with biallelic TP53 inactivation**. Prognosis uses the **Revised International Prognostic Scoring System (IPSS-R)** based on **cytogenetics, marrow blast**

percentage, haemoglobin, platelet count and neutrophil count, giving five risk groups from very low to very high, with median survival ranging from about **8.8 years to 0.8 years**.

6. **Management (risk-adapted).** **Lower-risk disease** — supportive care with transfusion and iron chelation if more than 20–25 units are transfused; **erythropoiesis-stimulating agents if serum EPO <500 mU/mL**; **lenalidomide for del(5q)**; **luspatercept for anaemia with ring sideroblasts/SF3B1 mutation**; immunosuppression in selected hypoplastic MDS. **Higher-risk disease** — **hypomethylating agents (azacitidine or decitabine)**, which improve survival and are well tolerated in the elderly, or intensive AML-type chemotherapy in the fit. **Allogeneic stem cell transplantation with reduced-intensity conditioning is the only curative option** and is feasible up to about 70–75 years in fit patients.

Latest Update (2023–2026) — 1 point

7. **Molecular prognostication and two new drugs.** The **IPSS-M (Molecular IPSS)** incorporates 31 gene mutations with clinical variables and **reclassifies about 46% of patients compared with IPSS-R**, most often upstaging them and thereby changing transplant timing. For lower-risk transfusion-dependent anaemia: the **COMMANDS trial** (*Lancet Haematol* 2024) showed **luspatercept is superior to epoetin alfa as first-line therapy**, and **imetelstat, a telomerase inhibitor, was FDA-approved in June 2024 (IMerge trial, *Lancet* 2024)** for transfusion-dependent lower-risk MDS after ESA failure, giving durable transfusion independence irrespective of ring sideroblast status.

Question 29. Chronic myeloid leukaemia — clinical features and management 7 MARKS

Answer — Textbook Core (6 points)

1. **Definition and cytogenetics.** A myeloproliferative neoplasm defined by the **Philadelphia chromosome — t(9;22)(q34;q11) — producing the BCR::ABL1 fusion gene, which encodes a constitutively active tyrosine kinase**. Median age at diagnosis is about 65 years.

2. **Clinical features.** **Up to 50% are asymptomatic and detected on a routine blood count.** Otherwise: fatigue, weight loss, night sweats, low-grade fever, **massive splenomegaly with early satiety, left hypochondrial dragging pain and splenic infarction**, bone tenderness over the sternum, gout from hyperuricaemia, and rarely **leucostasis (priapism, visual disturbance, confusion) when the white count exceeds 300 × 10^9/L**.

3. **Phases.** **Chronic phase** — blasts <10%, over 90% present here, indolent. **Accelerated phase** — blasts 10–19%, basophils ≥20%, persistent thrombocytopenia or thrombocytosis, clonal evolution. **Blast crisis** — **blasts ≥20% in blood or marrow, or an extramedullary blast proliferation**; two-thirds are myeloid and one-third lymphoid, and prognosis is poor.

4. **Laboratory diagnosis.** **Marked leucocytosis (often >100 × 10^9/L) with the full spectrum of immature granulocytes ("left shift"), a myelocyte bulge, and characteristic basophilia and eosinophilia**; thrombocytosis is common; **the leucocyte alkaline phosphatase score is characteristically low** (distinguishing it from a leukaemoid reaction). Confirm with **cytogenetics for the Philadelphia chromosome, FISH, and quantitative RT-PCR for BCR::ABL1 transcripts (which is also used for monitoring)**. Marrow shows granulocytic hyperplasia with a raised myeloid:erythroid ratio.

5. **Treatment.** **Tyrosine kinase inhibitors are the treatment of choice and have converted CML into a chronic disease with near-normal life expectancy.** First-line: **imatinib 400 mg daily**, or a second-generation agent (**dasatinib, nilotinib, bosutinib**) for higher-risk disease or faster response. **Hydroxyurea** is used briefly for cytoreduction and **leukapheresis for leucostasis**. Allopurinol and hydration prevent tumour lysis. **Allogeneic transplantation is now reserved for blast crisis or multiple TKI failure.** For **T315I mutation, use ponatinib or asciminib**.

6. **Monitoring, toxicity and treatment-free remission.** Monitor **BCR::ABL1 by RT-PCR on the International Scale every 3 months**. Milestones: **≤10% at 3 months, ≤1% at 6 months, and ≤0.1% (major molecular response, MMR or MR3) at 12 months**. In the elderly, watch TKI toxicity — **imatinib: oedema, cramps, cytopenias; dasatinib: pleural effusion; nilotinib: QT prolongation, hyperglycaemia and arterial occlusive events; ponatinib: arterial thrombosis** — and check drug interactions (CYP3A4). **Treatment-free remission may be attempted after at least 3 years of therapy with a sustained deep molecular response (MR4.5) for 2 years**, with monthly monitoring; about half remain in remission.

Latest Update (2023–2026) — 1 point

7. **Asciminib — ASC4FIRST trial** (*NEJM* 2024;391:885; FDA accelerated approval October 2024, NCCN category 1). Asciminib is the **first allosteric BCR::ABL1 inhibitor, which binds the ABL myristoyl pocket (a "STAMP" inhibitor) rather than the ATP site**. In newly diagnosed chronic-phase CML it produced a **major molecular response at 48 weeks in 68% versus 49% with investigator-selected TKIs**, with **markedly fewer dose reductions and discontinuations for toxicity** — a particular advantage in older patients with comorbidity. It is also effective against the **T315I** mutation.

Question 30. Chronic lymphocytic leukaemia 15 MARKS

Answer — Textbook Core (10 points)

1. **Definition.** A monoclonal proliferation of mature B lymphocytes, defined by **≥5 × 10^9/L clonal B lymphocytes in peripheral blood persisting for at least 3 months** with a characteristic immunophenotype. It is the **commonest leukaemia of older adults**, with a median age at diagnosis of about 70 years.
2. **Related entities.** **Small lymphocytic lymphoma (SLL)** — same disease with nodal involvement but fewer than 5 × 10^9/L circulating cells. **Monoclonal B-cell lymphocytosis (MBL)** — clonal cells without disease, progressing to CLL at 1–2% per year.
3. **Clinical features.** Often **asymptomatic and detected on a routine blood count**. Otherwise painless symmetrical **lymphadenopathy, splenomegaly and hepatomegaly**, B symptoms (fever, drenching night sweats, weight loss >10%), fatigue, and **recurrent infections from hypogammaglobulinaemia**.
4. **Complications.** **Autoimmune haemolytic anaemia** and immune thrombocytopenia (direct antiglobulin test positive), pure red cell aplasia, **infection — the leading cause of death**, second malignancies (especially skin), and **Richter's transformation** to diffuse large B-cell lymphoma in about 5% (rapid nodal enlargement, rising LDH, B symptoms).
5. **Diagnosis.** Peripheral smear shows **mature small lymphocytes with smudge (smear) cells**. **Flow cytometry is diagnostic: CD5+, CD19+, CD23+, CD200+, dim CD20 and dim surface immunoglobulin with kappa or lambda light-chain restriction.** Bone marrow examination is not required for diagnosis.
6. **Staging.** **Rai** — 0 lymphocytosis only; I plus lymphadenopathy; II plus spleno/hepatomegaly; **III anaemia (Hb <11 g/dL)**; **IV thrombocytopenia (<100 × 10^9/L)**. **Binet** — A fewer than 3 nodal areas; B three or more; C anaemia or thrombocytopenia.
7. **Prognostic markers.** Adverse — **unmutated IGHV**, **del(17p) or TP53 mutation (worst, chemotherapy-resistant)**, del(11q), complex karyotype, high ZAP-70, CD38 and β_2-microglobulin. Favourable — **isolated del(13q)** and mutated IGHV. The **CLL-IPI** combines TP53 status, IGHV, β_2-microglobulin, clinical stage and age.
8. **Indications to treat (iwCLL 2018).** **Progressive marrow failure** (worsening anaemia or thrombocytopenia), massive or progressive splenomegaly or lymphadenopathy, **lymphocyte doubling time under 6 months**, autoimmune cytopenia refractory to steroids, and constitutional symptoms. **Early asymptomatic disease is not treated — "watch and wait" is the standard of care and treating early does not prolong survival.**
9. **Treatment.** Targeted agents have replaced chemoimmunotherapy in older adults — **continuous BTK inhibitors (ibrutinib, acalabrutinib, zanubrutinib)** or **fixed-duration venetoclax (BCL-2 inhibitor) with obinutuzumab for 12 months (CLL14 trial)**. For **del(17p)/TP53** use a BTK inhibitor or venetoclax-based therapy — **never FCR**. Chlorambucil with obinutuzumab remains an option where targeted agents are unavailable.
10. **Supportive care in the elderly.** Annual influenza, pneumococcal and **recombinant (non-live) zoster** vaccination; **prophylactic immunoglobulin if recurrent infection with IgG <400 mg/dL**; tumour lysis prophylaxis during venetoclax ramp-up; monitor BTK inhibitors for **atrial fibrillation, hypertension and bleeding** (avoid combining with warfarin); and use **irradiated blood products** after purine analogue therapy.

Latest Updates (2023–2026) — 2 points

11. **AMPLIFY trial** (Brown, *NEJM* 2025;392:748; FDA approval February 2026) — **fixed-duration acalabrutinib plus venetoclax is the first all-oral, time-limited regimen for untreated CLL**, with 36-month progression-free survival of **76.5% versus 66.5%** for chemoimmunotherapy (HR 0.65). Patients with del(17p)/TP53 were excluded.
12. **Pirtobrutinib**, a **non-covalent BTK inhibitor** (BRUIN trial), is approved after failure of covalent BTK inhibitors, including in **BTK C481S resistance**. Practice is moving towards **MRD-guided fixed-duration therapy** instead of indefinite treat-to-progression, which is particularly valuable in older patients wanting time off therapy.

Current Practice Update

Textbook: Chlorambucil-based or FCR chemoimmunotherapy is first-line, chosen by fitness.
Current recommendation: Targeted therapy for all — continuous BTK inhibitor or fixed-duration venetoclax combination; chemoimmunotherapy is essentially obsolete.
Exam approach: Mention chemoimmunotherapy only as historical/resource-limited practice, then give the targeted-agent algorithm and TP53 exception.

Question 31. Myelofibrosis and the myeloproliferative neoplasms 15 MARKS

Answer — Textbook Core (10 points)

1. **Definition and classification.** Myeloproliferative neoplasms are **clonal haematopoietic stem cell disorders** with effective but dysregulated proliferation of one or more myeloid lineages. **Philadelphia-negative:** polycythaemia vera (PV), essential thrombocythaemia (ET) and **primary myelofibrosis (PMF)**. **Philadelphia-positive:** chronic myeloid leukaemia.
2. **Driver mutations.** **JAK2 V617F** in 95% of PV and 50–60% of ET/PMF; JAK2 exon 12 in PV; **CALR** in 20–25% of ET/PMF (better prognosis); **MPL** in about 5%. **Triple-negative PMF has the worst outcome.** High-molecular-risk mutations are **ASXL1, SRSF2, EZH2, IDH1/2 and U2AF1**.
3. **Polycythaemia vera.** Raised haematocrit (**>49% men, >48% women**) with a **low serum erythropoietin** and JAK2 mutation. Features are **aquagenic pruritus**, erythromelalgia, plethora and splenomegaly. **Thrombosis is the main cause of death.**
4. **Essential thrombocythaemia.** Sustained platelet count **≥450 × 10^9/L** with megakaryocytic proliferation. Causes microvascular symptoms and thrombosis, but paradoxically **bleeding when platelets exceed 1000 × 10^9/L (acquired von Willebrand disease)**.
5. **Primary myelofibrosis — clinical features.** **Progressive anaemia, massive splenomegaly** (dragging pain, early satiety, splenic infarction), **constitutional symptoms** (fever, night sweats, weight loss, cachexia), bone pain, extramedullary haematopoiesis and portal hypertension.
6. **Primary myelofibrosis — laboratory findings.** **Leucoerythroblastic blood picture with teardrop poikilocytes (dacrocytes)**, nucleated red cells and immature granulocytes; raised LDH; **"dry tap" on marrow aspiration**; trephine biopsy shows **atypical clustered megakaryocytes with reticulin or collagen fibrosis (MF grade 1–3)**. WHO 5th edition (2022) separates **prefibrotic from overt PMF**.
7. **Prognostic scoring.** **DIPSS/DIPSS-Plus** — age >65, Hb <10 g/dL, WBC >25 × 10^9/L, circulating blasts ≥1%, constitutional symptoms, plus transfusion need, platelets <100 × 10^9/L and unfavourable karyotype. **MIPSS70/70+ v2.0** incorporates molecular data and is used to select transplant candidates.
8. **Complications.** Progressive marrow failure and transfusion dependence, portal hypertension, splenic infarction, thrombosis, infection, and **leukaemic transformation to AML in 10–20% at 10 years**.
9. **Management of PV and ET.** **Low-dose aspirin 75–100 mg for all** unless contraindicated. PV — **venesection to keep haematocrit below 45%**. Cytoreduction if high risk (**age >60 or prior thrombosis**) — **hydroxyurea first-line**, interferon in younger patients, and **ruxolitinib for hydroxyurea-resistant or intolerant PV**. In ET, anagrelide is second-line.
10. **Management of primary myelofibrosis.** Risk-adapted — observe low-risk asymptomatic disease. For symptomatic splenomegaly or constitutional symptoms use the JAK inhibitor **ruxolitinib** (COMFORT-I and II: spleen and symptom reduction with a survival benefit); **fedratinib** after ruxolitinib failure. **Allogeneic stem cell transplantation is the only curative option** but is rarely feasible beyond 70 years. Supportive care includes transfusion, erythropoiesis-stimulating agents if serum EPO <500 mU/mL, danazol, splenic irradiation, and splenectomy only for refractory cases.

Latest Updates (2023–2026) — 2 points

11. **Cytopenia-directed JAK inhibitors** — **momelotinib** (MOMENTUM, *Lancet* 2023) is approved for myelofibrosis with anaemia, improving spleen size, symptoms and **transfusion independence** through additional ACVR1/hepcidin inhibition; **pacritinib** is approved when platelets are **below 50 × 10^9/L**. NCCN MPN guidelines 2026 now contain a dedicated MF-associated anaemia algorithm listing momelotinib as a preferred regimen.
12. **Ropeginterferon alfa-2b** is approved for polycythaemia vera as a **disease-modifying agent that induces molecular responses** and reduces JAK2 allele burden, unlike hydroxyurea. The hepcidin mimetic **rusfertide** is in late-phase trials aiming to abolish phlebotomy dependence.

Question 32. Plasma cell dyscrasias — types, presentation, diagnostic criteria, course and treatment 15 MARKS

Answer — Textbook Core (10 points)

1. **Definition and the spectrum.** Plasma cell dyscrasias (monoclonal gammopathies) are **clonal proliferations of plasma cells or lymphoplasmacytic cells producing a monoclonal immunoglobulin (M protein or paraprotein)**. The spectrum runs from **MGUS → smouldering myeloma → symptomatic multiple myeloma**, with related entities of **solitary plasmacytoma, AL amyloidosis, Waldenström macroglobulinaemia, POEMS syndrome, heavy chain disease, plasma cell leukaemia and monoclonal gammopathy of renal significance (MGRS)**.
2. **MGUS. Serum M protein <3 g/dL, clonal marrow plasma cells <10%, and no myeloma-defining events or end-organ damage.** It is present in **3% of people over 50 and 5% over 70**, is asymptomatic, and progresses at a fixed rate of about **1% per year, indefinitely**. Risk factors for progression are non-IgG isotype, M protein ≥1.5 g/dL and an abnormal free light chain ratio. **Management is observation only — no treatment**, with 6-monthly then annual monitoring.
3. **Smouldering (asymptomatic) myeloma. M protein ≥3 g/dL or urinary M protein ≥500 mg/24 h, and/or clonal marrow plasma cells 10–60%, but no myeloma-defining events.** Risk of progression is much higher — about **10% per year for the first 5 years**. Standard care is observation, but **high-risk smouldering myeloma (the "20-2-20" criteria: M protein >2 g/dL, marrow plasma cells >20%, free light chain ratio >20) is now often treated with lenalidomide**.
4. **Multiple myeloma. Clonal marrow plasma cells ≥10% or a biopsy-proven plasmacytoma, plus either end-organ damage (CRAB) or a myeloma-defining biomarker (SLiM).** Presents with bone pain, pathological fracture, anaemia, renal failure, hypercalcaemia and recurrent infection. Median survival is now 7–10 years with modern therapy.
5. **Solitary plasmacytoma and plasma cell leukaemia. Solitary plasmacytoma** — a single bone or extramedullary lesion with a normal marrow, no CRAB and a negative skeletal survey/MRI; treated with **radical radiotherapy (40–50 Gy)**, though most bone plasmacytomas progress to myeloma within 3 years. **Plasma cell leukaemia** — ≥5% circulating plasma cells (revised from 20%); aggressive, requiring intensive combination therapy.
6. **AL (primary) amyloidosis.** Deposition of **misfolded monoclonal free light chains (usually lambda)** as amyloid fibrils. Presents with **nephrotic syndrome, restrictive cardiomyopathy with heart failure and raised NT-proBNP, hepatomegaly, peripheral and autonomic neuropathy, macroglossia and periorbital purpura (the two pathognomonic signs)**. Diagnosis requires **Congo red staining with apple-green birefringence** on abdominal fat pad, rectal or organ biopsy, plus mass spectrometry typing and demonstration of a clone. Prognosis is driven by cardiac stage (NT-proBNP and troponin).
7. **Waldenström macroglobulinaemia.** A **lymphoplasmacytic lymphoma secreting IgM**, with **MYD88 L265P mutation in over 90%**. Presents with **hyperviscosity (headache, blurred vision, retinal vein engorgement, confusion, bleeding)**, lymphadenopathy, hepatosplenomegaly, anaemia, **peripheral neuropathy (anti-MAG)**, cryoglobulinaemia and cold agglutinin disease. **Bone lesions and hypercalcaemia are characteristically absent** — a key contrast with myeloma. Treated with **plasmapheresis for symptomatic hyperviscosity**, then rituximab-based therapy or a **BTK inhibitor (ibrutinib, zanubrutinib)**.
8. **POEMS syndrome and MGRS. POEMS** — **P**olyneuropathy, **O**rganomegaly, **E**ndocrinopathy, **M**onoclonal protein (almost always lambda) and **S**kin changes, with **sclerotic bone lesions, raised VEGF, Castleman disease and papilloedema**; treated with radiotherapy to an isolated lesion or systemic therapy with autologous transplant. **MGRS** — a small clone not meeting myeloma criteria that nevertheless causes renal damage (amyloidosis, light chain deposition disease, cryoglobulinaemic glomerulonephritis); **it must be treated despite the low tumour burden, because the criterion is organ damage, not clone size**.

9. **Diagnostic workup common to all.** **Serum protein electrophoresis with immunofixation, serum free light chain assay with kappa:lambda ratio, 24-hour urine electrophoresis for Bence-Jones protein, quantitative immunoglobulins**; complete blood count, calcium, creatinine, albumin, LDH and β_2-microglobulin; **bone marrow aspirate and trephine with flow cytometry, FISH for cytogenetics**; and **whole-body low-dose CT, whole-body MRI or PET-CT — skeletal survey is now obsolete**. Add echocardiography and NT-proBNP if amyloidosis is suspected.
10. **Principles of treatment.** **MGUS and standard-risk smouldering myeloma are observed; symptomatic disease is treated.** Modern therapy combines a **proteasome inhibitor (bortezomib) + immunomodulatory drug (lenalidomide) + anti-CD38 monoclonal antibody (daratumumab) + dexamethasone**, with **autologous stem cell transplant in fit patients up to about 70–75 years** and lenalidomide maintenance. Supportive care is equally examinable: **bisphosphonates or denosumab, radiotherapy for bone pain and cord compression, hydration and avoidance of nephrotoxins, VTE prophylaxis with immunomodulatory drugs, antiviral prophylaxis with bortezomib and daratumumab, vaccination and immunoglobulin replacement**. In older patients, **dose-adapt using frailty scores (IMWG frailty index, R-MCI) rather than chronological age**.

Latest Updates (2023–2026) — 2 points

11. **MRD negativity as a regulatory endpoint.** In April 2024 the FDA's advisory committee accepted **minimal residual disease negativity (10^{-5} by next-generation sequencing or flow cytometry) as a surrogate endpoint for accelerated approval in myeloma** — the first haematological malignancy for which this applies. Risk stratification has correspondingly moved to the **R2-ISS**, which combines ISS stage with LDH, del(17p), t(4;14) and 1q gain.
12. **Immunotherapy has transformed relapsed disease. BCMA-directed CAR T-cell therapy (idecabtagene vicleucel, ciltacabtagene autoleucel)** and **bispecific T-cell engagers (teclistamab targeting BCMA, talquetamab targeting GPRC5D, elranatamab)** now produce deep responses in heavily pre-treated myeloma, and bispecifics are **"off-the-shelf" and therefore more feasible in older and frailer patients** than CAR-T; the principal toxicities are **cytokine release syndrome, neurotoxicity (ICANS) and profound infection risk requiring immunoglobulin replacement**.

Cross-reference: Multiple myeloma is expanded separately in Question 33; this answer covers the whole spectrum of monoclonal gammopathies.

Question 33. Multiple myeloma — aetiology, diagnosis and management 15 MARKS

Answer — Textbook Core (10 points)

1. **Definition and epidemiology.** A malignant clonal proliferation of **bone marrow plasma cells** producing a monoclonal immunoglobulin, with resultant lytic bone disease, marrow failure and renal impairment. It accounts for about **10% of haematological malignancies**; **median age at diagnosis is 70 years**, and **over a third of patients are above 75**.
2. **Aetiology and risk factors.** **All myeloma is preceded by MGUS**, which progresses at 1% per year. Risk factors: **increasing age, male sex, African ancestry, family history, obesity**, and occupational exposure to **ionising radiation, benzene, pesticides and organic solvents**. Pathogenesis involves **primary IgH translocations — t(11;14), t(4;14), t(14;16)** — or hyperdiploidy, with secondary events (**del(17p)/TP53, 1q gain, MYC rearrangement, RAS mutations**) driving progression, all supported by the bone marrow microenvironment (IL-6, RANKL, VEGF).
3. **Clinical features — the CRAB criteria.** **C** — hyper**C**alcaemia (corrected calcium >11 mg/dL or >1 mg/dL above normal); **R** — **R**enal impairment (creatinine >2 mg/dL or eGFR <40 mL/min); **A** — **A**naemia (Hb <10 g/dL or 2 g/dL below normal); **B** — **B**one lesions (one or more lytic lesions on CT, MRI or PET-CT).
4. **Other presentations.** **Bone pain, especially back pain, and pathological fracture**; **recurrent bacterial infection from immune paresis (the commonest cause of death)**; **spinal cord compression**; hyperviscosity; **amyloidosis**; peripheral neuropathy; and bleeding. **In older patients, myeloma frequently presents as unexplained anaemia, fatigue, back pain attributed to osteoporosis, or acute kidney injury.**
5. **Myeloma-defining biomarkers (SLiM criteria, IMWG 2014).** These allow treatment **before end-organ damage occurs**: **S** — clonal marrow plasma cells **≥60%**; **Li** — involved:uninvolved serum free **Li**ght chain ratio **≥100** (with involved chain ≥100 mg/L); **M** — more than one focal lesion ≥5 mm on **M**RI. **Diagnosis requires ≥10% clonal plasma cells or a plasmacytoma PLUS one CRAB or SLiM feature.**
6. **Investigations.** **Serum protein electrophoresis with immunofixation and serum free light chains** (essential — **light-chain-only and non-secretory myeloma have no serum M spike**); 24-hour urine for Bence-Jones

protein; complete blood count with film showing **rouleaux and a markedly raised ESR**; calcium, creatinine, albumin, LDH, β_2-microglobulin; **bone marrow aspirate and trephine with FISH cytogenetics**; and **whole-body low-dose CT, whole-body MRI or FDG PET-CT — the plain skeletal survey is obsolete because it misses lesions until 30–50% of bone is lost**.

7. **Staging and prognosis.** **Revised International Staging System (R-ISS)** combines **β_2-microglobulin and albumin (the original ISS) with LDH and high-risk cytogenetics [del(17p), t(4;14), t(14;16)]**: **Stage I** — β_2M <3.5 mg/L and albumin ≥3.5 g/dL with normal LDH and standard-risk cytogenetics; **Stage III** — β_2M ≥5.5 mg/L with high LDH or high-risk cytogenetics. The older **Durie-Salmon** system used tumour burden.
8. **Management of newly diagnosed myeloma.** **Transplant-eligible (usually under 70–75 and fit):** induction with a triplet or quadruplet — **bortezomib + lenalidomide + dexamethasone (VRd)**, increasingly with daratumumab — followed by **high-dose melphalan with autologous stem cell transplant** and **lenalidomide maintenance**. **Transplant-ineligible: daratumumab + lenalidomide + dexamethasone (D-Rd, the MAIA regimen)** or VRd-lite, continued until progression, with dose reduction for frailty. **Avoid melphalan before stem cell harvest** in potential transplant candidates.
9. **Supportive care (heavily examined).** **Bone** — zoledronic acid or pamidronate monthly for at least 2 years (denosumab if renal impairment; dental review first because of osteonecrosis of the jaw); radiotherapy for pain and impending fracture; vertebroplasty/kyphoplasty. **Renal** — aggressive hydration, avoid NSAIDs, contrast and nephrotoxins, treat hypercalcaemia, use bortezomib-based therapy (no dose reduction needed in renal failure). **Infection** — vaccination, prompt antibiotics, **aciclovir prophylaxis with bortezomib and daratumumab**, immunoglobulin replacement for recurrent infection. **Thrombosis** — aspirin or LMWH prophylaxis with immunomodulatory drugs. Plus analgesia, anaemia management and **early palliative input**.
10. **Relapsed disease and the geriatric approach.** Relapse is inevitable; options include **carfilzomib, pomalidomide, isatuximab, elotuzumab, selinexor, bendamustine** and re-treatment. In older patients, **use the IMWG frailty index or R-MCI (age, comorbidity, ADL/IADL) to classify fit, intermediate-fit and frail**, and **apply dose-adjusted regimens rather than withholding treatment** — under-treatment of the fit elderly is as harmful as over-treatment of the frail. Steroid dose reduction (dexamethasone 20 mg weekly instead of 40 mg) markedly reduces toxicity without losing efficacy.

Latest Updates (2023–2026) — 2 points

11. **CEPHEUS trial** (*Nature Medicine* 2025; FDA approval 2026) — in **transplant-ineligible or transplant-deferred newly diagnosed myeloma**, adding daratumumab to VRd (**D-VRd quadruplet**) nearly doubled sustained MRD negativity (**60.9% vs 39.5%**) and significantly improved progression-free survival, with benefit maintained irrespective of frailty status. Together with **PERSEUS** (*NEJM* 2024) in transplant-eligible patients and the **MAIA** benchmark of 7.5 years median overall survival with D-Rd, **a daratumumab-containing quadruplet or triplet is now the standard of care for older patients**.
12. **T-cell redirecting therapy in relapsed disease. BCMA CAR T-cells (ide-cel, cilta-cel)** and the **bispecific antibodies teclistamab, elranatamab (anti-BCMA) and talquetamab (anti-GPRC5D)** achieve response rates of 60–80% in triple-class-refractory myeloma. Bispecifics are **off-the-shelf and given subcutaneously, making them more practical for older patients** than CAR-T; the trade-offs are **cytokine release syndrome, ICANS and a high rate of serious infection requiring routine immunoglobulin replacement and antimicrobial prophylaxis**.

Current Practice Update

Textbook: Melphalan-prednisolone or a bortezomib-based triplet for transplant-ineligible myeloma; skeletal survey for bone disease; treat only when CRAB features appear.
Current recommendation: Daratumumab-based triplet or quadruplet (D-Rd / D-VRd) is standard for the transplant-ineligible; whole-body low-dose CT, MRI or PET-CT replaces skeletal survey; SLiM biomarkers permit treatment before organ damage.
Exam approach: Three separate marks are available here — state the quadruplet, say "skeletal survey is obsolete", and quote the SLiM criteria.

Cross-reference: The wider spectrum of plasma cell dyscrasias, including AL amyloidosis, Waldenström macroglobulinaemia, POEMS and MGRS, is covered in Question 32.

Question 34. Bone marrow transplantation in older patients 7 MARKS

Answer — Textbook Core (6 points)

1. **Types.** **Autologous** transplantation uses the patient's own stem cells after high-dose chemotherapy (myeloma,

relapsed lymphoma) — **no graft-versus-host disease and lower transplant-related mortality**. **Allogeneic** transplantation uses donor cells (AML, MDS, myelofibrosis) and provides a **graft-versus-tumour effect** at the cost of GVHD.

2. **Age is no longer a barrier.** Allogeneic transplantation is now routinely performed up to **70–75 years** using **reduced-intensity conditioning (RIC)** or non-myeloablative regimens (fludarabine-melphalan, fludarabine-busulfan), which rely on the immunological graft-versus-tumour effect rather than dose intensity.
3. **Selection is by physiological, not chronological, age.** The **HCT-CI (Hematopoietic Cell Transplantation Comorbidity Index)** together with performance status, **comprehensive geriatric assessment**, frailty, nutrition, cognition and caregiver support predicts non-relapse mortality far better than age alone.
4. **Indications in older adults.** **AML in first remission with intermediate or adverse risk**, MDS with excess blasts, **myelofibrosis with DIPSS intermediate-2 or high risk**, and **autologous transplantation for multiple myeloma** (feasible to around 75 years in fit patients).
5. **Early complications.** Mucositis, prolonged pancytopenia, **neutropenic sepsis**, invasive fungal infection, **CMV reactivation**, haemorrhagic cystitis, **sinusoidal obstruction syndrome (veno-occlusive disease)**, engraftment syndrome, and **acute GVHD affecting skin, gut and liver within 100 days**.
6. **Late complications.** **Chronic GVHD**, prolonged immunodeficiency with encapsulated organism and zoster infection (requiring **revaccination from 6–12 months plus PCP and antiviral prophylaxis**), secondary malignancy, endocrine failure, cataract, osteoporosis, and **cognitive and functional decline** — older survivors need structured survivorship care.

Latest Update (2023–2026) — 1 point

7. **BMT CTN 1703 trial** (*NEJM* 2023) — **post-transplant cyclophosphamide with tacrolimus and mycophenolate mofetil is now standard GVHD prophylaxis for reduced-intensity allogeneic transplantation**, significantly improving GVHD-free, relapse-free survival. It has also **widened donor availability to haploidentical and mismatched unrelated donors**, making transplantation accessible to substantially more older patients.

PART VI — ONCOLOGY

Question 35. Prostatic cancer 7 MARKS

Answer — Textbook Core (6 points)

1. **Epidemiology and risk factors.** The commonest non-cutaneous cancer in men; incidence rises steeply with age, with **median age at diagnosis around 66–70 years** and **latent histological cancer present in over 70% of men above 80 at autopsy**. Risk factors: **age, African ancestry, family history, BRCA2 and Lynch syndrome**, obesity and a high-fat diet. Over 95% are **adenocarcinomas arising in the peripheral zone**.
2. **Clinical features.** **Early disease is asymptomatic** and detected by PSA testing or a hard, nodular, irregular prostate with loss of the median sulcus on digital rectal examination. Later: lower urinary tract symptoms, haematuria, haematospermia, retention. **Advanced disease characteristically presents with osteoblastic (sclerotic) bone metastases causing back pain, pathological fracture or spinal cord compression**, and with anaemia, weight loss or renal failure from ureteric obstruction — **in older men, bone pain is often the first presentation**.
3. **Diagnosis.** **PSA** (raised also in BPH, prostatitis, catheterisation, retention and after DRE; use age-specific ranges, free:total ratio, PSA density and velocity to improve specificity). **Multiparametric MRI before biopsy (PI-RADS scoring)** is now standard, followed by **MRI-targeted transperineal or transrectal biopsy**. Histology is graded by the **Gleason score, reported as ISUP Grade Groups 1–5 (Gleason 6 = GG1 up to Gleason 9–10 = GG5)**. Staging by **TNM with bone scan and CT, or PSMA PET-CT**.
4. **Risk stratification.** **Low risk** — PSA <10, Gleason ≤6 (GG1), ≤T2a. **Intermediate risk** — PSA 10–20, Gleason 7 (GG2–3), T2b–c. **High risk** — PSA >20, Gleason 8–10 (GG4–5), ≥T3. This determines whether surveillance, radical treatment or systemic therapy is appropriate.
5. **Management of localised disease.** **Active surveillance** for low-risk disease (PSA, DRE, MRI and repeat biopsy) — the preferred option in most older men. **Watchful waiting with deferred palliative hormonal treatment** is appropriate when life expectancy is under 10 years. **Radical prostatectomy or radical radiotherapy (external beam or brachytherapy) with neoadjuvant/adjuvant androgen deprivation for high-risk disease** is offered when life expectancy exceeds 10 years. **Radical treatment is justified by life expectancy and health status, not by chronological age**; complications are incontinence and erectile dysfunction (surgery) and radiation proctitis and cystitis.
6. **Management of advanced disease.** **Androgen deprivation therapy** is the backbone — **LHRH agonists (goserelin, leuprolide, with anti-androgen cover for 2 weeks to prevent tumour flare)**, the LHRH antagonist degarelix, or bilateral orchidectomy. **Adverse effects of ADT are heavily examinable in geriatrics: hot flushes, loss of libido and erectile dysfunction, sarcopenia and fatigue, weight gain, osteoporosis and fracture (give calcium, vitamin D and a bisphosphonate or denosumab with DEXA monitoring), metabolic syndrome, diabetes, cardiovascular events, anaemia, gynaecomastia, and cognitive change.** For **metastatic hormone-sensitive disease, ADT is intensified with an androgen receptor pathway inhibitor (abiraterone, enzalutamide, apalutamide, darolutamide) ± docetaxel**. In **castration-resistant disease**: abiraterone, enzalutamide, docetaxel, cabazitaxel, **radium-223 for bone-predominant disease**, olaparib for BRCA mutations, and **Lu-177 PSMA radioligand therapy**. Palliative radiotherapy for bone pain and cord compression, plus a bone-protecting agent.

Latest Update (2023–2026) — 1 point

7. **PSMA PET-CT and geriatric assessment are now embedded in guidelines.** The **EAU 2025 and AUA/ASTRO 2026 guidelines** recommend **PSMA PET-CT for staging high-risk disease and for biochemical recurrence when PSA >0.2 ng/mL**, since the **proPSMA trial** showed sensitivity for nodal disease of 91% versus 59% for conventional imaging, and it frequently changes management. Equally important for the geriatric answer, the EAU gives a **strong recommendation to use the Geriatric 8 (G8), mini-Cog and Clinical Frailty Scale to assess health status**, and states explicitly that **life expectancy and comorbidity, not age, should drive treatment decisions** — with more than 10 years of life expectancy considered mandatory before any radical local treatment.

Cross-reference: Positron emission tomography, including PSMA PET, is covered in Question 41.

Question 36. Vertebral secondaries with unknown primary — evaluation and treatment

7 MARKS

Answer — Textbook Core (6 points)

1. **Clinical recognition.** Suspect metastatic disease in any older patient with **back pain that is progressive, unremitting, worse at night or at rest, associated with weight loss or a known cancer, or accompanied by neurological signs**. **Red flags: age >50 with new back pain, thoracic pain, band-like radiation, bowel or bladder dysfunction, saddle anaesthesia and progressive weakness — these indicate possible metastatic spinal cord compression, an oncological emergency.**
2. **The likely primaries.** The mnemonic for bone metastasis is **"BLT with a Kosher Pickle" — Breast, Lung, Thyroid, Kidney and Prostate**, which together account for about 80%. **Osteolytic** lesions come typically from lung, kidney, thyroid and myeloma; **osteoblastic (sclerotic)** from prostate and, in women, breast; **mixed** from breast and gastrointestinal tumours. Spread is largely via the **valveless Batson venous plexus**, which explains vertebral predilection.
3. **Essential differential diagnosis.** **Multiple myeloma** (must always be excluded — it is not a "metastasis" and requires different treatment), **osteoporotic vertebral fracture**, **tuberculous spondylitis (Pott's disease — extremely important in India, typically involving the disc space with a paravertebral abscess, whereas malignancy characteristically spares the disc)**, pyogenic osteomyelitis, Paget's disease and lymphoma.
4. **Evaluation — step 1 (confirm and localise).** **MRI of the whole spine is the investigation of choice** (detects cord compression, multiple levels and soft tissue extension; malignancy typically involves the vertebral body and pedicle while sparing the disc). Then **CT chest, abdomen and pelvis**. Baseline tests: complete blood count, ESR, calcium, ALP, renal and liver function, LDH, urinalysis. **Serum protein electrophoresis with free light chains and urine Bence-Jones protein in every case.**
5. **Evaluation — step 2 (find the primary).** Careful **examination of breasts, thyroid, testes, skin, lymph nodes and a digital rectal examination**. Targeted tumour markers — **PSA in men**, and CA-125, CA 19-9, CEA and AFP as guided (of limited specificity). **Mammography in women**; **FDG PET-CT** to identify an occult primary and assess disease extent. **Biopsy of the most accessible lesion is mandatory** — with **immunohistochemistry (CK7/CK20 pattern, TTF-1 for lung and thyroid, PSA/NKX3.1 for prostate, ER/PR/GATA3 for breast, thyroglobulin, PAX8 for renal and ovarian)**. Despite full workup, **the primary remains unidentified in 20–30%**.
6. **Treatment.** **If cord compression is present, this is an emergency: give dexamethasone 8–16 mg immediately, then urgent MRI and neurosurgical or radiotherapy referral within 24 hours** — surgical decompression with stabilisation followed by radiotherapy gives better ambulatory outcomes than radiotherapy alone in fit patients with a single level and a prognosis over 3 months. Otherwise: **palliative external beam radiotherapy (a single 8 Gy fraction is as effective as multi-fraction regimens for uncomplicated bone pain)**; **bone-modifying agents — zoledronic acid or denosumab** to reduce skeletal-related events; **analgesia by the WHO ladder**; treatment of hypercalcaemia; vertebroplasty or kyphoplasty for mechanical pain; and **treat according to the identified primary**. Where no primary is found, treat by **histological subtype and immunohistochemical/molecular profile**, with early involvement of palliative care and honest prognostic discussion (median survival in carcinoma of unknown primary with bone metastases is only a few months).

Latest Update (2023–2026) — 1 point

7. **Molecularly guided therapy for carcinoma of unknown primary — the CUPISCO trial** (*Lancet* 2024). In patients with unfavourable-risk CUP who did not progress after platinum induction chemotherapy, **comprehensive genomic profiling followed by molecularly guided targeted therapy or immunotherapy significantly improved progression-free survival compared with continuing standard chemotherapy**. This, together with the **ESMO 2023 CUP guideline**, has changed practice: **next-generation sequencing and PD-L1/MSI testing should now be performed in every patient with unknown primary who is fit for treatment**, and **site-of-origin gene expression profiling** can further direct therapy.

Question 37. Paraneoplastic syndromes 7 MARKS

Answer — Textbook Core (6 points)

1. **Definition.** Remote, **non-metastatic** effects of malignancy mediated by tumour-secreted hormones, cytokines or **cross-reacting onconeural antibodies** — not by tumour mass, local invasion or metastasis. They are important because they are frequently the **first presentation of an occult cancer in an older adult**.
2. **Endocrine syndromes.** **SIADH with hyponatraemia (small cell lung cancer)**; **ectopic ACTH** (SCLC, carcinoid)

causing hypokalaemic alkalosis, hyperglycaemia and proximal myopathy rather than classic Cushingoid habitus; **humoral hypercalcaemia of malignancy from PTHrP** (squamous cell lung, renal and breast cancer); and hypoglycaemia from IGF-2 (hepatoma, sarcoma).

3. **Neurological syndromes.** **Lambert-Eaton myasthenic syndrome** (SCLC) — proximal weakness that **improves with exercise**, autonomic features and absent reflexes, with **anti-VGCC antibodies**; **paraneoplastic cerebellar degeneration** (anti-Yo in ovarian and breast cancer, anti-Hu in SCLC); **limbic encephalitis** (anti-Hu, anti-Ma2); sensory neuronopathy; and opsoclonus-myoclonus.
4. **Haematological and vascular syndromes.** Anaemia of chronic disease, **erythrocytosis from ectopic erythropoietin** (renal cell carcinoma, hepatoma, cerebellar haemangioblastoma), thrombocytosis, leukaemoid reaction, eosinophilia, **migratory thrombophlebitis — Trousseau's sign, classically pancreatic carcinoma**, non-bacterial thrombotic endocarditis and DIC.
5. **Dermatological and rheumatological syndromes.** **Acanthosis nigricans (gastric adenocarcinoma)**, **dermatomyositis** with heliotrope rash and Gottron's papules, **Leser-Trélat sign** (sudden eruptive seborrhoeic keratoses), acquired ichthyosis, **hypertrophic pulmonary osteoarthropathy with clubbing** (lung cancer), paraneoplastic pemphigus and palmar fasciitis.
6. **Approach and management.** Recognise the syndrome, then **search for the underlying tumour** (CT chest-abdomen-pelvis, mammography, PET-CT, antibody-directed screening). **Treating the tumour is the definitive treatment.** Symptomatic measures: fluid restriction or tolvaptan for SIADH; **hydration plus zoledronate for hypercalcaemia**; and immunotherapy (corticosteroids, IVIG, plasma exchange, rituximab) for antibody-mediated neurological syndromes — though response is often poor once neuronal loss has occurred, so **early recognition is critical**.

Latest Update (2023–2026) — 1 point

7. **PNS-Care diagnostic framework** (Graus et al., updated criteria now in general use) classifies paraneoplastic neurological syndromes as **definite, probable or possible** using the clinical phenotype, the antibody type (**high-risk antibodies such as anti-Hu, anti-Yo and anti-Ma2 versus intermediate-risk antibodies**) and the presence of tumour. A parallel development is the growing recognition of **immune checkpoint inhibitor-triggered paraneoplastic neurological syndromes**, increasingly seen as immunotherapy use expands in older patients with cancer.

Question 38. Tumour lysis syndrome 7 MARKS

Answer — Textbook Core (6 points)

1. **Definition and mechanism.** An **oncological emergency caused by massive lysis of tumour cells, spontaneously or after cytotoxic therapy**, releasing intracellular contents into the circulation faster than the kidney can clear them. This produces **hyperuricaemia, hyperkalaemia, hyperphosphataemia and secondary hypocalcaemia**, with **acute kidney injury** from uric acid and calcium phosphate crystal deposition.
2. **Risk factors.** **High tumour burden and rapid proliferation** — Burkitt lymphoma, acute lymphoblastic leukaemia, high-grade non-Hodgkin lymphoma, AML with high white cell count, and CLL treated with venetoclax; **high sensitivity to therapy**; bulky disease with raised LDH; and **patient factors — pre-existing chronic kidney disease, dehydration, oliguria, hyperuricaemia and nephrotoxic drugs**. **Older patients are at higher risk because of reduced GFR and lower physiological reserve.** Solid tumours rarely cause it.
3. **Cairo-Bishop definitions.** **Laboratory TLS** — two or more of the following within 3 days before to 7 days after chemotherapy: **uric acid ≥8 mg/dL, potassium ≥6 mmol/L, phosphate ≥4.5 mg/dL, or calcium ≤7 mg/dL** (or a 25% change from baseline). **Clinical TLS** — laboratory TLS plus **acute kidney injury (creatinine ≥1.5× upper limit of normal), cardiac arrhythmia or sudden death, or seizure**.
4. **Clinical features.** Nausea, vomiting, lethargy, oliguria and fluid overload; **hyperkalaemia causing muscle weakness, paraesthesiae, peaked T waves, widened QRS and cardiac arrest**; **hypocalcaemia causing tetany, carpopedal spasm, Chvostek and Trousseau signs, prolonged QT and seizures**; and acute kidney injury. Symptoms typically appear **12–72 hours after starting treatment**.
5. **Prevention (the most important element).** **Risk-stratify every patient before chemotherapy.** Give **aggressive intravenous hydration with 2–3 litres/m²/day of normal saline to maintain a urine output of 80–100 mL/m²/hour** — started 24–48 hours before treatment and continued for 48–72 hours afterwards, **with careful monitoring for fluid overload in older patients with heart failure**. **Allopurinol 300 mg daily (a xanthine oxidase inhibitor, renally dose-adjusted) for low and intermediate risk**; **rasburicase (recombinant urate oxidase, which converts uric acid to allantoin) for high risk or established TLS** — it acts within hours, unlike

allopurinol which only prevents new urate formation. **Rasburicase is contraindicated in G6PD deficiency (haemolysis and methaemoglobinaemia) — screen first, which is particularly relevant in India. Urinary alkalinisation is no longer recommended** because it promotes calcium phosphate precipitation. Stop nephrotoxins and monitor biochemistry 6–12 hourly.

6. **Treatment of established TLS.** Continue vigorous hydration and rasburicase; treat **hyperkalaemia** with calcium gluconate for cardioprotection, insulin-dextrose, salbutamol nebulisation and potassium binders; treat **hyperphosphataemia** with oral phosphate binders; and **treat hypocalcaemia only if symptomatic**, because giving calcium in the presence of a high phosphate promotes further calcium phosphate deposition. **Refer early for renal replacement therapy** — indications are refractory hyperkalaemia, severe hyperphosphataemia, volume overload, uraemia or persistent oliguria; the threshold for dialysis should be lower in the elderly. Manage jointly with nephrology and critical care.

Latest Update (2023–2026) — 1 point

7. **TLS in the era of targeted therapy and evidence for lower rasburicase dosing.** TLS is now an important toxicity of **venetoclax in CLL and AML**, which is why venetoclax must be given by a **graded weekly dose ramp-up (20 → 50 → 100 → 200 → 400 mg) with risk-adapted hydration, uric acid-lowering therapy and inpatient monitoring for high tumour burden**; it also occurs with **CAR T-cell therapy and bispecific antibodies**. In parallel, multiple studies support **a single fixed low dose of rasburicase (3–6 mg) rather than daily weight-based dosing for 5 days**, achieving equivalent urate control at a fraction of the cost — an important consideration in Indian practice.

PART VII — CANCER SCREENING AND IMAGING

Question 39. Indications and rationale for cancer screening in the elderly 7 MARKS

Answer — Textbook Core (6 points)

1. **Principles of screening (Wilson and Jungner).** Screening is justified when the **condition is an important health problem with a recognisable latent or early symptomatic stage**, the **natural history is understood**, there is an **acceptable, safe, sensitive and specific test**, an **accepted and effective treatment exists**, facilities for diagnosis and treatment are available, and the process is **continuous and cost-effective**.
2. **The rationale for screening older adults.** **Cancer is fundamentally a disease of ageing** — incidence and mortality both peak in old age, and over 60% of cancers occur in those above 65 — so the **absolute benefit of detecting a screen-detectable cancer is greatest in this group**. Older people have historically been **under-screened and under-treated**, presenting at later stage with poorer outcomes.
3. **The counter-argument — why screening can harm the old.** There is a **lag time to benefit of approximately 10 years for mammography and colonoscopy and about 5 years for FIT**: harms (bleeding, perforation, anxiety, false positives) are immediate while benefits are delayed. Additional harms are **overdiagnosis of indolent tumours that would never have caused symptoms** (especially prostate and breast), **lead-time and length-time bias** creating an illusion of benefit, and the burden of investigating false positives in frail patients.
4. **The decisive rule — life expectancy, not chronological age.** **Screening should be offered when estimated life expectancy exceeds the lag time to benefit — in practice, more than 10 years.** Estimate life expectancy from comorbidity, function and validated tools (**ePrognosis, Lee index, Charlson comorbidity index**), not from the birth date. A fit 80-year-old woman may have 10 years of life expectancy and benefit; a frail 68-year-old with heart failure and dementia will not.
5. **Practical age limits in current guidance.** **Breast — biennial mammography to age 74** (individualised beyond). **Colorectal — screen 45–75; individualise 76–85 based on health and prior screening; do not screen after 85. Cervical — stop at 65 if there has been adequate prior negative screening** (but continue if never screened, which is common among older Indian women). **Prostate — shared decision-making between 55 and 69; do not screen routinely above 70. Lung — annual low-dose CT for ages 50–80 with a ≥20 pack-year history who currently smoke or quit within 15 years.**
6. **How to decide and communicate.** Use **shared decision-making** — present absolute benefit and harm in natural frequencies, elicit the patient's values and goals of care, and consider **whether the patient would accept the investigation and treatment that a positive result would demand**. If they would decline colonoscopy or surgery, the screening test should not be done. **Deprescribing screening ("stopping screening") should be framed positively as a shift towards care that helps now**, rather than as withdrawal of care, and documented. In the Indian context, screening should also be balanced against limited resources and the very low baseline coverage — opportunistic screening at the point of care is often more realistic than organised programmes.

> **Latest Update (2023–2026) — 1 point**
>
> 7. **USPSTF 2024 breast cancer screening statement** (*JAMA* 2024;331:1918) — now recommends **biennial mammography for all women aged 40 to 74 (Grade B)**, lowering the starting age from 50, while **concluding that evidence remains insufficient for screening women aged 75 and older (Grade I)**. This crystallises the geriatric teaching point: the upper limit of screening is not defined by a trial but by **individual life expectancy and shared decision-making**, which is why the answer must always pivot to functional status rather than age.

Cross-reference: The individual screening programmes, tests and their sensitivity and specificity are tabulated in Question 40.

Question 40. Screening for breast, colorectal and cervical cancer — population, tests, performance and guidelines 7 MARKS

Answer — Textbook Core (6 points)

1. **Breast cancer — target population and guidelines.** **Women aged 40–74 (USPSTF 2024), biennial mammography**; the American Cancer Society advises annual screening from 45 and biennial from 55, continuing while life expectancy exceeds 10 years. High-risk women (BRCA carriers, chest irradiation) start earlier with **annual MRI plus mammography**. In India, the **ICMR and national programme (NPCDCS) recommend clinical breast examination every 3 years from age 30** because population mammography is not feasible — a crucial contextual

point.

2. **Breast cancer — tests and performance.** **Mammography: sensitivity approximately 75–85% (falling to 60–70% in dense breasts, but rising with age as breasts become fattier), specificity 90–95%. Digital breast tomosynthesis** improves cancer detection and reduces recall. **Clinical breast examination: sensitivity about 50–60%. Ultrasound** is adjunctive for dense breasts; **MRI has the highest sensitivity (over 90%) but lower specificity** and is reserved for high risk.
3. **Colorectal cancer — target population and guidelines.** **Average-risk adults aged 45–75 (USPSTF 2021)**; **individualise between 76 and 85 years** according to health status and prior screening, and **do not screen beyond 85**. Earlier and more intensive surveillance for a family history, Lynch syndrome, FAP or inflammatory bowel disease.
4. **Colorectal cancer — tests and performance.** **FIT (faecal immunochemical test) annually — sensitivity about 75–80% for cancer but low (about 25%) for advanced adenoma, specificity about 95%**; it is the most practical test for population screening in India. **Multitarget stool DNA-FIT every 3 years — sensitivity about 92% for cancer, specificity about 87%. Colonoscopy every 10 years — the gold standard, sensitivity over 95%, but carries risks of perforation and bleeding that rise with age and sedation risk. CT colonography every 5 years**; flexible sigmoidoscopy every 5 years. **Any positive non-colonoscopic test must be followed by colonoscopy.**
5. **Cervical cancer — target population and guidelines.** **Women aged 21–65.** Cytology alone every 3 years from 21–29; from 30–65, **primary HPV testing every 5 years (preferred), co-testing every 5 years, or cytology every 3 years. Stop at 65 if there has been adequate prior negative screening** (three consecutive negative cytology or two negative HPV tests in the preceding 10 years). **Continue screening in older women who have never been screened** — a large group in India, where cervical cancer remains a leading cause of cancer death. WHO recommends **HPV DNA testing as the primary screening method from age 30, twice in a lifetime, in low-resource settings**, alongside HPV vaccination.
6. **Cervical cancer — tests and performance.** **Conventional Pap smear: sensitivity only about 50–60%, specificity over 95%** — its programme effectiveness came from repetition, not single-test accuracy. **Liquid-based cytology has similar sensitivity with fewer unsatisfactory samples. HPV DNA testing: sensitivity about 90–95% with lower specificity (about 85–90%)** — hence a longer screening interval and the need for triage of positives by cytology or genotyping. **Visual inspection with acetic acid (VIA): sensitivity about 60–70%, specificity about 85%** — cheap, single-visit, and the basis of India's national "see-and-treat" programme.

Latest Update (2023–2026) — 1 point

7. **Self-collection for HPV testing and blood-based colorectal screening.** The FDA has approved **self-collected vaginal samples for primary HPV screening (2024)**, which markedly improves uptake among women who avoid pelvic examination — directly relevant to older and rural Indian women and now endorsed by WHO. For colorectal cancer, **blood-based cell-free DNA testing (Shield, approved 2024) detects cancer with about 83% sensitivity but only about 13% sensitivity for advanced adenoma**, so it is positioned as an option only for those who decline all other screening, not as an equivalent alternative to FIT or colonoscopy.

Cross-reference: The principles governing whether an older person should be screened at all are given in Question 39.

Question 41. Positron emission tomography 6 MARKS

Answer — Textbook Core (5 points)

1. **Principle.** PET is a **functional (metabolic) imaging technique** using a positron-emitting radionuclide attached to a biologically active molecule. The emitted positron annihilates with an electron to release **two 511 keV gamma photons at 180° to each other**, which are detected in coincidence and reconstructed into a three-dimensional map of tracer uptake. It is almost always performed as **PET-CT (or PET-MRI), which fuses metabolic information with anatomical localisation**.
2. **The commonest tracer.** **^{18}F-fluorodeoxyglucose (FDG)**, a glucose analogue taken up by GLUT transporters and phosphorylated by hexokinase, after which it becomes **metabolically trapped** in the cell. Uptake therefore reflects glucose utilisation, which is high in malignant tissue (the Warburg effect), in inflammation and infection, and in brain and myocardium. Uptake is quantified by the **standardised uptake value (SUV)**. Patients must **fast for 4–6 hours with blood glucose controlled below about 150–200 mg/dL**, since hyperglycaemia and recent insulin markedly reduce tumour uptake.
3. **Oncological indications (the main use).** **Staging and restaging** of lung cancer, lymphoma, oesophageal, head and neck, colorectal, melanoma and cervical cancer; **characterising a solitary pulmonary nodule**; **assessing**

treatment response, where PET is far superior to CT because a residual mass may be fibrotic rather than viable (the Deauville 5-point score in lymphoma); **detecting recurrence and identifying the primary in carcinoma of unknown origin**; and **radiotherapy planning**.

4. **Non-oncological indications relevant to geriatrics.** **Dementia** — FDG-PET shows characteristic hypometabolism patterns (**temporoparietal and posterior cingulate in Alzheimer's disease; frontal and anterior temporal in frontotemporal dementia; occipital hypometabolism with the "cingulate island sign" in dementia with Lewy bodies**), and **amyloid PET (florbetapir, flutemetamol) and tau PET (flortaucipir)** demonstrate the underlying pathology. **Cardiology** — assessment of **myocardial viability (hibernating myocardium: perfusion-metabolism mismatch)** and of cardiac sarcoidosis. **Infection and inflammation** — **pyrexia of unknown origin, large vessel vasculitis and giant cell arteritis, prosthetic joint and vascular graft infection**. **Epilepsy** — localisation of an interictal hypometabolic focus.
5. **Limitations and pitfalls.** **False positives** from infection, granulomatous disease (**tuberculosis and sarcoidosis — a major limitation in India**), post-operative and post-radiotherapy inflammation, recent chemotherapy, and physiological uptake in brown fat, muscle and bowel. **False negatives** in small lesions (below about 8–10 mm, from partial volume effect), in **indolent tumours with low metabolic activity such as prostate cancer, renal cell carcinoma, hepatocellular carcinoma, mucinous adenocarcinoma, bronchoalveolar carcinoma and low-grade lymphoma**, and in the brain because of high background cortical uptake. Other drawbacks: **high cost and limited availability, radiation exposure of about 7–10 mSv, the need for a nearby cyclotron because of the short 110-minute half-life of ^{18}F, and difficulty with prolonged lying still in frail or demented patients**.

Latest Update (2023–2026) — 1 point

6. **Disease-specific tracers have moved PET from staging tool to treatment selector. PSMA PET-CT** is now recommended in the EAU 2025 and AUA 2026 guidelines for staging high-risk prostate cancer and for biochemical recurrence, and the same ligand labelled with lutetium-177 delivers **Lu-177 PSMA radioligand therapy** — a true "theranostic" pairing, mirrored by **Ga-68 DOTATATE PET with Lu-177 DOTATATE for neuroendocrine tumours**. In dementia, **amyloid PET has become a prerequisite for prescribing anti-amyloid monoclonal antibodies (lecanemab, donanemab) and for monitoring amyloid-related imaging abnormalities (ARIA)**, shifting it from a research tool to a treatment-gating investigation.

APPENDIX — RAPID REVISION GRID

Every examinable number, named trial and guideline from all 41 answers, in one place. This is the page to read on the night before the examination.

Hepatology, upper GI disease and clinical nutrition (Questions 1, 4–6, 9–20)

Topic	Numbers, criteria and cut-offs	Trial / guideline to quote
Jaundice	Clinically visible at bilirubin >2.5–3 mg/dL; R value ≥5 hepatocellular, ≤2 cholestatic; Courvoisier's law; PT/INR = best severity marker	MASLD/MetALD nomenclature (Hepatology 2023)
Drug-induced liver injury	**Hy's Law** — ALT >3×ULN + bilirubin >2×ULN → ~10% mortality; commonest agent co-amoxiclav; RUCAM → RECAM	ACG DILI guideline; LiverTox; ICI hepatitis
Autoimmune hepatitis	Interface hepatitis, rosettes, emperipolesis; ANA/ASMA + raised IgG; prednisolone 0.5–1 mg/kg + azathioprine 1–2 mg/kg; no budesonide in cirrhosis	IAIHG simplified criteria; **CAMARO** (J Hepatol 2024;80:576)
Alcoholic hepatitis	**DF ≥32** or **MELD >20** = severe; AST:ALT >1.5, both <400; prednisolone 40 mg × 28 days; **Lille >0.45** at day 7 = stop steroids	ACG ALD 2024 (AJG 119:30); NIAAA criteria
Hepatitis B	Treat if cirrhosis + any DNA; or DNA >2000 IU/mL + raised ALT; or ≥F2/APRI >0.5; TAF or entecavir preferred in elderly; 6-monthly USG for HCC	**WHO 2024** (March 2024) expanded criteria
Decompensated cirrhosis	HVPG >10 = CSPH; CTP A 5–6, B 7–9, C 10–15; MELD >15 → transplant referral; LSM >15 kPa + platelets <150 = CSPH; SBP albumin 1.5 g/kg d1 + 1 g/kg d3	**Baveno VII** (J Hepatol 2022;76:959); PREDESCI; resmetirom 2024
Hepatic encephalopathy	West Haven I–IV; lactulose to 2–3 soft stools/day; rifaximin 550 mg BD; protein 1.2–1.5 g/kg (**no restriction**); ammonia not for monitoring	AASLD/EASL Practice Guidance 2022
Hepatorenal syndrome	HRS-AKI: creatinine ↑≥0.3 mg/dL in 48 h or ≥50% in 7 days; urine Na <10 mmol/L, FENa <0.2%, proteinuria <500 mg/day; terlipressin + albumin 20–40 g/day	ICA criteria; **ADQI 29/ICA 2024** (J Hepatol 81:163); CONFIRM
Ascites	**SAAG ≥1.1** = portal hypertension; SBP if neutrophils ≥250/mm³; spironolactone:furosemide **100:40** up to 400:160; albumin **8 g per litre** tapped; TIPS if ≥3 LVP/year	Baveno VII; ANSWER (Lancet 2018)
GORD	LA grades A–D; PPI 30–60 min before breakfast × 8 weeks; pH study **off** PPI = gold standard; endoscope all new dyspepsia >60 y	AGA P-CAB Update 2024 (Gastro 167:1228); **ASGE GERD 2025**
Malabsorption	Faecal fat >7 g/day; faecal elastase-1 <200 µg/g = PEI; lipase 40,000–50,000 U per meal; tTG-IgA + total IgA; Marsh grading	ACG Coeliac 2023 (AJG 118:59); AGA PEI Update 2023
Bristol chart / laxatives	1–2 constipation, 3–4 normal (4 ideal), 5 low fibre, 6–7 diarrhoea; PEG first-line; docusate ineffective; avoid liquid paraffin and bulking agents in the frail	AGA/ACG 2023; WGO 2025
Parenteral nutrition	20–30 kcal/kg, protein 1.0–1.5 g/kg, glucose ≤4–5 mg/kg/min; start if gut unusable >7 days; CRBSI = commonest serious complication	ESPEN geriatric practical guideline
Refeeding syndrome	BMI <16, weight loss >15%, no intake >10 days; start **10 kcal/kg/day** (5 if extreme); thiamine 200–300 mg before feeding; phosphate is the hallmark	NICE CG32; **ASPEN consensus 2020**
Inanition	MNA-SF screening; GLIM = 1 phenotypic + 1 aetiologic criterion; protein 1.2–1.5 g/kg/day; "9 Ds"; supplements **between** meals	GLIM; ESPEN geriatrics; **AGS Beers 2023** (avoid megestrol)

Gastroenterology, haematology and oncology (Questions 2–3, 7–8, 22–25, 30–31, 34, 37)

Topic	Numbers, criteria and cut-offs	Trial / guideline to quote
H. pylori	Stop PPI 2 weeks, antibiotics 4 weeks before UBT; confirm cure at 4 weeks; BQT 14 days; clarithromycin resistance 22–32%	ACG 2024 (AJG 119:1730); Maastricht VI
UGI bleeding	GBS 0–1 = outpatient; endoscopy <24 h; PPI 80 mg then 8 mg/h × 72 h; erythromycin 250 mg; Forrest Ia/Ib/IIa treat (rebleed 55/43/22%)	ACG 2021; ESGE 2021; CAG 2025
Transfusion	Hb <7 restrictive; <8 cardiac/orthopaedic surgery; **<10 in acute MI**; TACO = commonest transfusion death	AABB 2023 (JAMA 330:1892); AABB 2025 (Ann Intern Med 178:1469); MINT (NEJM 2023)
Colorectal cancer	≥12 nodes for staging; N1 = 1–3, N2 ≥4; 5-y survival I 90%, II 70–80%, III 50–65%, IV 15%; dMMR 15%	AJCC 8th; DYNAMIC; Cercek NEJM 2025; ATOMIC (ASCO 2025)
Constipation	Rome IV: 2 of 6, <3 BM/week; PEG first-line; docusate ineffective; PAMORAs for OIC; avoid bulking agents in OIC	AGA/ACG 2023; WGO 2025; ASCRS 2024
CLL	≥5 × 10^9/L B cells × 3 months; CD5/CD19/CD23/CD200+; Rai III Hb <11, IV plt <100; doubling time <6 months; Richter's 5%	iwCLL 2018; CLL14; AMPLIFY (NEJM 2025;392:748)
Myelofibrosis / MPN	JAK2 V617F 95% PV; Hct <45% target; pacritinib if platelets <50; DIPSS-Plus; AML transformation 10–20% at 10 y	COMFORT-I/II; MOMENTUM (Lancet 2023); NCCN MPN 2026
Iron deficiency	Ferritin <30 ng/mL (<45–100 if inflamed); TSAT <16%; RDW >15%; Mentzer >13; alternate-day oral iron	BSG IDA guideline; hepcidin dosing studies
B_{12} deficiency	B_{12} <200 pg/mL; indeterminate 200–350 → MMA; MCV often >110; hydroxocobalamin 1000 µg alternate days then 2–3 monthly; stores last 3–5 y	NICE NG239 (2024); BSH cobalamin guideline
Transplantation	RIC feasible to 70–75 y; HCT-CI predicts NRM; acute GVHD <100 days	BMT CTN 1703 (NEJM 2023)
Paraneoplastic	SIADH & ectopic ACTH → SCLC; PTHrP → squamous; anti-Yo → ovary; anti-VGCC → LEMS; acanthosis nigricans → gastric	PNS-Care criteria (Graus)
Malignant bowel obstruction	Octreotide 300–600 µg/24 h; dexamethasone 6–16 mg/24 h, stop at 5 days; hyoscine butylbromide 60–120 mg/24 h; metoclopramide contraindicated in complete obstruction	ASCO Palliative Care Update 2024 (JCO 42:2336); S1316

Anaemia, haematological malignancy, oncology and screening (Questions 21, 24, 26–29, 32–33, 35–36, 38–41)

Topic	Numbers, criteria and cut-offs	Trial / guideline to quote
Anaemia in the elderly	WHO Hb <13 (M), <12 (F); prevalence 10% >65 y, >20% >85 y; **rule of thirds**; reticulocyte count = key branch point	NHANES; WHO Hb thresholds 2024; CHIP
Anaemia of chronic disease	Ferritin normal/high, TIBC **low**, sTfR normal, hepcidin high; use ferritin <100 in inflammation; ESA target Hb 10–11.5	Hepcidin–ferroportin axis; HIF-PHIs (roxadustat, daprodustat)
Thrombocytopenia	<150; bleeding <30; serious risk <10–20; ITP treat if <20–30 or bleeding; **4Ts score** for HIT; marrow exam in most >60 y	ASH ITP guideline; ADVANCE (efgartigimod, Lancet 2023)
DIC	**ISTH score ≥5** = overt DIC; platelets <50 + bleeding → transfuse; FFP 15 mL/kg; cryoprecipitate if fibrinogen <1.5 g/L; heparin only if thrombotic phenotype	ISTH overt DIC score; SIC score
Plasma cell dyscrasias	MGUS: M <3 g/dL, PC <10%, 1%/year progression; smouldering: M ≥3 g/dL or PC 10–60%, 10%/year; "20-2-20" high risk; plasma cell leukaemia ≥5% circulating	IMWG criteria; R2-ISS; FDA MRD endpoint 2024
Multiple myeloma	**CRAB** + **SLiM** (PC ≥60%, FLC ratio ≥100, >1 focal MRI lesion ≥5 mm); R-ISS uses β2M, albumin, LDH, cytogenetics; skeletal survey obsolete	IMWG 2014; **CEPHEUS** (Nat Med 2025); PERSEUS (NEJM 2024); MAIA
Myelodysplasia	Blasts <20% (≥20% = AML); Pelger-Huët, ring sideroblasts; IPSS-R uses cytogenetics, blasts, Hb, platelets, neutrophils; EPO <500 predicts ESA response	WHO 2022; **IPSS-M**; COMMANDS 2024; IMerge/imetelstat 2024
CML	t(9;22) BCR::ABL1; **low LAP score**; blast crisis ≥20% blasts; milestones ≤10% at 3 mo, ≤1% at 6 mo, **≤0.1% (MMR) at 12 mo**; TFR after MR4.5 × 2 y	**ASC4FIRST** (NEJM 2024;391:885); ELN recommendations
Prostate cancer	Gleason → ISUP Grade Group 1–5; low risk PSA <10/GG1/≤T2a; high risk PSA >20/GG4-5/≥T3; radical treatment needs >10 y life expectancy; osteoblastic metastases	EAU 2025 (G8, mini-Cog, CFS); proPSMA; ARASENS/PEACE-1
Vertebral secondaries / CUP	"BLT with a Kosher Pickle"; malignancy spares the disc, TB does not; dexamethasone 8–16 mg + MRI within 24 h; **single 8 Gy** for uncomplicated bone pain; primary unfound in 20–30%	ESMO CUP 2023; **CUPISCO** (Lancet 2024)
Tumour lysis syndrome	Cairo-Bishop: urate ≥8, K ≥6, PO_4 ≥4.5, Ca ≤7 mg/dL; hydration 2–3 L/m²/day; rasburicase contraindicated in **G6PD deficiency**; no alkalinisation	Cairo-Bishop; venetoclax ramp-up; single-dose rasburicase
Cancer screening (general)	Lag time to benefit ~10 y (mammography, colonoscopy), ~5 y (FIT); screen if life expectancy >10 y; CRC 45–75, individualise 76–85, stop >85; cervical stop at 65	**USPSTF 2024 breast 40–74 (Grade B)**; ePrognosis; Lee index
Screening test performance	Mammography Sn 75–85%/Sp 90–95%; FIT Sn ~75–80% for cancer; colonoscopy Sn >95%; Pap Sn 50–60%/Sp >95%; HPV DNA Sn 90–95%; VIA Sn 60–70%	USPSTF; WHO HPV strategy; ICMR/NPCDCS (CBE every 3 y from 30)
PET	Two 511 keV photons at 180°; fast 4–6 h, glucose <150–200 mg/dL; ^{18}F half-life 110 min; ~7–10 mSv; false positive in TB and sarcoid; false negative <8–10 mm and in prostate, RCC, HCC, mucinous tumours	Deauville score; PSMA/DOTATATE theranostics; amyloid PET for lecanemab

Curriculum domains still outstanding across the answer bank: rheumatology; endocrinology and thyroid disease; dermatology; urology and incontinence; falls and osteoporosis; ophthalmology; non-pharmacological management of dementia; rehabilitation; comprehensive geriatric assessment as a standalone topic; and disorders of potassium, calcium and magnesium.

DELIRIUM & ACUTE CNS INFECTION IN THE ELDERLY

MD Geriatric Medicine — Mark-wise Exam Notes (Q29–Q33) | Primary source: Pathy's Principles and Practice of Geriatric Medicine, 6th ed. (Ch. 61, Ch. 102) with Kane's Essentials of Clinical Geriatrics (Ch. 6) | Updates to August 2026

RECURRING TOPIC. Q29, Q30 (asked twice), Q31 and Q32 are all the same subject — **delirium** — asked at different mark weights and from different angles. Each answer below is deliberately given a **different pivot** so nothing is repeated. Q33 is the same syndrome presenting as an emergency with a specific treatable cause. Extra recall material is at the end.

Format used: 6-mark note = **5 textbook points + 1 latest update** • 7-mark note = **6 textbook points + 1 latest update** • 15-mark essay = **10 textbook points + 2 latest updates**.

Q29. Acute confusional state — 6-MARK NOTE — 5 + 1

*Pivot for this question: **what the term means and how to sort out its cause**.*

Textbook Core — 5 Points

1. **Definition.** Acute confusional state is the older clinical name for **delirium** (also called **acute brain failure** or altered mental status). It is an **acutely developing, fluctuating disturbance of attention, awareness and cognition** caused by an underlying medical illness, drug or withdrawal state. It is a **syndrome, not a diagnosis** — the cause must always be sought.
2. **Clinical picture.** There is **impaired consciousness with global cognitive impairment of acute onset**, disorientation, and a disturbed **sleep–wake cycle**. Psychomotor activity may be increased (**hyperactive**) or reduced (**hypoactive**) or both (**mixed**). **Illusions** (misinterpreting bedsheet patterns as insects), **visual hallucinations** and paranoid delusions occur. Symptoms are **worse at night** with lucid intervals by day.
3. **Causes.** Almost always **multifactorial** — a vulnerable brain plus a precipitant. Use the Pathy mnemonic **"DELIRIUM IS VERY PAINFUL"**: **D**rugs and withdrawal, **E**yes/Ears, **L**ow oxygen or perfusion, **I**nfection, **R**etention of urine (cystocerebral syndrome), **I**ndwelling catheter, **U**nderhydration (BUN:creatinine > 17:1), **M**etabolic, **I**mmobility, **S**leep problems, **V**ascular change on imaging, **E**ndocrinopathy, **R**estraints, ps**Y**chiatric disorder, **P**ain, **A**ttachments, **I**mpaction, **N**eurological injury, **F**railty, **U**ndernutrition, **L**ights and loud noise. In a highly predisposed patient a **single dose of diphenhydramine** may be enough to precipitate it.
4. **Differential diagnosis. Dementia** (gradual, no clouding of consciousness, attention preserved), **delirium superimposed on dementia**, **depression** (pseudodementia), late-onset psychosis or mania, **non-convulsive status epilepticus** and Wernicke's encephalopathy. The most reliable discriminator is to focus on the **core features — attention, awareness and level of consciousness — not on behaviour**. Kane's three questions: was the onset **acute**? are there **physical** factors? are there **psychological** factors?
5. **Approach.** Screen with **CAM or 4AT**, confirm against **DSM-5/ICD-10**, then perform a targeted search for every reversible contributor — drug chart, infection screen, glucose, electrolytes, hydration, bladder and bowel, oxygenation, pain. **Neuroimaging, lumbar puncture and EEG are not routine**; they are ordered only for focal signs, head injury, unexplained coma, suspected encephalitis or suspected seizures. Treatment is to correct the cause plus **multicomponent non-pharmacologic care**; drugs are reserved for danger to self or others.

Latest Update — 1 Point

6. The **APA Practice Guideline for the Prevention and Treatment of Delirium (September 2025)** — the first full revision since 1999 — shifts the emphasis of the whole field from treatment to **prevention and early identification**. It requires a thorough medication, predisposing- and precipitating-factor history in every patient, restricts physical restraints to circumstances of **"imminent harm"**, and adds a structured **transitions-of-care** requirement covering medication reconciliation, delirium follow-up and caregiver education. **NICE CG103 (updated January 2023)** complements this by recommending the **4AT** as the routine bedside assessment tool.

Q30 (a). Delirium 7-MARK NOTE — 6 + 1

*Pivot for this version: **the complete syndrome — mechanism, diagnosis, management, prognosis**.*

Textbook Core — 6 Points

1. **Definition and DSM-5 criteria.** Delirium is an acute disorder of attention and cognition. **DSM-5** requires: (i) disturbance of **attention and awareness**; (ii) **acute onset** with **fluctuation** over the day, a change from baseline; (iii) an additional **cognitive** disturbance; (iv) not better explained by a neurocognitive disorder and not occurring in **coma**; (v) evidence of a **medical cause, substance, withdrawal or multiple aetiologies**. DSM-5 and ICD-10 remain the **gold standard**.
2. **Epidemiology and subtypes.** Rates: **ICU 30–80%**, hospice 26–44%, **general medical and oncology wards 18–35%**, nursing home/post-acute care 16%; cardiac and vascular surgery 29–50%; hip fracture repair 12–48%. Subtypes are **hyperactive, hypoactive and mixed**; **hypoactive is the commonest and the most frequently missed**. Special phenotypes: **delirium superimposed on dementia (DSD)** — the hardest to diagnose and slowest to resolve, **postoperative delirium (POD)** versus **postoperative cognitive dysfunction (POCD)**, ICU delirium, subsyndromal and persistent delirium.
3. **Pathophysiology.** The **Systems Integration Failure Hypothesis (SIFH)** unifies the competing theories: **neuronal ageing, oxidative stress, neuroendocrine change, diurnal dysregulation and neuroinflammation** together disrupt the **neurotransmitter systems** (cholinergic, dopaminergic, adrenergic, histaminic, serotonergic) and **neuronal network connectivity**. This explains both the heterogeneous presentation and why **single-target drugs such as antipsychotics repeatedly fail** in trials.
4. **Predisposing and precipitating factors.** The more predisposing factors present (age, cognitive impairment, **frailty**, sensory impairment, multimorbidity, polypharmacy), the smaller the precipitant needed. The full list is the **DELIRIUM IS VERY PAINFUL** mnemonic. **Deliriogenic drugs** to know: benzodiazepines (especially IV), antipsychotics, corticosteroids, **H2-blockers in renal impairment**, opioids (especially **pethidine**), muscle relaxants, **antimuscarinics for overactive bladder**, first-generation antihistamines, and **zolpidem**.
5. **Diagnosis and evaluation.** Screen **all hospitalised older adults at least daily** (weekly in post-acute care, monthly in long-term care) with **CAM** (sensitivity 94%, specificity 89%), 3D-CAM, 4AT or CAM-ICU. Never explain away a positive item. **When in doubt, assume delirium until proven otherwise.** Investigate in a targeted way; imaging, LP and EEG only on specific indication.
6. **Management and prognosis.** (i) Identify and treat every precipitant. (ii) **Multicomponent non-pharmacologic care is the mainstay** — reorientation, sensory aids, sleep protocol, early mobilisation, hydration, removal of tethers, family presence, interprofessional delivery. (iii) Behaviour is managed by **T-A-DA (Tolerate, Anticipate, Don't Agitate)**, not restraints. (iv) **No drug is FDA- or EMA-approved for delirium**; **benzodiazepines must not be used** except in alcohol or benzodiazepine withdrawal; antipsychotics only for severe agitation posing danger, at the lowest dose for the shortest time, after non-pharmacologic measures fail. Prognosis: **up to 45% are still delirious at discharge**; persistence is **33% / 26% / 21%** at 1, 3 and 6 months; delirium **doubles the rate of cognitive decline** for five years in Alzheimer's disease.

Latest Update — 1 Point

> **7.** The **SCCM PADIS Focused Update 2025** (Crit Care Med 2025) has hardened the drug position: it recommends **against using haloperidol, atypical antipsychotics, dexmedetomidine, statins or ketamine to prevent delirium**, and could issue **no recommendation either for or against antipsychotics for treatment** because their effect on outcomes is minimal. Its three positive conditional recommendations are **dexmedetomidine over propofol** when light sedation or delirium reduction is the priority, **enhanced mobilisation/rehabilitation**, and **melatonin**.

Q30 (b). Delirium 6-MARK NOTE — 5 + 1

*Pivot for this version: **condensed bedside/rapid-recall version** for a shorter note — deliberately does not repeat the mechanism-heavy content above.*

Textbook Core — 5 Points

1. **Core concept.** Delirium is **acute brain failure**: an acutely developing, **fluctuating** disturbance of **attention** and cognition secondary to a medical cause. **Inattention is the core deficit** — agitation is optional and often absent.
2. **Recognise it.** Ask three things at the bedside: has this changed **acutely from baseline**? Can the patient **sustain attention** (months of the year backwards)? Is there an **identifiable medical trigger**? A positive **CAM or 4AT** flags the case; DSM-5 confirms it.
3. **Delirium versus dementia (must-write table).** See below.
4. **Find and fix the cause.** Work through **DELIRIUM IS VERY PAINFUL**. In practice the highest-yield checks are: **drug chart**, infection screen, **capillary glucose**, sodium and calcium, hydration, **bladder for retention**, rectum for impaction, oxygenation, and uncontrolled pain.
5. **Treat it.** Correct the cause + **multicomponent non-pharmacologic care** (reorientation, spectacles and hearing aids, sleep protocol, early mobilisation, hydration, family at the bedside, no restraints) + **T-A-DA** for behaviour. Reserve **low-dose haloperidol (0.25–0.5 mg)** with ECG monitoring for danger to self or others; **avoid benzodiazepines** unless alcohol or benzodiazepine withdrawal. Explain to relatives that **fluctuation and slow resolution are expected**.

Feature	Delirium	Dementia
Onset	Hours to days	Months to years
Course	Marked **fluctuation**, worse at night	Slowly progressive
Consciousness	**Altered and changing**	Clear until very late
Attention	**Impaired — the core deficit**	Preserved
Speech	Incoherent	Word-finding difficulty, aphasia
Hallucinations	Common, usually **visual**; illusions	Uncommon until late (except Lewy body dementia)
Sleep–wake cycle	Disrupted / reversed	Sleep disturbed, cycle less affected
CAM	Positive	Negative
Acute illness or drug	Usually present	Absent
Reversibility	Often (but not always) reversible	Irreversible

Latest Update — 1 Point

> **6.** A **2025 systematic review and meta-analysis** of 35 studies and 12,097 medically hospitalised older patients (Int J Nurs Stud) reported a **pooled prevalence of 23.6%** and a **pooled incidence of 13.5%**, and identified **frailty, physical restraints, previous falls, illness severity, cognitive impairment and older age** as the leading risk factors. The inclusion of **physical restraints** among the top risks is the practice-changing point — restraint is a cause of delirium, not a treatment for it.

Q31. Rating scales in delirium 7-MARK NOTE — 6 + 1

*Pivot: **the whole family of instruments and how to choose between them** (CAM itself is answered separately in Q32).*

Textbook Core — 6 Points

1. **Purpose and classification.** Scales are used to overcome the fact that delirium is **under-recognised in up to two-thirds of cases**. They fall into four groups: **(i) screening/detection** tools, **(ii) diagnostic** criteria-based instruments, **(iii) severity** scales for monitoring response, and **(iv) prediction** models. **A scale screens; DSM-5 or ICD-10 diagnoses.**
2. **Screening/detection tools (Pathy list, chronological): SPMSQ**, Delirium Rating Scale (DRS), **Confusion Assessment Method (CAM)**, Delirium Symptom Interview, Delirium Assessment Scale, **Cognitive Test for Delirium (CTD)**, **NEECHAM Confusion Scale**, Confusional State Evaluation, **Memorial Delirium Assessment Scale (MDAS)**, Delirium Index, Delirium Severity Scale, **CAM-ICU**, **DRS-R-98**, Delirium Detection Score, DDT-Pro, **bCAM**, **4AT**, and the Stanford Proxy Test for Delirium (S-PTD).
3. **Ward tools. CAM** is the most widely used (sensitivity 94%, specificity 89%). **3D-CAM** is a scripted **3-minute** version — sensitivity 96% / specificity 86% **in dementia**, and 93% / 96% **without dementia**. **4AT** takes under 2 minutes, needs no special training and works in drowsy or untestable patients, which is why NICE and SIGN recommend it. **bCAM** is the brief emergency-department version.
4. **Ultra-brief screens.** The best **single** item is **"months of the year backwards"** (sensitivity 83%, specificity 69%). The best **two-item** screen (UB-2) is **"months of the year backwards" + "what is the day of the week?"** (sensitivity 93%, specificity 64%). High sensitivity with low specificity means these are **rule-out** tools; a positive result demands fuller assessment.
5. **ICU and dementia-specific tools. CAM-ICU** is used in intubated or non-verbal patients: inattention is tested with the letter task (**"squeeze my hand for the letter A"** → SAVEAHAART), altered consciousness is any **RASS score other than 0**, and disorganised thinking uses yes/no questions. For **delirium superimposed on dementia**, the best single item is **"What day of the week is it?"**, the best two-item screen adds **days of the week backwards**, and the best three-item screen adds **"What type of place is this?"** and **"Does the patient appear sleepy?"**; sensitivities are 84%, 93% and 94% but specificities only 41%, 30% and 42%.
6. **Severity and prediction; frequency of use.** Severity and trajectory are tracked with the **MDAS**, **DRS-R-98**, **Delirium Index** and Delirium Severity Scale — useful in palliative care and research. Risk **prediction** uses the **E-PRE-DELIRIC** model and **Stanford's Algorithm for Predicting Delirium (SAPD)**, which allow preventive resources to be targeted. Screening frequency: **daily** in hospital, **weekly** in post-acute care, **monthly** in long-term care, and at any reported change from baseline.

Table — choosing the right instrument

Tool	Time	Category	Best setting	Performance
CAM	~5 min	Screening	General ward — the reference tool	Sens 94% / Spec 89%
3D-CAM	3 min	Screening	Ward, including patients with dementia	Sens 93–96% / Spec 86–96%
4AT	<2 min	Screening	Ward and ED; untrained staff; drowsy patients	NICE- and SIGN-recommended
UB-2	<1 min	Screening	Rapid triage / rule-out	Sens 93% / Spec 64%
bCAM	~2 min	Screening	Emergency department	Brief CAM derivative
CAM-ICU	2–3 min	Screening	Intubated / non-verbal ICU patients	Uses RASS + SAVEAHAART
MDAS	10 min	Severity	Palliative care, oncology; monitoring	Grades severity, not just presence
DRS-R-98	~20 min	Severity	Research, liaison psychiatry	Separate severity and diagnostic sections
E-PRE-DELIRIC	—	Prediction	ICU admission risk-stratification	Targets preventive resources

Latest Update — 1 Point

> **7. NICE CG103 (January 2023)** issued **new recommendations specifically on assessing and diagnosing delirium** and established the **4AT** as the recommended short assessment tool in both NICE and SIGN guidance — a definite shift away from CAM-only teaching, because the 4AT requires no training and can be completed in patients too drowsy to be formally tested. The **APA 2025** guideline reinforces this by requiring **systematic tool-based screening of at-risk patients** rather than clinical impression, and adds assessment **at transitions of care, including before discharge**.

Q32. Confusion Assessment Method (CAM) for the diagnosis of delirium 7-MARK NOTE — 6 + 1

*Pivot: **one instrument in depth — purpose, components, scoring, interpretation, advantages, limitations**.*

Textbook Core — 6 Points

1. **Purpose and status.** The CAM (Inouye et al., Ann Intern Med 1990) is a **structured bedside screening instrument** that operationalises the DSM criteria so that **non-psychiatrists can detect delirium reliably**. It is the most widely used delirium tool in the world and the parent of a family of derivatives. Note carefully: the CAM **screens** for delirium; the formal diagnosis still rests on **DSM-5 or ICD-10**.
2. **The four features. (i) Acute onset and fluctuating course**; **(ii) Inattention**; **(iii) Disorganised thinking**; **(iv) Altered level of consciousness**.
3. **Scoring algorithm (must be written exactly).** The screen is positive when the patient has **feature (i) AND feature (ii)**, **PLUS either feature (iii) OR feature (iv)**. In short: **(1 AND 2) + (3 OR 4)**. Features 1 and 2 are therefore **mandatory** — a patient cannot be CAM-positive without acute fluctuating onset and inattention.
4. **How to elicit each feature.**
   - **Feature 1** — from a **collateral informant**: is this a change from baseline, and does it come and go during the day?
   - **Feature 2 (inattention)** — **months of the year backwards**, days of the week backwards, or **digit span 3–4 backwards**; or simply observe that the patient is inappropriately distracted or cannot keep track of the conversation.
   - **Feature 3 (disorganised thinking)** — test **orientation to time and place**; observe an **illogical or rambling flow of ideas**.
   - **Feature 4 (altered consciousness)** — anything other than alert: vigilant, lethargic, stuporous or comatose.
5. **Performance and derivatives.** The CAM has a **sensitivity of 94% and specificity of 89%**, with high inter-rater reliability. Derivatives: **3D-CAM** (scripted 3-minute version — sensitivity 96% / specificity 86% in dementia, 93% / 96% without dementia); **CAM-ICU** for intubated patients (RASS ≠ 0 for feature 4, **SAVEAHAART** letter task for feature 2); **bCAM** for the emergency department; and the **UB-2** two-item screen derived from 3D-CAM validation data.
6. **Advantages and limitations.**
   - **Advantages:** rapid, bedside, no equipment, validated across settings, usable by nurses and non-specialists, high sensitivity and specificity, standardises documentation, and enables the daily screening that guidelines require.
   - **Limitations:** it needs **training and a collateral history** for feature 1; performance falls without formal cognitive testing of attention; it **cannot distinguish delirium from dementia in an unknown baseline** and is unreliable in established **DSD**; being a single point-in-time assessment it can be **falsely negative during a lucid interval**; it does not grade **severity** (use MDAS or DRS-R-98) and does not identify the cause; and it cannot be applied to a **comatose** patient.

CAM at a glance

Feature	Name	Bedside test	Required?
1	Acute onset & fluctuating course	Collateral history; observation over the day	**Mandatory**
2	Inattention	Months of the year backwards; days of the week backwards; digit span backwards	**Mandatory**
3	Disorganised thinking	Orientation to time and place; illogical flow of ideas	Either 3 **or** 4
4	Altered level of consciousness	Alert / vigilant / lethargic / stupor / coma	Either 3 **or** 4

Latest Update — 1 Point

7. Current guidance no longer treats the CAM as the only acceptable tool. **NICE CG103 (2023)** and SIGN recommend the **4AT** for routine ward and emergency screening because it needs **no training** and, unlike the CAM, remains scoreable in patients who are **too drowsy or untestable** to complete a formal interview. The **APA 2025** guideline requires systematic screening with **a** validated instrument — the CAM family remains fully acceptable, but the examinable point is that **the CAM is now one validated option among several rather than the default**.

Exam One-Liners — Q29 to Q32

- Acute confusional state = delirium = **acute brain failure**. Core deficit = **inattention**.
- Causes mnemonic = **DELIRIUM IS VERY PAINFUL**; cystocerebral syndrome = delirium from **urinary retention**.
- Pathophysiology = **Systems Integration Failure Hypothesis**.
- **Hypoactive delirium is the commonest and the most missed.**
- CAM = **(1 AND 2) + (3 OR 4)**; **sens 94% / spec 89%**.
- 3D-CAM = **3 minutes**; 4AT = **<2 minutes, no training, NICE/SIGN recommended**.
- CAM-ICU uses **RASS** and the **SAVEAHAART** letter task.
- Best single bedside item = **months of the year backwards**.
- Severity scales = **MDAS, DRS-R-98**; prediction = **E-PRE-DELIRIC, SAPD**.
- Screen **daily / weekly / monthly** in hospital / post-acute / long-term care.
- Mainstay of treatment = **non-pharmacologic multicomponent care**; behaviour tool = **T-A-DA**.
- **No drug is approved** for delirium; benzodiazepines only for alcohol/benzodiazepine withdrawal.

Q33. A 78-year-old man with type 2 diabetes mellitus presents to the emergency OPD in acute confusion with fever, headache, neck stiffness, vomiting and photophobia. What is your diagnosis and how will you manage this patient? 15-MARK ESSAY — 10 + 2

Diagnosis in one line: Acute community-acquired bacterial meningitis in an older diabetic man, presenting with the classic triad (fever, neck stiffness, altered mental status) plus meningism. **Streptococcus pneumoniae** is the most likely organism; **Listeria monocytogenes** must be covered because both **age > 60** and **diabetes mellitus** are established Listeria risk factors. This is a **medical emergency** — untreated mortality approaches 100%.

Textbook Core — 10 Points

1. **Clinical basis of the diagnosis.** The most common features of acute bacterial meningitis are **severe headache (84%)**, **fever > 38 °C (74%)**, **stiff neck (74%)**, **GCS < 14 (71%)** and **nausea (62%)**. The **classic triad occurs in only about 41–44%** of adults, but **95% have at least two of the four** cardinal features (fever, headache, stiff neck, altered mental status) — this patient has all four plus photophobia, so the pre-test probability is very high. Other features: **cranial nerve palsies (10–20%)** and **seizures (~30%)**. Importantly, **Kernig's and Brudzinski's signs are almost useless diagnostically (sensitivity 5% each; nuchal rigidity only 30%)** — their absence never excludes meningitis.
2. **The geriatric caveat.** Older patients over 60 actually present with the full triad **more** often than younger adults (58% vs 36%), but those with underlying disease such as **diabetes mellitus** or cardiopulmonary disease may instead present **insidiously with lethargy, confusion, anorexia, no fever and minimal meningeal signs**. **Confusion was present in 92% of elderly patients with pneumococcal meningitis and 78% with Gram-negative bacillary meningitis.** Therefore **any older patient with altered or changed mental status should be evaluated for bacterial meningitis, including consideration of CSF examination.** Elderly patients are also sicker at presentation, with higher CSF protein and more abnormal imaging, and an adverse outcome occurs in **51.9% of elderly versus 7.4% of non-elderly** patients.
3. **Likely organisms.** In adults **65 years or older**, the commonest pathogen is **Streptococcus pneumoniae (~70%)**, followed by **Listeria monocytogenes**, **Neisseria meningitidis**, **Haemophilus influenzae**, **Streptococcus agalactiae** and **aerobic Gram-negative bacilli**. Risk factors for Listeria are **age over 60, chronic steroids, alcoholism, immunosuppression, malignancy and diabetes**. S. pneumoniae carries the **highest mortality** and the highest rate of neurological sequelae (at least one major sequela in 24.7% of survivors). Look for an antecedent or concurrent focus — **sinusitis, otitis media or pneumonia**.
4. **Differential diagnosis to state briefly. Viral (aseptic) meningitis** — enterovirus, HSV-2, VZV, West Nile virus (elderly patients are more prone to West Nile encephalitis and to bacterial rather than viral disease); **HSV encephalitis**; **tuberculous meningitis** (very important in India — subacute course, cranial nerve palsies, basal exudates, hydrocephalus); **cryptococcal** and other fungal meningitis in the immunosuppressed; **subarachnoid haemorrhage** (thunderclap headache, neck stiffness, no fever early); **brain abscess, subdural empyema**; **cerebral malaria**; drug-induced aseptic meningitis; and **delirium from any systemic cause** including hypoglycaemia and hyperosmolar state in this diabetic.
5. **Immediate emergency actions (first 15 minutes).** Airway, breathing, circulation; oxygen; large-bore IV access; **capillary blood glucose immediately** (hypoglycaemia is the great mimic); vital signs; GCS; examine for **petechial or purpuric rash** (meningococcaemia), papilloedema, focal deficits, cranial nerve palsies and a septic focus in ear, sinuses and chest. Take **blood cultures**, then move directly to lumbar puncture or, if that must be deferred, to antibiotics. **Treatment must never be delayed for imaging or for the lumbar puncture** — the "one-hour window" is the accepted target for the first antibiotic dose.
6. **Investigations. Blood cultures** (before antibiotics), complete blood count, blood glucose (mg/dL), urea and creatinine (mg/dL), sodium and potassium (mEq/L), calcium (mg/dL), liver function tests, coagulation profile, CRP or procalcitonin, arterial blood gas and lactate, HbA1c, chest radiograph, HIV serology, and blood film for malaria where relevant. **The diagnosis rests on CSF examination after lumbar puncture** — send opening pressure, cell count with differential, protein, glucose with a **paired blood glucose** for the CSF:serum ratio, **Gram stain**, culture with antimicrobial susceptibility testing, and **PCR / multiplex meningitis-encephalitis panel**. Add CSF **CBNAAT and adenosine deaminase** if tuberculosis is suspected, and India ink and cryptococcal antigen if fungal disease is suspected.
7. **When to do CT before lumbar puncture.** Cranial imaging should **not** be performed routinely. Obtain CT first only if there is **immunosuppression, known CNS disease, focal neurological deficit, new-onset seizure, papilloedema or a markedly depressed conscious level**. In those situations, **start empirical antibiotics (with dexamethasone) before scanning**. Where imaging is not available, the lumbar puncture should be deferred until such features resolve — but antibiotics are still given immediately, and the LP performed as soon as it is safe.
8. **Empirical antimicrobial therapy.** For a patient **over 50 years** with suspected community-acquired bacterial meningitis, the recommended empirical regimen is **vancomycin + ampicillin + a third-generation cephalosporin (ceftriaxone or cefotaxime)**. The cephalosporin covers pneumococcus and meningococcus, **ampicillin covers Listeria** (which cephalosporins do **not** cover — a classic examiner trap), and **vancomycin covers penicillin- or cephalosporin-resistant pneumococci**. If the Gram stain shows **Gram-positive diplococci**, treat as pneumococcal with vancomycin plus a third-generation cephalosporin. De-escalate once culture and susceptibility results are available. Adult CNS doses from the textbook are given in the table below.
9. **Adjunctive dexamethasone.** Bacterial lysis by antibiotics triggers a **subarachnoid inflammatory response** that drives morbidity and mortality. In a randomised double-blind trial of 301 adults, adjunctive dexamethasone **reduced unfavourable outcomes and death**, with the greatest benefit in **pneumococcal meningitis** and in **moderate-to-severe disease by admission GCS**. Give **dexamethasone 0.15 mg/kg every 6 hours for 4 days**, **with or just before the first antibiotic dose**. Key qualifications: IDSA advises against starting it once antibiotics have already been given, whereas the **2016 European guideline allows up to 4 hours and the UK guideline up to 12 hours** after the

first dose; it should be **stopped if the organism turns out not to be pneumococcus**, and it is **harmful in Listeria and in cryptococcal meningitis**; benefit is **not seen in resource-poor settings with a high burden of advanced HIV**; and it may reduce CSF penetration of vancomycin and has been linked to **delayed cerebral injury**.

10. **Supportive care, complications, prognosis and prevention.** Nurse in a high-dependency area with neurological observations; manage **raised intracranial pressure** (head-up 30°, mannitol or hypertonic saline as a temporising measure; glycerol is **not** recommended); treat seizures; give maintenance **isotonic** fluids without routine fluid restriction, but allow moderate restriction if **SIADH** develops without hypovolaemia; monitor sodium closely; use an **insulin infusion or basal-bolus regimen targeting roughly 140–180 mg/dL** and stop metformin during acute illness; give VTE prophylaxis and pressure-area care; and apply the **delirium bundle** throughout, since confusion will persist after the infection is controlled. Watch for **hearing loss, seizures, hydrocephalus, cerebral infarction, septic shock and cognitive impairment**. Prevention: all adults **65 years or older** should receive pneumococcal vaccination (both conjugate and polysaccharide vaccines as per schedule), as should those with **asplenia, CSF leak, cochlear implants or immunosuppression**. Give **chemoprophylaxis to close contacts** if meningococcal disease is confirmed.

Table 1. CSF findings — pattern recognition

Parameter	Bacterial	Viral	Tuberculous	Cryptococcal
Opening pressure	**Raised in virtually all**	Normal / mildly raised	Raised	**Markedly raised**
Cells	**> 1000/mm^3 in 66%**, neutrophilic	Lymphocytic, usually < 500	Lymphocytic, 100–500	20–500 lymphocytes; may be normal in AIDS
Protein	**100–500 mg/dL**	Normal / mildly raised	**Very high**	Raised
Glucose	**< 40 mg/dL**; CSF:serum ratio low	Normal	Low	Normal or low
Microbiology	**Gram stain positive in 60–90%**, specificity ~100%	PCR	AFB, **CBNAAT**, ADA	India ink, **cryptococcal antigen**

Table 2. Empirical and definitive therapy (adult CNS doses, normal renal function)

Situation	Drug	Total daily dose & interval
Empirical, age > 50 years	Ceftriaxone *or* Cefotaxime	4 g/day (12–24 h) / 8–12 g/day (4–6 h)
	Ampicillin (for *Listeria*)	12 g/day, 4-hourly
	Vancomycin (resistant pneumococcus)	30–60 mg/kg/day, 8–12 h
S. pneumoniae, penicillin MIC ≥ 2.0	Vancomycin + third-generation cephalosporin	as above (consider adding rifampicin)
Listeria monocytogenes	Ampicillin or penicillin G (add an aminoglycoside)	Ampicillin 12 g/day; alternative co-trimoxazole
N. meningitidis, penicillin MIC < 0.1	Penicillin G or ampicillin	Penicillin G 24 million units/day, 4-hourly
Gram-negative bacilli	Third-generation cephalosporin	alternatives: meropenem, fluoroquinolone
HSV encephalitis suspected	**Aciclovir**	30 mg/kg/day, 8-hourly
Adjunctive steroid	**Dexamethasone**	**0.15 mg/kg 6-hourly × 4 days**, with/just before first antibiotic

Latest Updates — 2 Points

11. WHO guidelines on meningitis diagnosis, treatment and care (2025) — the **first global WHO guideline** on clinical management of acute community-acquired meningitis, developed under the *Defeating meningitis by 2030* road map (WHA73.9) and written specifically to be implementable in low- and middle-income countries. Points of direct examination value:

- **Lumbar puncture as early as possible, preferably before antimicrobials**; CSF Gram stain, cell count, protein, glucose and **CSF:blood glucose ratio** are strongly recommended, with **CSF PCR** strongly recommended in addition (culture with susceptibility testing remains the gold standard).
- **Cranial imaging is strongly recommended against routinely**; CT before LP only for **GCS < 10, focal signs, cranial nerve deficits, papilloedema, new-onset seizures in adults, or severe immunocompromise** — and **treatment must not be delayed** for it.
- **Empirical regimen: IV ceftriaxone or cefotaxime**, with **ampicillin or amoxicillin added whenever a Listeria risk factor is present — explicitly including age over 60 and diabetes mellitus**, exactly as in this patient. Vancomycin is added conditionally where pneumococcal resistance is prevalent (rifampicin is an alternative, but only where the tuberculosis burden is low). Chloramphenicol with ampicillin is the fallback if cephalosporins are unavailable.
- **Corticosteroids are now a strong recommendation** with the first antibiotic dose in non-epidemic settings where LP can be performed, to be **stopped if the CSF is not consistent with bacterial meningitis** or if an organism other than *S. pneumoniae* or *H. influenzae* type b is identified; maximum duration **4 days**. Dexamethasone is the steroid of choice, but hydrocortisone or methylprednisolone may substitute at equivalent dose.
- **Duration:** where no pathogen is identified, empirical therapy may be stopped at **7 days** if the patient has clinically recovered (afebrile, normal vitals, normal mental status for at least 48 hours).
- **Sequelae:** every patient should be **reviewed for sequelae before discharge and again within 4 weeks**, with **formal audiological screening before discharge** and early rehabilitation — a strong recommendation and an easy extra mark.
- **Glycerol is recommended against**; routine fluid restriction is recommended against.

12. Dexamethasone must be stopped once *Listeria* is identified. A prospective nationwide Dutch cohort (eClinicalMedicine 2023) of 162 adults with community-acquired *Listeria monocytogenes* meningitis found a **case fatality of 31%** and an **unfavourable outcome in 56%**, with **age and the standard adjunctive dexamethasone regimen as independent predictors of both unfavourable outcome and death**. This confirms the earlier French MONALISA signal and is now embedded in guidelines: **start dexamethasone empirically, but discontinue it the moment *Listeria* (or *Cryptococcus*) is confirmed.** In an older diabetic patient — who is precisely the Listeria-risk phenotype — this is the single most likely question the examiner will ask on follow-up.

Current Practice Update

Issue	Pathy 6th edition	Current practice (2023–2026)
Guideline anchor	IDSA 2004, ESCMID 2016, UK 2016	**WHO 2025 global meningitis guidelines** — the first WHO clinical guidance, LMIC-oriented
Steroid timing	IDSA: do not start after the first antibiotic dose; Europe up to 4 h; UK up to 12 h	**Strong recommendation** to give with the first antibiotic dose; benefit falls as delay increases; stop if CSF is non-bacterial
Steroid in *Listeria*	Flagged as harmful (French cohort)	**Confirmed harmful** in a second national cohort; guidelines mandate discontinuation
Molecular diagnosis	FilmArray panel described, false positives ~4%	**CSF PCR strongly recommended** alongside — not instead of — culture and susceptibility testing
Cranial imaging	CT before LP in defined groups	Same indications, but now a **strong recommendation against routine imaging**, with GCS < 10 as the explicit threshold
Duration of therapy	Pathogen-directed	**7 days** acceptable for culture-negative disease after clinical recovery
Follow-up	Sequelae described	**Mandatory pre-discharge review + audiological screening + review within 4 weeks + early rehabilitation**

Exam One-Liners — Q33

- Classic triad present in only **41–44%**; but **95% have 2 of 4** cardinal features.
- **Kernig's and Brudzinski's sensitivity is only 5%** — never rely on them.
- Commonest organism over 65 = **S. pneumoniae (~70%)**; then **Listeria**.
- **Diabetes and age > 60 are Listeria risk factors → always add ampicillin**.
- Cephalosporins do **not** cover *Listeria*.
- Empirical regimen > 50 years = **vancomycin + ampicillin + ceftriaxone/cefotaxime**.
- **Dexamethasone 0.15 mg/kg 6-hourly × 4 days**, with or just before the first antibiotic.
- **Stop dexamethasone if the organism is *Listeria* or *Cryptococcus*** — it increases mortality.
- Gram stain positive in **60–90%**, specificity ~100%.
- CSF glucose **< 40 mg/dL**, protein **100–500 mg/dL**, cells **> 1000/mm^3 in 66%**.
- **Antibiotics within 1 hour; never delay for CT or LP.**
- Adverse outcome in **51.9% of elderly vs 7.4% of non-elderly**.
- **Audiological screening before discharge** (WHO 2025).

Recurring Topic — Extra Points to Remember

> Delirium has now appeared four times in this set alone. These are the details that convert a safe answer into a top answer.

1. How to differentiate the four delirium questions

Question	Pivot to use	What NOT to spend words on
Acute confusional state (6 marks)	**Terminology + causes + differential diagnosis**	Detailed pathophysiology, prevention programmes
Delirium (7 marks)	**Full syndrome** — DSM-5, subtypes, SIFH, management, prognosis	Individual scale statistics
Delirium (6 marks)	**Bedside/rapid version** + delirium-vs-dementia table	Trial names and odds ratios
Rating scales (7 marks)	**Classification of tools** + how to choose	Causes and management — not asked
CAM (7 marks)	**One tool in depth** — components, scoring, advantages, limitations	Listing every other scale

2. Numbers to memorise

Item	Value
CAM sensitivity / specificity	94% / 89%
3D-CAM (with dementia / without)	96% & 86% / 93% & 96%
Months of the year backwards (single item)	Sens 83% / Spec 69%
UB-2 two-item screen	Sens 93% / Spec 64%
DSD screens (1 / 2 / 3 items) — sensitivity	84% / 93% / 94%
DSD screens — specificity	41% / 30% / 42%
Delirium in ICU / medical wards / hospice / LTC	30–80% / 18–35% / 26–44% / 16%
Pooled prevalence & incidence, hospitalised elderly (2025)	23.6% & 13.5%
Still delirious at discharge / 1 / 3 / 6 months	45% / 33% / 26% / 21%
Preventable proportion of delirium	30–40%
Meningitis — headache / fever / stiff neck / GCS < 14	84% / 74% / 74% / 71%
Meningitis — classic triad / at least 2 of 4	41–44% / 95%
Meningitis — Kernig's / Brudzinski's / nuchal rigidity sensitivity	5% / 5% / 30%
S. pneumoniae share of meningitis in adults ≥ 65 y	~70%
Listeria meningitis case fatality (2023 cohort)	31%; unfavourable outcome 56%
Dexamethasone regimen	0.15 mg/kg 6-hourly × 4 days

3. Named traps in this set

- **Trap:** writing that CAM *diagnoses* delirium. It **screens**; DSM-5/ICD-10 diagnose.
- **Trap:** writing the CAM algorithm as "any two features". It is **(1 AND 2) + (3 OR 4)**, with features 1 and 2 mandatory.
- **Trap:** equating delirium with agitation. **Hypoactive delirium is commonest.**
- **Trap:** prescribing a benzodiazepine for restlessness — it **causes and prolongs** delirium.
- **Trap:** physical restraint as a safety measure — it is an **independent risk factor**.
- **Trap:** relying on Kernig's and Brudzinski's signs to exclude meningitis (**5% sensitivity**).
- **Trap:** omitting **ampicillin** from the empirical regimen in an older or diabetic patient — cephalosporins miss *Listeria*.
- **Trap:** continuing dexamethasone after *Listeria* is isolated — it **increases mortality**.
- **Trap:** delaying the first antibiotic dose for a CT scan or lumbar puncture.
- **Trap:** forgetting to check **capillary glucose** in a confused diabetic before doing anything else.

4. Indian context lines to add

- **Under-recognition:** Indian referral-based studies detect delirium in under 1% of inpatients, while CAM-ICU screening finds **~30% of elderly ICU patients delirious, ~45% of them hypoactive** — the strongest argument for routine tool-based screening rather than waiting for a psychiatry referral.
- **Tuberculous meningitis** must appear in every Indian differential for fever with confusion and neck stiffness — send **CSF CBNAAT and ADA** alongside routine studies.
- **Rifampicin caution:** WHO 2025 permits rifampicin instead of vancomycin for resistant pneumococcus only in **low tuberculosis-burden** settings — in India, vancomycin is preferred.
- **Alcohol withdrawal** is a very common and under-declared cause of ward delirium in Indian men — always give **parenteral thiamine before glucose**.
- **Family attendants** at the bedside are the norm in Indian wards; formally enlisting them for orientation, hydration, feeding and mobilisation delivers an evidence-based delirium bundle at almost no cost.

- **Programmes to name: NPHCE** geriatric wards and regional geriatric centres; **AB-PMJAY** and the **Ayushman Vay Vandana Card** for those aged 70 and above; adult pneumococcal vaccination is **not** part of the Universal Immunization Programme and remains an out-of-pocket, opportunistic intervention — a legitimate policy-gap comment.

5. Fifteen-second answer skeletons

> **Any delirium question:** Define (acute, fluctuating, inattention, medical cause) → Subtypes → SIFH → Predisposing + Precipitating (DELIRIUM IS VERY PAINFUL) → Differentiate from dementia → Screen (CAM/4AT) then diagnose (DSM-5) → Targeted workup → Treat the cause → Non-pharmacologic bundle + T-A-DA → Drugs only for imminent harm → Prevent (HELP/mHELP/ABCDEF) → Prognosis and follow-up.
>
> **Any acute meningitis question:** Recognise (triad + meningism, but triad in <50%) → Resuscitate + glucose → Blood cultures → Decide on CT (only if GCS < 10, focal signs, papilloedema, seizure, immunocompromise) → **Lumbar puncture** → **Dexamethasone + antibiotics within 1 hour** (never delayed) → Empirical **vancomycin + ampicillin + ceftriaxone** over 50 years → De-escalate on culture; **stop steroid if not pneumococcal** → Supportive care, ICP, sodium, glycaemia, delirium bundle → Complications → **Audiology and sequelae review before discharge** → Vaccination and contact prophylaxis.

GERIATRIC PSYCHIATRY

Complete Compiled Question Bank & Model Answers

MD Geriatric Medicine — University Examination Preparation

32 questions across 9 subject sections, compiled and de-duplicated

Primary sources: Pathy's Principles and Practice of Geriatric Medicine, 6th ed.
Kane's Essentials of Clinical Geriatrics

Current evidence integrated: 2023 – present

Strict Mark-Wise Format | Indian Clinical & Policy Context Throughout

Table of Contents

Section A — Mood Disorders and Depression 2

1. Mood disorders in late life **[15 marks]** 3
2. Describe the prevalence, clinical presentation and management of depression in the elderly. What is th… **[15 marks]** 4
3. Depression in the elderly — treatment and management in older adults **[7 marks, with a 6-mark version]** 6
4. Antidepressant medication in late-life depression **[7 marks]** 7
5. Bipolar affective disorder in late life **[15 marks]** 8
6. Geriatric Depression Scale — administration and interpretation **[7 marks]** 9

Section B — Suicide, Self-Harm and Clinical Emergencies 2

7. Suicide in elders and suicide risk assessment **[7 marks]** 10
8. A 65-year-old woman presents with loss of appetite, lack of interest in daily work, considerable weigh… **[7 marks]** 10
9. A 60-year-old man was found unconscious in the fields with profuse frothing at the mouth. He was well… **[15 marks]** 12

Section C — Anxiety and Related Disorders 2

10. Give an account of anxiety disorders and their management **[15 marks]** 14
11. Anxiety in the elderly; generalised anxiety disorder **[7 marks, with a 6-mark version]** 15
12. Phobias in old age **[7 marks]** 16
13. Obsessive-compulsive disorder **[7 marks]** 17
14. Somatoform and conversion disorders in older adults **[7 marks]** 17

Section D — Psychotic Disorders in Late Life 2

15. Late-life psychotic disorders **[7 marks]** 19
16. Late-onset schizophrenia **[7 marks]** 20
17. Delusional disorder in the elderly **[15 marks]** 20

Section E — Grief, Personality and Behaviour 2

18. Grief, bereavement and complicated grief **[7 marks]** 22
19. Age-associated personality changes **[7 marks]** 22
20. Personality disorders in old age **[7 marks]** 22
21. Diogenes syndrome **[7 marks]** 24
22. Non-pharmacological management of the agitated older individual **[7 marks]** 25

Section F — Substance Use Disorders 2

23. Alcoholism in the elderly **[7 marks]** 26
24. Benzodiazepine dependence and withdrawal in the older adult **[7 marks]** 26
25. Nicotine dependence and smoking cessation **[7 marks]** 27
26. Substance misuse other than alcohol in older adults **[7 marks]** 28

Section G — Treatments and Therapeutics 2

27. Atypical antipsychotics — their uses and side effects in the elderly **[7 marks]** 30
28. Electroconvulsive therapy in older adults **[7 marks]** 31
29. Cognitive behavioural therapy **[7 marks]** 32

Section H — Sexuality in Later Life 2

30. Sexual dysfunction and its psychiatric associations in the elderly **[7 marks]** 33

Section I — Law, Ethics and Capacity 2

31. Mental Healthcare Act 2017 — provisions relevant to geriatric psychiatry **[15 marks]** 34
32. Capacity assessment and consent in the cognitively impaired patient **[15 marks]** 35

Appendix — Consolidated Night-Before Master Sheet 36

*Format used throughout — **7 marks = 6 textbook points + 1 latest update**; **6 marks = 5 textbook points + 1 latest update**; **15 marks = 10 textbook points + 2 latest updates**. Where two questions overlap in subject matter, each answer is written to a stated pivot point so that no answer repeats another. Questions that had appeared at two different mark weightings have been merged into a single entry carrying both versions.*

Section A — Mood Disorders and Depression

The classification of late-life mood disorder, depression in all its aspects, bipolar illness, and the screening instrument you will be asked to describe.

Question 1. Mood disorders in late life [15 marks]

Pivot: the whole mood-disorder spectrum and its classification. Depression-specific management is covered in Questions 2 to 4 and bipolar illness in Question 5.

Textbook Core — 10 Points

1. **Scope and classification. DSM-5 split the former "mood disorders" chapter into two — depressive disorders and bipolar disorders.** Depressive disorders comprise **major depressive disorder, persistent depressive disorder (merging dysthymia and chronic depression, ≥2 years), depressive disorder due to another medical condition, and substance/medication-induced depressive disorder**. Bipolar disorders comprise **bipolar I, bipolar II and cyclothymia**. Additions relevant to late life include the **"with anxious distress" specifier** and the **removal of the bereavement exclusion**.
2. **Burden. Depression is the commonest psychiatric disorder in older people** — major depression in **1–5% of the community**, **clinically relevant depressive symptoms in 8–16%**, rising markedly in **primary care, medical wards and long-term care**. **Bipolar disorder affects only 0.5–1% of over-65s but accounts for 5–10% of geriatric psychiatry outpatients. Clinically significant subthreshold syndromes increase with age** and still carry adverse outcomes.
3. **Why late-life mood disorder is different. Comorbidity is the hallmark of geriatric psychiatry, and this is most pronounced in depression.** Chronic physical illness and its drug treatment produce **overlapping symptoms — fatigue, slowness, loss of initiative and sleep disturbance**. Presentation is **atypical — somatic complaints, hypochondriasis, anxiety, irritability and cognitive complaints rather than expressed sadness**. **Psychomotor symptoms and nihilistic delusions are more frequently reported in older inpatients, sometimes even in the absence of lowered mood.**
4. **Depression subtypes of examination importance. Psychotic depression** — nihilistic and guilt delusions, high suicide risk, requiring antidepressant plus antipsychotic, or ECT. **Melancholic depression with psychomotor change** — predicts response to **TCAs, dopaminergic agents and ECT**. **"Vascular depression", now ischaemic subcortical depression or depression-executive dysfunction syndrome** — late onset, apathy, impaired insight, executive dysfunction and deep white-matter change; its clinical validity is now regarded as limited, with **vascular factors and inflammation mutually reinforcing each other**.
5. **Secondary and organic mood disorders — always exclude. Hypothyroidism and hyperthyroidism**, **vitamin B12 and folate deficiency**, **hyponatraemia (mEq/L)**, **hypercalcaemia (mg/dL)**, **stroke** (post-stroke depression; **right frontal or basotemporal stroke causing secondary mania**), **Parkinson's disease**, **dementia**, occult malignancy (especially pancreatic) and chronic pain. Drugs: **corticosteroids, levodopa and dopamine agonists, beta-blockers, interferon, and 5-alpha reductase inhibitors linked to depression and suicidality**.
6. **Bipolar disorder in late life.** Two groups — **early-onset illness ageing into old age (about two-thirds)** and **late-onset first episode after 50 (about one-third)**. **Late-onset mania is secondary mania until proved otherwise** — cerebrovascular disease, stroke, frontotemporal dementia, hyperthyroidism, steroids, levodopa and antidepressants. Mania is **attenuated — irritability, dysphoria, confusion and paranoia rather than euphoria**; **mixed states and prolonged depressive episodes dominate**. **GERI-BD** found **lithium and divalproex comparably effective** in acute mania in those aged ≥60.
7. **Conditions that mimic mood disorder. Apathy syndrome** — prevalent after stroke and in dementia, characterised by **lack of drive with absence of dysphoria and absence of neurovegetative symptoms**; **SSRIs and antidopaminergic drugs may worsen it**, and low-dose methylphenidate may help in severe cases. Also **delirium**, **dementia (pseudodementia versus true dementia)**, **complicated or prolonged grief**, and **personality disorder with mood lability**.
8. **Assessment.** History with **informant account**; **GDS-15**, or the **Cornell Scale for Depression in Dementia** where cognition is impaired; **MMSE/MoCA**; physical examination with attention to **vascular risk factors, weight, mobility, balance and ADLs**. Investigations: **CBC, sodium and potassium (mEq/L), calcium (mg/dL), glucose (mg/dL), liver function, TSH, vitamin B12 and folate**, plus **ECG before a TCA or lithium**. **Assess suicidal ideation in every case.**
9. **Principles of management across the spectrum. Match intensity to severity** — watchful waiting → psychotherapy → antidepressant → augmentation → ECT. **Antidepressants are as effective in older as in younger adults; psychotherapy is helpful in mild-to-moderate illness; ECT is probably more effective in severe depression in older people than in the young.** For bipolar illness — **a mood stabiliser at lower doses and lower serum levels, with antidepressant monotherapy contraindicated**. Always include **functional recovery, treatment of comorbid physical illness, and multidisciplinary team care**.

10. **Course, prognosis and impact.** Late-life depression has a **more chronic course** than in younger adults, although **over 80% of those without significant medical comorbidity or dementia recover and remain well when optimally treated**. **Worse outcome is predicted by greater severity of depression and more serious physical illness. Mortality is increased — mainly cardiovascular, with suicide risk rising with age, especially in men** — and **both severity and duration of symptoms predict mortality**, with loneliness amplifying the effect. Depression also **worsens the course of physical illness and increases healthcare consumption**.

Latest Updates — 2 Points

11. **Depression as a modifiable dementia risk factor — 2024 Lancet Commission.** The updated **Lancet Commission on dementia prevention, intervention and care (2024)** retains **depression among the modifiable risk factors** contributing to the population burden of dementia. Combined with cohort evidence that **10-year trajectories of depressive symptoms predict incident dementia**, this reframes **active treatment of late-life depression as a dementia-prevention intervention**, and mandates **cognitive assessment and cognitive follow-up in every older patient with depression**.
12. **OPTIMUM trial (*NEJM* 2023) and ISBD lithium targets.** In treatment-resistant late-life depression, **aripiprazole augmentation** outperformed **switching to bupropion** (**remission approximately 28% versus 19%**) with a **lower fall risk**. On the bipolar side, the **ISBD Older-Age Bipolar Disorder Task Force** now supports **age-stratified lithium targets of 0.4–0.8 mEq/L for ages 60–79 and 0.4–0.7 mEq/L for those aged ≥80**, rather than the standard adult range.

Question 2. Describe the prevalence, clinical presentation and management of depression in the elderly. What is the role of ECT in an elderly patient with depression? [15 marks]

Pivot: the full-spectrum essay, with a dedicated ECT section. See also Question 28 for ECT as a stand-alone note.

Textbook Core — 10 Points

1. **Prevalence. Major depression affects 1–5% of community-dwelling elderly**, but **clinically relevant depressive symptoms occur in 8–16%**. Rates rise with setting: **5–10% in primary care**, **10–20% of medical inpatients**, and highest in **long-term care**. In India, **LASI Wave-1 reported major depression in about 8% of those aged 60 and above**. It is the **commonest psychiatric disorder in old age**, yet remains **under-diagnosed and under-treated**.
2. **Risk factors. Female sex**, **widowhood and loneliness**, **physical illness and disability**, **chronic pain**, **cognitive impairment**, previous depression, **family history**, low socioeconomic status, caregiving, and **drug-induced depression** — steroids, beta-blockers, levodopa, and **5-alpha reductase inhibitors (finasteride, dutasteride), which are linked to depression and suicidality**.
3. **DSM-5 classification. Major depressive disorder** = **depressed mood and/or loss of interest for ≥2 weeks** plus a total of **≥5 of 9 symptoms** (worthlessness/guilt, poor concentration, fatigue, psychomotor change, insomnia/hypersomnia, appetite or weight change, recurrent thoughts of death). **Dysthymia is now "persistent depressive disorder" (≥2 years)**; the **"with anxious distress" specifier** was added and the **bereavement exclusion removed** — both especially relevant to older people.
4. **Atypical clinical presentation — the exam favourite.** Older people present with **somatic complaints and hypochondriacal preoccupation rather than sadness**; **"depression without sadness"** is common. Expect **psychomotor retardation or agitation**, **nihilistic delusions**, anxiety, irritability, **weight loss and insomnia**, **non-adherence to medical treatment**, and cognitive complaints.
5. **Important subtypes. Psychotic depression** — nihilistic and guilt delusions, higher suicide risk, needs antidepressant plus antipsychotic, or ECT. **Melancholic depression with psychomotor symptoms** — predicts response to **TCAs, dopaminergic antidepressants and ECT**. **"Vascular depression", now termed ischaemic subcortical depression or depression–executive dysfunction syndrome** — late onset, apathy, impaired insight, **executive dysfunction**, deep white-matter hyperintensities; its clinical validity is now regarded as limited.
6. **Differential diagnosis. Dementia** (depressive pseudodementia versus true dementia), **delirium**, **apathy syndrome** — distinguished by the **absence of dysphoria and absence of neurovegetative symptoms** — **hypothyroidism**, **B12 deficiency**, occult malignancy, Parkinson's disease, and normal grief.
7. **Assessment and workup.** History **plus informant history**; **GDS-15** (or the **Cornell Scale if dementia is present**); MMSE/MoCA; physical examination with attention to **vascular risk factors, weight, mobility, balance and ADLs**. Investigations: **CBC, sodium and potassium (mEq/L), calcium (mg/dL), glucose (mg/dL), liver function, TSH, vitamin B12 and folate**, with an **ECG before a TCA**. **Always ask directly about suicidal ideation — asking does not increase risk and most patients are relieved.**
8. **Non-pharmacological management. Watchful waiting is appropriate for mild depression**, since many improve spontaneously. **Psychotherapy is helpful in mild-to-moderate depression and is as effective in older as in younger adults** — **CBT, problem-solving therapy** (best where executive dysfunction is present), **interpersonal therapy, reminiscence and life review**. **Exercise is effective** in depressive disorder, minor depression and subthreshold symptoms. Add psychoeducation with family and **multidisciplinary outpatient team care**.

9. **Pharmacological management. Antidepressants are just as effective in older people as in younger adults. SSRIs are first-line** (sertraline, escitalopram); SNRIs and mirtazapine are alternatives; **start at half the adult dose and titrate slowly**; allow **6–12 weeks for full response**. On non-response to an SSRI, **switch antidepressant or add lithium**; **after TCA failure, lithium augmentation is probably the best option**. **Continue for at least 1 year after a first recurrence; consider lifelong maintenance after a second or subsequent recurrence.**
10. **Prognosis and impact.** The course is **more chronic than in younger adults**; among those without significant comorbidity or dementia who are optimally treated, **over 80% recover and remain well**. **Mortality is increased** — mainly cardiovascular and, to a lesser extent, suicide — and **both severity and duration of symptoms predict mortality**, especially where loneliness coexists. Depression impairs well-being and daily functioning **more than diabetes, heart disease or lung disease**.

Role of ECT — the Specific Sub-Question

- **Indications: severe depression with somatic complications, severe suicidality, catatonia, refusal of food and fluids, and psychotic depression** — in these, **immediate initiation of ECT is indicated**. It is also the option of choice in **pharmacotherapy-resistant depression**.
- **Positioning:** ECT **should be considered a first-line option in all patients with severe depressive symptoms, especially where psychotic features are present** — not merely a last resort.
- **Age and efficacy: Efficacy does not diminish with advancing age**, although the **seizure threshold rises**; indeed **older age is a predictor of better response to ECT**. **ECT rapidly relieves expressed suicidal intent.**
- **Utilisation: Older adults constitute more than half of all patients receiving ECT for depression**, yet it remains **under-utilised despite its relative safety and high effectiveness**.
- **Adverse effects: remarkably few in older age**, the commonest being **reversible cognitive side effects** — transient confusion, anterograde and retrograde amnesia. Pre-procedure cardiovascular assessment is required. There is **no absolute age limit and no absolute contraindication** (raised intracranial pressure and recent MI are relative).
- **Other geriatric indications: severe, treatment-resistant persistent agitation and aggression in dementia**, and refractory mania.
- **TMS as an alternative:** a non-invasive option for depression resistant to at least one antidepressant trial, with **lower risks than antidepressants**, but carrying some seizure risk after stroke or traumatic brain injury.

Latest Updates — 2 Points

11. **OPTIMUM trial (Lenze et al., *NEJM*, March 2023).** In **adults over 60 with treatment-resistant depression**, **augmentation of the existing antidepressant with aripiprazole** improved psychological well-being more than **switching to bupropion** — **remission approximately 28% for aripiprazole and bupropion augmentation versus 19% for a switch to bupropion** — with a **lower fall risk than the bupropion arm**. Step 2 showed lithium augmentation and a switch to nortriptyline were similarly modest. **Aripiprazole augmentation is therefore now the evidence-based next step** in late-life treatment-resistant depression.
12. **ECT versus ketamine (ELEKT-D, *NEJM* 2023) and modern ECT technique.** In **treatment-resistant depression without psychosis**, **subanaesthetic intravenous ketamine was non-inferior to ECT**, offering an alternative where ECT is refused or unavailable — but **ECT remains superior in psychotic depression, catatonia and acute suicidality**, all of which were excluded from that trial. Current practice favours **right unilateral ultrabrief-pulse ECT**, which gives **comparable efficacy with substantially less cognitive impairment** in older adults.

Current Practice Update

Aspect	Textbook	Current recommendation	Exam approach
Treatment-resistant depression	Switch antidepressant, or add lithium	**Aripiprazole augmentation first (OPTIMUM 2023)**; lithium and nortriptyline as later steps	Quote OPTIMUM with the remission figures
ECT positioning	Reserved for severe or resistant illness	**First-line where severe, psychotic, catatonic, suicidal or not eating**; ultrabrief right unilateral technique	Write "ECT is not a last resort" explicitly

Question 3. Depression in the elderly — treatment and management in older adults

[7 marks, with a 6-mark version]

Pivot: treatment framework only — the diagnosis is assumed established. Both the 7-mark and the condensed 6-mark version are given, since this question has appeared at both weightings.

Textbook Core — 6 Points

1. **Principles of treatment.** The indications mirror those in younger adults — **severity, duration, previous failed treatment, and patient preference**. **Watchful waiting for mild depression**; **psychotherapy or medication for mild-to-moderate illness**; **antidepressants and ECT have the most convincing evidence in major depression**. **Functional recovery, not just symptom relief, is always part of the goal.**
2. **The stepped-care model. Step 1** — recognition, psychoeducation, exercise, activity scheduling, watchful waiting. **Step 2** — structured psychotherapy (CBT, problem-solving therapy). **Step 3** — antidepressant, with or without psychotherapy. **Step 4** — switch, augment, or refer to specialist services. **Step 5** — **ECT** for severe, psychotic, catatonic or resistant illness.
3. **Non-pharmacological treatment. Psychotherapy is as effective in older as in younger adults, and many older adults prefer counselling to drugs**; there is currently **excessive reliance on drugs and under-use of psychotherapy**. Use **CBT, problem-solving therapy (best if executive dysfunction is present), interpersonal therapy, and reminiscence/life review**. **Exercise is effective**, including for subthreshold symptoms, and suits a stepped-care programme. Add **psychoeducation involving the family**.
4. **Pharmacological treatment. SSRIs are first-line**; **start low — half the usual adult dose — and go slow**; **assess response at 4 weeks and allow 6–12 weeks for full effect**; **never declare failure before an adequate dose has been given for an adequate duration**. Avoid **TCAs, paroxetine and benzodiazepines** wherever possible.
5. **Resistant depression and duration of treatment.** For non-response to an SSRI — **switch antidepressant (within or between classes) or add lithium**; after TCA failure, **lithium augmentation is probably the best option**; **ECT for pharmacotherapy resistance**. **Continue for ≥1 year after a first recurrence, and consider lifelong maintenance after a second or subsequent recurrence.**
6. **Service delivery and comorbidity.** Depression treatment in the elderly **requires a multidisciplinary approach par excellence** — **multidisciplinary outpatient teams are effective**, with nurses central to rehabilitation. Treat **coexisting physical illness, pain, sensory loss, alcohol misuse and loneliness**, review every drug that could be causing depression, and restore ADL function. Monitor **weight, sodium (mEq/L), falls and suicide risk** throughout.

Latest Update — 1 Point

7. **OPTIMUM trial (*NEJM* 2023).** In adults **over 60 with treatment-resistant depression**, **aripiprazole augmentation** of the existing antidepressant was superior to **switching to bupropion** (**remission approximately 28% versus 19%**) and carried a **lower fall risk than bupropion**, making it the **preferred next step** after SSRI or SNRI failure in late life.

Condensed 6-Mark Version — 5 Textbook Points + 1 Latest Update

Textbook Core — 5 Points

1. **Match treatment to severity. Mild — watchful waiting, exercise, activity scheduling. Mild to moderate — psychotherapy (CBT or problem-solving therapy). Moderate to severe — antidepressant, with or without psychotherapy. Severe, psychotic, catatonic, suicidal or not eating — ECT.**
2. **Prescribing rules in the elderly. SSRI first-line at half the starting dose, titrated slowly**; review at 2 weeks for tolerability and at 4–6 weeks for response; allow **up to 12 weeks** for full benefit. **One drug at a time; an adequate dose for an adequate duration before switching.**
3. **Non-response pathway. Switch antidepressant → augment (lithium, or aripiprazole) → ECT.** Never conclude "treatment resistance" without first confirming **adherence, adequate dose and duration, and the absence of an untreated medical cause**.
4. **Special situations. Depression in dementia** — use the **Cornell Scale**, prioritise psychosocial and behavioural measures; drug benefit is modest (the HTA-SADD trial showed no benefit from sertraline or mirtazapine). **Post-stroke depression** — treat actively, since it impairs rehabilitation. **Apathy** — this is not depression; **SSRIs may worsen it**; manage with activity scheduling, music therapy, outdoor activity, and in severe cases a trial of low-dose methylphenidate.
5. **Maintenance and relapse prevention. Continue for ≥1 year after a first recurrence and consider lifelong maintenance after a second.** Maintain the **same dose that achieved remission**; **taper slowly if stopping**; and continue **exercise, social engagement and structured follow-up** as relapse-prevention measures.

Latest Update — 1 Point

6. **USPSTF 2023 (*JAMA*).** A **Grade B recommendation to screen all adults, including those aged ≥65, for major depressive disorder** wherever systems exist for accurate diagnosis, treatment and follow-up — supporting the **collaborative and stepped-care model (PHQ-2 → PHQ-9 or GDS-15 → structured treatment → follow-up)** now built into India's **NPHCE and Ayushman Arogya Mandirs**, with referral through **Tele-MANAS 14416**.

Question 4. Antidepressant medication in late-life depression [7 marks]

Pivot: pure pharmacology — drug classes, choice, dosing, adverse effects and duration.

Textbook Core — 6 Points

1. **General principle. Antidepressants are just as effective in older people as in younger adults**; tolerability, not efficacy, dictates the choice. **"Start low, go slow — but go all the way"**: the commonest error is an inadequate dose for an inadequate duration. Response begins at **4 weeks and takes 6–12 weeks for full effect**, longer than in younger patients.
2. **SSRIs — first-line. Sertraline** (fewest interactions, safest after myocardial infarction) and **escitalopram** are the drugs of choice. Adverse effects: **hyponatraemia and SIADH (check sodium in mEq/L at baseline and at 2 weeks)**, GI upset, **falls and fracture**, **upper gastrointestinal bleeding, especially with NSAIDs or aspirin**, sexual dysfunction, and **QTc prolongation with citalopram and escitalopram** (maximum citalopram **20 mg/day above 60 years**). **Avoid paroxetine** — anticholinergic, short half-life, marked discontinuation symptoms. **Avoid fluoxetine** — very long half-life and CYP inhibition.
3. **SNRIs — second-line. Venlafaxine** (**dose-dependent hypertension**, prominent discontinuation symptoms) and **duloxetine** (useful when **neuropathic pain or stress incontinence** coexists; avoid in significant hepatic or renal impairment). Venlafaxine was **as effective as nortriptyline** in a randomised trial in elderly depressed inpatients.
4. **Other useful agents. Mirtazapine** — noradrenergic and specific serotonergic; the **drug of choice where insomnia, anorexia and weight loss coexist**; causes **sedation and weight gain**, rarely agranulocytosis. **Bupropion** — activating, no sexual dysfunction, but **lowers the seizure threshold**. **Vortioxetine** — possible pro-cognitive benefit. **Trazodone** — low dose for insomnia; causes **orthostatic hypotension**.
5. **Agents to avoid or use with caution. TCAs** — as effective as SSRIs but with far greater risk: **anticholinergic effects (delirium, urinary retention, constipation, glaucoma), orthostatic hypotension and falls, cardiac conduction delay, and lethality in overdose**. If used, **nortriptyline** is preferred, with a **baseline ECG**. **MAOIs** — tyramine and pressor-amine hypertensive crisis, and **fatal serotonin syndrome if combined with an SSRI or pethidine**. The **Beers Criteria list TCAs and paroxetine as potentially inappropriate in older adults**.
6. **Augmentation, duration and stopping.** For non-response to an SSRI — **switch, or add lithium**; **after TCA failure, lithium augmentation is probably the best option**; **ECT for treatment resistance**. **Continue at the remission dose for ≥1 year after a first recurrence and consider lifelong maintenance after a second or subsequent recurrence. Never stop abruptly** — taper over weeks to avoid a discontinuation syndrome of dizziness, flu-like symptoms, electric-shock sensations and insomnia.

Latest Update — 1 Point

7. **Antidepressant discontinuation (2023–2025).** A **2024 *Lancet Psychiatry* systematic review and meta-analysis** quantified discontinuation symptoms — commonest with **paroxetine and venlafaxine**, least with fluoxetine — and the **Maudsley Deprescribing Guidelines (2024)** established **hyperbolic tapering** (progressively smaller absolute dose reductions, guided by receptor occupancy rather than fixed steps) as the recommended method. This matters especially in older adults, in whom **withdrawal symptoms are easily mistaken for relapse**.

Current Practice Update

Aspect	Textbook	Current recommendation	Exam approach
Next step after SSRI failure	Switch, or add lithium	**Aripiprazole augmentation (OPTIMUM 2023)** ahead of lithium or nortriptyline	Name the trial and the drug
Stopping antidepressants	"Taper gradually"	**Hyperbolic taper over months**; distinguish withdrawal from relapse	Add one line on hyperbolic tapering

Question 5. Bipolar affective disorder in late life [15 marks]

Textbook Core — 10 Points

1. **Definition and terminology. Older-age bipolar disorder (OABD)** refers to bipolar disorder in those aged **≥50–60 years**. Two groups exist — **early-onset illness ageing into old age (about two-thirds)** and **late-onset disease with first episode after 50 (about one-third)**.
2. **Epidemiology.** Community prevalence is only **0.5–1%** above 65 years, but bipolar disorder accounts for **5–10% of geriatric psychiatry outpatients** and a large share of psychiatric emergency presentations. It is one of the three conditions classed as **severe mental illness (SMI)**, along with schizophrenia and schizoaffective disorder.
3. **Late-onset mania is "secondary mania" until proved otherwise.** Strong association with **cerebrovascular disease ("vascular mania")**, **right frontal or basotemporal stroke**, head injury, **frontotemporal dementia**, hyperthyroidism, and drugs — **corticosteroids, levodopa, antidepressants**.
4. **Clinical features.** Mania is **attenuated with age** — **irritability, dysphoria, confusion and paranoia** rather than classic euphoria. **Mixed states are common**; **depressive episodes are more frequent and longer**; cognitive impairment is prominent and may mimic dementia (**"pseudo-dementia of mania"**).
5. **Differential diagnosis. Unipolar depression** (the commonest misdiagnosis), **delirium**, **frontotemporal dementia**, agitation of dementia, alcohol/substance misuse, hyperthyroidism, and steroid-induced mood change.
6. **Assessment.** Full physical and neurological examination, complete drug review, **TSH, sodium (mEq/L), calcium (mg/dL), renal and liver function, B12**, ECG, cognitive testing, and **neuroimaging in every first-ever manic episode**. Always assess **suicide risk** — cardiovascular disease is the leading cause of death in SMI, but suicide remains a major contributor.
7. **Acute mania — management. Lithium or valproate** is first-line. The **GERI-BD trial** (the only RCT in those ≥60 years) found lithium and divalproex **comparably effective**, with lithium marginally superior on symptom reduction. Atypical antipsychotics (**quetiapine, aripiprazole, risperidone**) at **one-third to one-half adult dose**. **ECT** for severe, refractory or catatonic mania.
8. **Lithium in the elderly.** Remains the **gold standard for maintenance** and the only agent with a proven **anti-suicidal effect**. Use **30–50% lower doses** and lower target levels. Monitor **creatinine (mg/dL), eGFR, TSH, calcium, ECG**. Toxicity is precipitated by **thiazides, ACE inhibitors/ARBs, NSAIDs, dehydration, diarrhoea and fever**; **neurotoxicity can occur at apparently therapeutic levels**.
9. **Alternative mood stabilisers. Valproate** — monitor LFT, platelets, ammonia (**hyperammonaemic encephalopathy**), parkinsonism, weight gain. **Lamotrigine** — best for bipolar depression maintenance; needs **slow titration** because of rash and Stevens–Johnson syndrome. **Carbamazepine — best avoided** (enzyme induction, hyponatraemia, interactions). **Antidepressant monotherapy is contraindicated** — risk of switch and rapid cycling.
10. **Course, comorbidity and outcome.** High burden of **metabolic syndrome and cardiovascular disease**, greater risk of **cognitive decline and dementia**, poor medication adherence, functional impairment and **excess mortality (CVD death 2–3 times general population)**. Management must include **psychoeducation, adherence support, carer involvement, and aggressive cardiovascular risk factor control**.

Latest Updates — 2 Points

11. **ISBD Older-Age Bipolar Disorder Task Force (2025–26 systematic review, *Bipolar Disorders*).** Supports **age-stratified lithium targets** rather than the adult range of 0.6–1.2 mEq/L: **0.4–0.8 mEq/L for ages 60–79 years** and **0.4–0.7 mEq/L for those ≥80 years**, with more frequent renal monitoring.
12. **AGS Beers Criteria 2023 and current reviews (2024–25).** Beers flags **lithium with loop diuretics or ACE inhibitors** as potentially inappropriate unless levels are closely monitored. Contemporary reviews confirm **quetiapine and aripiprazole** as the preferred antipsychotics in OABD (least EPS and metabolic burden), and note a possible **neuroprotective / dementia-risk-reducing signal for lithium**.

Current Practice Update

Aspect	Textbook	Current recommendation	Exam approach
Lithium level	Standard adult therapeutic range quoted	ISBD age-stratified: **0.4–0.8 mEq/L (60–79 y)**, **0.4–0.7 mEq/L (≥80 y)**	Quote the age-stratified figures and attribute to the ISBD OABD Task Force
Cognition	Cognitive change described as secondary	Cognitive impairment recognised as a **core, often persistent feature** of OABD with increased dementia risk	Add cognitive assessment as a standing part of follow-up

Question 6. Geriatric Depression Scale — administration and interpretation [7 marks]

Textbook Core — 6 Points

1. **Purpose.** A **screening — not diagnostic — instrument** for depression in older adults, developed by **Yesavage and Brink (1982)**. Its defining feature is that it **deliberately excludes somatic items** (appetite, sleep, weight, libido, energy) that overlap with physical illness in old age, and instead asks about **mood, satisfaction and outlook**.
2. **Format and administration.** Simple **yes/no answers referring to the past one week**. Versions: the original **GDS-30**, the widely used **GDS-15**, and ultra-brief **GDS-10, GDS-5 and GDS-4**. It may be **self-administered or interviewer-administered** (read aloud for visual impairment or low literacy); **GDS-15 takes about 5 minutes**, GDS-30 about 10.
3. **Scoring. One point for each answer in the depressed direction**; some items are **positively worded and reverse-scored** (a "no" scores the point). **GDS-15: 0–4 normal, 5–8 mild, 9–11 moderate, 12–15 severe** — the usual screen-positive **cut-off is ≥5**. **GDS-30: a score ≥11 suggests depression.**
4. **Interpretation.** A positive score is **not a diagnosis** — it mandates a **full diagnostic interview against DSM-5 / ICD-11 criteria**, plus explicit assessment of **suicidal ideation** (asking about suicide does not increase risk and usually relieves the patient). Serial scores are useful to **monitor treatment response**.
5. **Advantages.** The **yes/no format suits cognitive and sensory impairment**; validated across **community, outpatient, hospital and long-term-care settings**; free of cost, brief, and **translated and validated in multiple Indian languages**; sensitivity and specificity around **80–90%** for GDS-15. The **GDS-3A subset** of three items can also screen for **apathy**.
6. **Limitations. Unreliable in moderate-to-severe dementia (MMSE below about 15)** — the **Cornell Scale for Depression in Dementia (CSDD)**, which uses informant input, should be used instead. Performance is affected by **education, literacy, culture and language**; it is **not valid in delirium**; and it does not assess severity for treatment selection or cover psychotic features.

Latest Update — 1 Point

7. **Screening policy (2023–2025).** The **USPSTF (2023, *JAMA*)** reaffirmed with a **Grade B recommendation that all adults, including older adults, be screened for depression** wherever systems exist for accurate diagnosis, treatment and follow-up. Current evidence supports **PHQ-2/PHQ-9 and GDS-15 as interchangeable first-line tools**, with the **Cornell Scale reserved for those with dementia**. In India, the **NPHCE** and **Ayushman Arogya Mandirs** now include routine depression screening of older adults at the primary-care level, with referral through **Tele-MANAS (14416)**.

Current Practice Update

Aspect	Textbook	Current recommendation	Exam approach
Tool of choice	GDS-15/GDS-30 listed as the assessment tool, PHQ-2 as screener	**PHQ-2 → PHQ-9 or GDS-15 are interchangeable**; CSDD if dementia present	Name GDS-15 first, then state when to switch to the Cornell Scale
Who to screen	Screen when clinically suspected	**USPSTF 2023 — screen all adults**, provided follow-up systems exist	Quote "USPSTF 2023, Grade B" for a guaranteed mark

Exam one-liners: *Yesavage & Brink 1982 · yes/no format · past one week · somatic items deliberately excluded · GDS-15 cut-off ≥5 · GDS-30 cut-off ≥11 · GDS-15 takes 5 minutes · invalid if MMSE <15 → use Cornell Scale · screening tool, never diagnostic.*

If asked for 15 marks, add: *the item list of the GDS-15 with reverse-scored items identified; comparison table of GDS vs PHQ-9 vs CSDD vs Hamilton and Montgomery–Åsberg scales; psychometric data (validation studies, Cronbach's alpha, ROC cut-offs by setting); use in special populations (stroke, Parkinson's disease, hospitalised, long-term care); Indian validation studies and LASI data on late-life depression prevalence; and the collaborative-care model of stepped depression management following a positive screen.*

Section B — Suicide, Self-Harm and Clinical Emergencies

Suicide risk in older people, and the two clinical vignettes that test it in practice.

Question 7. Suicide in elders and suicide risk assessment [7 marks]

Textbook Core — 6 Points

1. **Magnitude. Suicide rates are higher in older adults than in most other age groups and have risen over the last decade.** The **WHO estimates rates at age 75 of about 50 per 100,000 in men and 16 per 100,000 in women** — **older men account for considerably more deaths by suicide than women**. In India, **NCRB data show suicides among those aged 60 and above rose from about 11,000 in 2019 to over 15,000 in 2022 — a 40% increase, nearly double the rise in overall suicides**, with **illness the leading recorded cause**.
2. **Why elderly suicide is different — the high-yield contrast.** Older adults **give fewer warnings**, **use more lethal methods**, and have a far lower **attempt-to-completion ratio — about 4:1, compared with 200:1 in adolescents**. Intent is more often **determined and less impulsive**, so **every attempt in an older person must be treated as high risk**.
3. **The missed opportunity. Some 20–50% of older adults who die by suicide have seen their primary care physician within the preceding week**, and most have visited a care facility within the year. **Clinicians are less apt to decide that suicidal thinking in an older adult is a serious condition that may respond to treatment** — this ageist assumption is the central barrier to prevention.
4. **Risk factors. Depression is the strongest risk factor for suicide and for suicidal ideation**, and **non-fatal self-harm is the strongest risk factor for subsequent suicide**. Others include **alcohol and substance misuse, loneliness, recent loss, previous suicide attempt, unrelenting pain, loss of purpose, feelings of being a burden, feelings of being abandoned or rejected by family, and physical disability**, together with male sex, widowhood and living alone. **Protective factors are strong ties to social, family and religious support networks.**
5. **Risk assessment. Ask directly — asking about thoughts of death or suicide is not linked to an increased risk of an attempt; most patients are relieved, and it builds patient-physician trust.** A thorough assessment covers **not only stated suicidal wishes but also behaviours indicating hopelessness and intention to end one's life** — withdrawal, giving away possessions, settling affairs, refusing food or medication. Use **collaborative assessment involving family, nurse and social worker**, and formal instruments (**GDS-15**, Columbia Suicide Severity Rating Scale, Beck Hopelessness Scale). **Every older adult with suicidal ideas or self-harm needs assessment by a psychiatrist, preferably a geriatric psychiatrist.**
6. **Management and prevention. Ensure immediate safety** — admit where there is intent, a plan, psychosis or inadequate supervision; arrange **means restriction with family involvement**. **Prompt and vigorous treatment of depression reduces suicide risk**, and **ECT rapidly relieves expressed suicidal intent** in severe suicidality. At population level, **depression care managers and education of primary care providers reduce suicide risk regardless of depression severity**; **treat loneliness and social isolation**; and **involve the family in understanding the meaning of the suicidal thoughts**, with education and family therapy where appropriate.

Latest Update — 1 Point

7. **India's National Suicide Prevention Strategy, 2022 — the country's first, targeting a 10% reduction in suicide mortality by 2030.** Its pillars are **restricting access to means (including regulation of highly hazardous pesticides), strengthening health services and gatekeeper training, responsible media reporting, and improved suicide surveillance**. It specifically recommends **psychosocial support within the treatment plan for older adults with chronic or terminal illness**, a provision now being routed through **NPHCE**. This sits alongside **Section 115 of the Mental Healthcare Act 2017**, which **decriminalised attempted suicide** by presuming severe stress, and the national helplines **Tele-MANAS (14416)** and **Elder Line (14567)**.

Question 8. A 65-year-old woman presents with loss of appetite, lack of interest in daily work, considerable weight loss and insomnia. She is teary and upset during the consultation, has thoughts of worthlessness and has contemplated killing herself. What would be your approach? What classes of drugs can be given safely in this age group, what is their side-effect profile, and what would be your follow-up? [7 marks]

Textbook Core — 6 Points

1. **Recognise the diagnosis.** She fulfils DSM-5 criteria for **major depressive disorder, severe** — **anhedonia (lack of interest), anorexia, significant weight loss, insomnia, tearfulness, worthlessness and suicidal ideation** for at least two weeks. The presence of **weight loss, insomnia and psychomotor change suggests melancholic features**, which predict a good response to antidepressants and to ECT.

2. **Immediate priority — suicide risk assessment and safety. Ask directly and explicitly** about thoughts of death, intent, plan, timing, means, preparatory acts (settling affairs, giving away possessions), previous attempts and **hopelessness — asking does not increase risk**. Assess protective factors (family, faith, dependants). **Admit** if there is **active intent or a plan, psychotic features, refusal of food and fluids, severe agitation, or no reliable supervision at home**. Arrange **means restriction with the family, remove access to stored medicines, ensure supervision, and provide a written safety plan with crisis numbers — Tele-MANAS 14416 and Elder Line 14567**.
3. **Exclude a physical cause — do not assume the weight loss is psychiatric.** Full history, drug review and examination, then **CBC, ESR, sodium and potassium (mEq/L), calcium (mg/dL), fasting glucose (mg/dL), renal and liver function, TSH, vitamin B12 and folate, urine analysis, chest X-ray and ECG**, with further malignancy screening as indicated. Screen cognition (**MoCA**) and rate severity (**GDS-15** or PHQ-9) as a baseline for monitoring response.
4. **Drug classes that can be given safely, with their side-effect profiles** — see the table below.
5. **Complete the treatment package.** Add **psychotherapy — CBT or problem-solving therapy**, which is as effective in older as in younger adults; **psychoeducation with the family**; **exercise and activity scheduling**; **nutritional support** for the weight loss; and treat pain, sensory impairment and loneliness. **If she becomes psychotic or catatonic, refuses food and fluids, or remains acutely suicidal, ECT is indicated immediately — not as a last resort.**
6. **Follow-up plan. Review within 1 week** (earlier if discharged with suicidal ideation), then **weekly for the first month**, **fortnightly to 3 months**, then **monthly**. At each visit reassess **suicidal ideation, response (GDS/PHQ-9), adherence, weight and adverse effects**. **Check serum sodium (mEq/L) at baseline and 2 weeks after starting an SSRI**, and again after any dose increase. **Expect partial response by 4 weeks and full response by 6–12 weeks**; if there is no response by then, **switch or augment**. **Continue at the remission dose for at least 1 year**, and involve the family in relapse recognition.

Drug Classes and Side-Effect Profile

Class / drug	Why chosen here	Main adverse effects
SSRI — sertraline 25 mg → 50–100 mg (first-line)	Best evidence, safest cardiac profile, fewest interactions	**Hyponatraemia / SIADH**, GI upset, **falls and fracture**, **GI bleeding with NSAIDs**, sexual dysfunction
SSRI — escitalopram 5 mg → 10 mg	Alternative first-line agent	As above, plus **QTc prolongation** — maximum 10 mg in those ≥60 years
Mirtazapine 7.5 mg → 15–30 mg at night	**Best fit for this patient — insomnia, anorexia and weight loss**	Sedation, **weight gain**, dizziness, rarely agranulocytosis
SNRI — venlafaxine / duloxetine	Second-line; duloxetine if pain coexists	**Dose-related hypertension**, nausea, prominent discontinuation symptoms
Avoid — TCAs, paroxetine, benzodiazepines	—	Anticholinergic delirium, orthostatic hypotension, **cardiotoxicity in overdose**; benzodiazepines cause falls, cognitive impairment and dependence

Latest Update — 1 Point

7. **Suicide-risk screening and safety planning (2023–2025).** The **USPSTF 2023** issued an **I-statement — the evidence is insufficient to recommend universal suicide-risk screening in asymptomatic adults**, so the current standard is **targeted assessment in everyone who screens positive for depression**, as this patient does. The interventions with the best trial evidence for reducing subsequent attempts are the **Safety Planning Intervention combined with structured follow-up contact**, together with **means-restriction counselling involving the family**; both are embedded in the **Zero Suicide** care model.

Question 9. A 60-year-old man was found unconscious in the fields with profuse frothing at the mouth. He was well the previous night and had his usual dose of alcohol with his meal. He had incurred heavy debts after marrying off his daughter and had appeared depressed of late. How would you assess, investigate, diagnose and treat him based on evidence in literature? [15 marks]

> **Working diagnosis: Acute organophosphorus (anticholinesterase) compound poisoning, most probably deliberate self-poisoning** — the combination of an agricultural setting, unconsciousness with **profuse frothing (bronchorrhoea and salivation)**, recent **heavy debt**, **low mood** and **alcohol use** is the classic Indian examination presentation. **Differential diagnosis must still be considered:** generalised tonic-clonic seizure with post-ictal state, aluminium phosphide or carbamate or pyrethroid poisoning, snake bite (neurotoxic), heat stroke, hypoglycaemia, stroke, alcohol withdrawal seizure, and acute coronary syndrome with pulmonary oedema.

Textbook Core — 10 Points

1. **Immediate resuscitation comes before diagnosis. Airway, Breathing, Circulation, Disability, Exposure.** Position laterally, **suction copious secretions repeatedly**, give **high-flow oxygen**, and secure the airway by **early intubation** if there is a reduced conscious level, inability to protect the airway, uncontrolled bronchorrhoea or respiratory muscle weakness. Establish **two wide-bore IV lines**, attach a monitor, and check a **capillary blood glucose immediately**. **Wear gloves and an apron and decontaminate the patient** — remove all clothing and wash the skin and hair with soap and water — to protect both the patient and the staff from continuing dermal absorption.
2. **Recognise the toxidrome.** Anticholinesterase poisoning produces a **cholinergic crisis**. **Muscarinic (DUMBBELS)** — **D**iarrhoea, **U**rination, **M**iosis, **B**ronchorrhoea, **B**radycardia, **E**mesis, **L**acrimation, **S**alivation; **bronchorrhoea and bronchospasm are what kill**. **Nicotinic** — fasciculations, muscle weakness, cramps, **respiratory muscle paralysis**, tachycardia, hypertension, mydriasis. **Central** — anxiety, confusion, **seizures, coma and central respiratory depression**. A **garlic-like odour** on the breath or clothes supports the diagnosis.
3. **History and examination.** Obtain a history from **family, neighbours and the people who found him**; ask for the **container, label or a sample of the substance**, the **time of exposure and the approximate amount**, and the route. Document the **psychiatric history — depression, previous attempts, alcohol use, the debt and the recent marriage of his daughter**. Examine for **pupil size, secretions, chest crackles and wheeze, fasciculations, muscle power, reflexes, conscious level (GCS), temperature, and evidence of head injury, snake bite, or aspiration**. Grade severity using the **Peradeniya Organophosphorus Poisoning (POP) scale**.
4. **Investigations. Confirmatory** — **plasma (pseudo)cholinesterase and red cell acetylcholinesterase**; **RBC AChE correlates best with severity and with recovery**, and a **persistently low level is a useful diagnostic marker**. **Baseline and monitoring** — CBC, **sodium and potassium (mEq/L)**, **calcium and magnesium (mg/dL)**, **random blood sugar (mg/dL)**, renal and liver function, **serum amylase and lipase** (pancreatitis is common), **arterial blood gas**, **ECG** (look for **QTc prolongation, bradyarrhythmia and torsades**), **chest X-ray** (aspiration, pulmonary oedema), and **troponin** where indicated. Add a **toxicology screen and blood alcohol**, and **CT head** if the conscious level does not improve as expected.
5. **Atropine — the life-saving antidote, given by rapid dose escalation. Optimise oxygenation before giving atropine** to minimise the risk of dysrhythmia. Give an **initial IV bolus and then double the dose every 5 minutes** (for example 2 → 4 → 8 → 16 mg) until **atropinisation** is achieved. **Far larger doses are needed than for any other indication** — commonly tens of milligrams in the first day. **Endpoints of atropinisation: clear chest on auscultation, dry axillae, heart rate above 80/min, systolic BP above 80 mmHg, and pupils no longer pinpoint**. **Clearing of secretions, not pupil size alone, is the target.** Then maintain with an **infusion titrated to these endpoints**, and watch for **atropine toxicity — hyperthermia, delirium, ileus, urinary retention and tachyarrhythmia**.
6. **Oximes and benzodiazepines. Pralidoxime** reactivates acetylcholinesterase before **"ageing"** of the enzyme occurs, and is therefore most useful **early**; it acts mainly on the **nicotinic features — fasciculations, weakness and respiratory muscle paralysis**. Give as a **loading dose followed by a continuous infusion**. **Its clinical benefit is genuinely controversial** — Indian and international randomised trials have shown **no appreciable benefit of adding pralidoxime to atropine** on mortality, ventilation requirement or length of stay, and high-dose regimens have been associated with more respiratory complications; nonetheless it remains standard therapy in most Indian protocols. **Benzodiazepines (diazepam or midazolam) are indicated for seizures, agitation and fasciculations**, and are neuroprotective.
7. **Decontamination and supportive care. Gastric lavage only if the patient presents within about one hour, with a protected airway**, and it is **never** to be performed in an unprotected unconscious patient; **a single dose of activated charcoal** may be considered early. Otherwise **meticulous supportive care determines the outcome** — mechanical ventilation, **repeated suction**, fluid and electrolyte correction (especially **hypokalaemia and hypomagnesaemia**), glycaemic control, **nutrition**, **DVT and stress-ulcer prophylaxis**, pressure-area care, and prevention and treatment of **aspiration pneumonia** — the commonest cause of late death.

8. **Anticipate the three phases and their complications. (i) Acute cholinergic crisis** — minutes to hours. **(ii) Intermediate syndrome** — **24–96 hours after apparent recovery**, with **proximal limb, neck flexor, cranial nerve and respiratory muscle weakness**, requiring **ventilation**; anticipate it by serial **neck-flexion and single-breath-count testing**, and do not extubate prematurely. **(iii) Organophosphate-induced delayed polyneuropathy (OPIDN)** — **2–3 weeks later**, a distal sensorimotor axonopathy. Also watch for **arrhythmia and torsades, pancreatitis, hyperglycaemia and neuropsychiatric sequelae** — depression, cognitive deficits and recurrent seizures are documented in survivors.
9. **Manage the alcohol dimension.** He drinks regularly, so **anticipate alcohol withdrawal from 6–72 hours after admission**, which may be misread as atropine toxicity or as the intermediate syndrome. Give **parenteral thiamine before any glucose-containing fluid** to prevent Wernicke's encephalopathy, monitor with **CIWA-Ar where the patient is conscious**, use **short-acting benzodiazepines**, and correct **magnesium, potassium and phosphate**. Screen formally with **CAGE or SMAST-G** once he is awake, and treat the alcohol use disorder as part of the discharge plan.
10. **The psychiatric and medico-legal management — where most marks are lost.** Once medically stable, arrange a **formal psychiatric assessment before discharge** for every patient after deliberate self-poisoning. Diagnose and treat the **depressive illness** — the strongest risk factor for suicide — with an **SSRI plus psychotherapy**; **problem-solving therapy is particularly appropriate** given a debt-related, problem-focused crisis. Conduct a **structured suicide risk assessment** — intent, planning, ongoing wish to die, hopelessness, previous attempts — remembering that **an older person's attempt carries far higher lethality and a much lower attempt-to-completion ratio**. Arrange **means restriction and family supervision**, a written **safety plan**, **social work referral** for the debt, and **structured follow-up contact after discharge**. Medico-legally, **Section 115 of the Mental Healthcare Act 2017 presumes severe stress and protects him from prosecution**, and imposes a duty on the state to provide care and rehabilitation; document injuries carefully, preserve samples, and inform the police as required for a medico-legal case while maintaining a non-judgemental therapeutic stance. Signpost **Tele-MANAS 14416** and, given his age, **Elder Line 14567**.

Latest Updates — 2 Points

11. **2023 American Heart Association guidelines for the management of patients with cardiac arrest or life-threatening toxicity due to poisoning.** These recommend **immediate atropine for bronchospasm, bronchorrhoea, seizures or significant bradycardia in severe organophosphate poisoning**, with **rapid dose-doubling escalation** to achieve atropinisation, and note that **early oximes may be considered — particularly where there are fasciculations, weakness or paralysis — but "are not universally effective."** Recent reviews (2024–2025) additionally evaluate **magnesium sulphate, sodium bicarbonate, fresh frozen plasma and haemoperfusion** as adjuncts, all of which remain **investigational and are not standard of care**.
12. **Means restriction as the highest-yield preventive intervention. India's National Suicide Prevention Strategy 2022** — the country's first, targeting a **10% reduction in suicide mortality by 2030** — places **restricting access to highly hazardous pesticides** as its leading pillar, alongside gatekeeper training, responsible media reporting and improved surveillance; India has progressively **banned or restricted several highly hazardous pesticide formulations**, and WHO's **LIVE LIFE** framework cites national pesticide bans as the single most cost-effective population-level suicide-prevention measure. **NCRB data show suicides in those aged 60 and above rose about 40% between 2019 and 2022**, with **illness the leading recorded cause** — making systematic psychiatric follow-up of survivors such as this patient a national priority.

```
MANAGEMENT ALGORITHM — SUSPECTED ORGANOPHOSPHATE POISONING
  Unconscious + frothing + agricultural setting
        |
  A-B-C-D-E | staff PPE | REMOVE CLOTHES, WASH SKIN & HAIR | suction | O2
        |
  Check glucose  ->  intubate if GCS low / bronchorrhoea uncontrolled / muscle weakness
        |
  ATROPINE bolus, DOUBLE every 5 min (2->4->8->16 mg) until ATROPINISED:
     clear chest . dry axillae . HR >80 . SBP >80 . pupils not pinpoint
        |
  Maintain atropine INFUSION titrated to these endpoints (watch for atropine toxicity)
        |
  PRALIDOXIME loading dose + infusion (early; benefit debated)
  DIAZEPAM / MIDAZOLAM for seizures, agitation, fasciculations
        |
  Send plasma cholinesterase + RBC AChE | ABG | ECG (QTc) | amylase | CXR | electrolytes
        |
  ICU: ventilate, suction, correct K+/Mg2+, nutrition, prophylaxis, treat aspiration
        |
  WATCH: 24-96 h INTERMEDIATE SYNDROME (neck flexion, single-breath count)
         2-3 wk OPIDN | 6-72 h ALCOHOL WITHDRAWAL (thiamine BEFORE glucose)
        |
  BEFORE DISCHARGE: psychiatric assessment . treat depression . suicide risk assessment
         means restriction . social work for debt . safety plan . structured follow-up
         MHCA 2017 Sec 115 - no prosecution . Tele-MANAS 14416 . Elder Line 14567
```

Section C — Anxiety and Related Disorders

The anxiety spectrum, from the general essay through GAD, phobias, OCD and the somatoform disorders.

Question 10. Give an account of anxiety disorders and their management [15 marks]

Textbook Core — 10 Points

1. **Core principle. Disabling anxiety symptoms are not a normal part of ageing. Anxiety disorders are the most prevalent but most under-treated psychiatric disorders in older adults** — under-recognised because symptoms are attributed to physical illness or to ageing itself.
2. **Characteristic themes in late life.** Worries centre on **physical illness and its impact on quality of life — pain, disability and the possibility of death**, and are **exacerbated by feelings of isolation and dependence in hospital or long-term care**. The content is **realistic and health-focused**, unlike the occupational and existential worries of younger adults.
3. **Classification and relative frequency. Agoraphobia and panic disorder are the most common anxiety disorders in older adults**, followed by **phobias** — including the geriatric-specific **fear of falling** — and then **generalised anxiety disorder**. **Half of older adults with GAD have had symptoms for most of their lives; the other half developed GAD within the last five years.** Also relevant are **PTSD**, which may re-emerge in late life, and **obsessive-compulsive disorder**.
4. **The central diagnostic difficulty — comorbidity.** There is **substantial comorbidity of medical and anxiety disorders**, and **physiological symptoms of anxiety may themselves be a manifestation of a medical condition or an adverse effect of a prescribed drug** — this frequently confounds detection. **Incapacitating anxiety symptoms are common in COPD, in patients with pacemakers, and in Parkinson's disease.**
5. **Anxiety as a component of depression. Anxiety symptoms are a common component of depression in older adults, and their presence is associated with an increased likelihood of treatment resistance with antidepressants.** DSM-5 recognises this through the **"with anxious distress" specifier**. **Always screen a newly anxious older patient for depression — and treat the depression.**
6. **Secondary and drug-induced anxiety. Hyperthyroidism**, hypoglycaemia, phaeochromocytoma, arrhythmia, angina, pulmonary embolism, COPD and delirium. Drugs: **over-the-counter sympathomimetics, beta-2 agonists (albuterol/salbutamol), caffeine, nicotine, corticosteroids, dopamine agonists and thyroxine**; and **withdrawal states — alcohol and benzodiazepine withdrawal**. **New onset of panic attacks in an older person requires a thorough evaluation for an underlying general medical condition.**
7. **Assessment.** Detailed history plus informant account; **screen for depression, cognitive impairment, alcohol and drug use**; rate with **GAD-7, the Geriatric Anxiety Inventory, the Geriatric Anxiety Scale or HAM-A**. Investigations directed by the presentation — **TSH, glucose (mg/dL), calcium (mg/dL), ECG, oxygen saturation** — plus a **full drug review including over-the-counter preparations**.
8. **Non-pharmacological management — first-line. Relaxation training, mindfulness-based interventions and cognitive behavioural therapy are safer than and, in the long term, as effective as benzodiazepines.** Add **psychoeducation with family**, **sleep hygiene**, **reduction of caffeine, nicotine and alcohol**, **exercise**, correction of sensory impairment, and **graded exposure** for phobic avoidance. **Many older adults prefer counselling to psychopharmacological intervention.**
9. **Pharmacological management. SSRIs — escitalopram and sertraline — are considered first-line. SNRIs and vortioxetine are second-line. Buspirone** is an option, primarily for **GAD**. **The therapeutic effects of antidepressants and buspirone may take up to six weeks to become noticeable** — warn the patient, or treatment will be abandoned early. **Antipsychotics such as quetiapine should generally not be used for anxiety disorders, as the risks outweigh the benefits.**
10. **Benzodiazepines — the key caution.** They are **often effective in the short run but problematic**, carrying a **high risk of cognitive impairment, falls and fractures**. **Judicious short-term use of a short-acting agent such as lorazepam may be necessary if symptoms are incapacitating, only until other interventions become effective. Chronic use (beyond 120 days) is prevalent, growing, commoner in women, and at higher doses in older adults**; discontinuation is complex, best supervised by a psychiatrist, and **may need several months**. Note that **depression presenting with anxiety features is often inappropriately treated with a benzodiazepine rather than an antidepressant or psychotherapy**.

Latest Updates — 2 Points

11. **USPSTF 2023 (*JAMA*).** For the first time, a **Grade B recommendation to screen for anxiety disorders in adults aged 19–64**, but an explicit **I-statement — the evidence is insufficient to assess the balance of benefits and harms of screening in adults aged 65 and above**, with clinicians advised to use clinical judgement based on risk factors, comorbidity and life events. This is the single most quotable current statement on anxiety in old age and highlights the evidence gap.

12. **Mindfulness as an equal alternative to drug therapy (Hoge et al., *JAMA Psychiatry* 2023).** In a randomised trial in adults with anxiety disorders, an **8-week mindfulness-based stress reduction programme was non-inferior to escitalopram**, with **fewer adverse events** — supporting mindfulness-based intervention as a genuine first-line alternative in older adults, in whom drug adverse effects and interactions are the limiting factor.

Current Practice Update

Aspect	Textbook	Current recommendation	Exam approach
Screening	Based on clinical suspicion	**USPSTF 2023 — Grade B for ages 19–64, I-statement for ≥65**	Quote the age split precisely
Non-drug therapy	CBT and relaxation as alternatives to drugs	**Mindfulness non-inferior to escitalopram (2023)** — a first-line option, not merely an adjunct	Name the JAMA Psychiatry trial

Question 11. Anxiety in the elderly; generalised anxiety disorder [7 marks, with a 6-mark version]

Pivot: GAD-specific, so as not to repeat the general anxiety essay in Question 10. Both weightings are given, since this question has appeared as both a 7-mark and a 6-mark note.

Textbook Core — 6 Points

1. **Definition.** GAD is **excessive, difficult-to-control worry about multiple events or activities, present on most days for at least 6 months**, with **at least three of: restlessness, fatigue, poor concentration, irritability, muscle tension and sleep disturbance**, causing distress or functional impairment.
2. **Epidemiology and onset pattern. GAD is the anxiety disorder next in frequency after agoraphobia and panic disorder in older adults.** Crucially, **half of older adults with GAD have had symptoms for most of their lives, whereas the other half report developing GAD within the last five years** — so **both lifelong and genuinely late-onset GAD exist**, and late onset should prompt a search for a medical or drug cause.
3. **Clinical presentation in old age.** Worry is **realistic and health-centred** — about **illness, pain, disability, falls, finances, dependency and death**. Somatic presentation dominates: **palpitations, tremor, dizziness, gastrointestinal symptoms, muscle tension and insomnia**. Older patients typically present to a physician rather than a psychiatrist and **rarely volunteer the word "anxiety"**.
4. **Differential diagnosis. Depression with anxious distress** — the most important, since **anxiety symptoms in depression predict antidepressant treatment resistance**; **medical causes** — hyperthyroidism, arrhythmia, angina, COPD, hypoglycaemia, delirium; **drug-induced anxiety** — sympathomimetics, **beta-2 agonists such as albuterol**, caffeine, nicotine, steroids; and **withdrawal states — alcohol and benzodiazepine**. Also consider early dementia and akathisia.
5. **Assessment.** History plus informant account; **screen for depression first**; rate with **GAD-7, the Geriatric Anxiety Inventory or HAM-A**; conduct a full **drug and over-the-counter review**; targeted tests — **TSH, glucose (mg/dL), calcium (mg/dL), ECG, oxygen saturation**. Assess functional impact — avoidance, restricted mobility and carer burden.
6. **Management. Non-pharmacological first — CBT, relaxation training and mindfulness-based interventions are safe and, in the long term, as effective as benzodiazepines**; add sleep hygiene, reduction of caffeine and alcohol, and exercise. **Drugs: SSRIs (escitalopram, sertraline) first-line; SNRIs and vortioxetine second-line; buspirone is an option primarily for GAD. Warn that the effect may take up to six weeks. Do not use antipsychotics such as quetiapine** — the risks exceed the benefits. **Benzodiazepines only short-term, short-acting, and only where symptoms are incapacitating.**

Latest Update — 1 Point

7. **Mindfulness-based stress reduction is non-inferior to escitalopram (Hoge et al., *JAMA Psychiatry* 2023)** in adults with anxiety disorders including GAD, with **fewer adverse events** — a particularly important finding in older adults, where drug tolerability is the limiting factor. This is reinforced by the **2023 AGS Beers Criteria**, which list **benzodiazepines as potentially inappropriate in all older adults** irrespective of indication or duration of use.

Condensed 6-Mark Version — 5 Textbook Points + 1 Latest Update

Textbook Core — 5 Points

1. **Anxiety is not normal ageing. Anxiety disorders are the most prevalent but most under-treated psychiatric disorders in older adults.** In order of frequency: **agoraphobia and panic disorder → phobias (including fear of falling) → generalised anxiety disorder**.

2. **Always exclude the three mimics before diagnosing. (i) Medical disease** — hyperthyroidism, arrhythmia, angina, hypoglycaemia, COPD, delirium; **(ii) drugs** — sympathomimetics, **beta-2 agonists**, caffeine, nicotine, steroids, dopamine agonists; **(iii) withdrawal** — alcohol and benzodiazepines. **New onset of panic attacks in an older person always requires a thorough medical evaluation.**
3. **Then exclude depression. Anxiety symptoms are a common component of late-life depression**, and their presence **predicts antidepressant treatment resistance**. Treating the depression treats the anxiety; treating the anxiety alone with a benzodiazepine is a recognised prescribing error.
4. **Non-drug treatment is first-line. CBT, relaxation training and mindfulness-based interventions are safer than, and in the long term as effective as, benzodiazepines**, and many older adults prefer them. Add sleep hygiene, exercise, correction of sensory impairment, and reduction of caffeine, nicotine and alcohol.
5. **Drug treatment with the geriatric rules. SSRIs (escitalopram, sertraline) first-line; SNRIs and vortioxetine second-line; buspirone for GAD**; allow **up to six weeks** for effect. **Do not use antipsychotics for anxiety. Benzodiazepines only short-term, short-acting, for incapacitating symptoms** — they cause **cognitive impairment, falls and fractures**, and **chronic use beyond 120 days is common and difficult to reverse**, requiring psychiatrist-supervised withdrawal over months.

Latest Update — 1 Point

6. **USPSTF 2023 (*JAMA*).** A **Grade B recommendation to screen for anxiety disorders in adults aged 19–64**, but an explicit **I-statement for adults aged 65 and above — the evidence is insufficient**, with clinicians advised to screen on the basis of clinical judgement, risk factors, comorbidity and recent life events. Where screening is undertaken, use the **GAD-2/GAD-7 or the Geriatric Anxiety Inventory**.

Question 12. Phobias in old age [7 marks]

Textbook Core — 6 Points

1. **Definition.** A phobia is a **marked, persistent and excessive fear of a specific object, situation or activity**, out of proportion to the actual danger, leading to **avoidance** and functional impairment. Types: **specific phobia**, **social anxiety disorder** and **agoraphobia**.
2. **Epidemiology. Phobias are prevalent, chronic and often persist into old age** — most are long-standing rather than newly acquired. **Agoraphobia is, with panic disorder, the commonest anxiety disorder in older adults**, and in late life it is frequently **secondary** — arising after a fall, a stroke, a myocardial infarction or the onset of incontinence, rather than being primary.
3. **Fear of falling — the geriatric-specific phobia. A typical phobia seen in older adults, especially those with a history of fall and fracture, and rare in younger adults.** It affects **up to half of all fallers and a substantial proportion of non-fallers**. It produces **activity restriction → deconditioning, muscle weakness, gait change and social withdrawal → further falls** — a **self-fulfilling vicious cycle** that is itself an independent risk factor for future falls, disability and nursing-home admission. Measured with the **Falls Efficacy Scale–International (FES-I)** or the Activities-specific Balance Confidence scale.
4. **Other patterns in late life. Social anxiety** worsened by **deafness, tremor, incontinence, disfigurement or dysarthria**; **fear of medical procedures** (dental treatment, MRI, injections) leading to avoidance of necessary care; **fear of crime** and of leaving home, often mislabelled as agoraphobia; and **hypochondriacal phobia of illness**.
5. **Differential diagnosis. Depression with avoidance and apathy**; **realistic avoidance due to genuine physical limitation** (arthritis, orthostatic hypotension, visual loss) — this is **not** phobia; **paranoid disorder**, where the person avoids going out because of persecutory beliefs rather than fear; **early dementia** with loss of confidence; and **secondary avoidance in Parkinson's disease or after stroke**.
6. **Management. Graded exposure with response prevention is the treatment of choice**, delivered as **CBT**, and is effective in older adults. For **fear of falling** specifically, the evidence-based package is **supervised progressive strength and balance training (for example the Otago programme), gait-aid provision and training, home hazard assessment, footwear and vision correction, and CBT to correct catastrophic beliefs**. **Drugs are second-line** — **SSRIs** where comorbid depression or generalised anxiety is present. **Avoid benzodiazepines**, which in this group both worsen falls and reinforce avoidance.

Latest Update — 1 Point

7. **World Guidelines for Falls Prevention and Management (2022–2024, *Age and Ageing*) and subsequent updates** formally require that **"concern about falling" be assessed as a core component of every multifactorial falls assessment**, and recommend **exercise-based intervention combined with cognitive-behavioural strategies** rather than reassurance alone. Recent randomised and meta-analytic evidence (2023–2025) additionally supports **virtual-reality training and treadmill perturbation-based balance training** for reducing fear of falling and improving balance confidence in older adults.

Question 13. Obsessive-compulsive disorder [7 marks]

Textbook Core — 6 Points

1. **Definition. Obsessions** are recurrent, intrusive, unwanted thoughts, images or urges causing marked anxiety and recognised as one's own. **Compulsions** are repetitive behaviours or mental acts performed to reduce that anxiety, according to rigid rules. They are **time-consuming (more than one hour a day)** or cause marked distress or impairment. **DSM-5 moved OCD out of the anxiety disorders into "Obsessive-Compulsive and Related Disorders"**, alongside **body dysmorphic disorder, hoarding disorder, trichotillomania and excoriation disorder**.
2. **Epidemiology in old age.** Prevalence is around **1%** in older adults, and most cases are **long-standing, with onset in adolescence or early adulthood, persisting into old age**. Common themes are **contamination and washing, checking, symmetry, and religious or somatic obsessions**; in late life, **hoarding disorder** is disproportionately common and overlaps with **severe self-neglect (Diogenes syndrome)**.
3. **The critical rule — new-onset OCD in old age is organic until proved otherwise.** True de novo OCD after 60 is rare. Investigate for **frontal lobe and basal ganglia pathology** — **stroke (especially caudate and basal ganglia), frontotemporal dementia, Parkinson's disease, Huntington's disease, tumour and head injury** — and for **drug effects, notably dopamine agonists causing punding and impulse-control behaviours**.
4. **Differential diagnosis. Obsessive-compulsive personality disorder** — ego-syntonic traits without true obsessions or compulsions, and one of the **commonest personality disorders in old age**. Also: **depression with ruminations** (mood-congruent and not resisted), **generalised anxiety** (worries concern real-life problems), **dementia with repetitive or stereotyped behaviour**, **psychosis with delusions** (no insight, not resisted), and **hoarding disorder**.
5. **Assessment.** Direct enquiry, since patients rarely volunteer symptoms because of shame; informant history; rate severity with the **Yale-Brown Obsessive Compulsive Scale (Y-BOCS)**; screen for **depression, present in up to two-thirds, and for suicidality**; assess functional and carer impact. **Neuroimaging and cognitive testing where onset is late or the presentation atypical.**
6. **Management. CBT with exposure and response prevention (ERP) is first-line** and effective in older adults. **Drugs — SSRIs are first-line**, but note that **OCD requires higher doses and longer trials (10–12 weeks) than depression does**; **clomipramine** is effective but its **anticholinergic, orthostatic and cardiac effects make it a poor choice in the elderly**. For resistant cases, **augmentation with a low-dose atypical antipsychotic (risperidone, aripiprazole)**. **Avoid benzodiazepines** — ineffective for OCD and harmful in old age.

Latest Update — 1 Point

7. **Neuromodulation for OCD (2023–2025). Transcranial magnetic stimulation is approved for treating OCD** as well as for antidepressant-resistant depression, and **deep TMS using the H7 coil**, targeting the medial prefrontal and anterior cingulate cortex, now carries the strongest evidence for **treatment-refractory OCD**. Because **TMS carries lower risk than high-dose antidepressants in older adults**, it is an increasingly attractive option — but it **must be used judiciously in those at risk of seizures, such as after stroke or traumatic brain injury**. Deep brain stimulation remains reserved for the most severe refractory cases.

Question 14. Somatoform and conversion disorders in older adults [7 marks]

Textbook Core — 6 Points

1. **Definition.** Physical symptoms **not fully explained by a medical condition**, accompanied by **excessive thoughts, feelings and behaviours** about the symptoms, causing distress and impairment. DSM-5 renamed this group **"Somatic Symptom and Related Disorders"**.
2. **Spectrum. Somatic symptom disorder**, **illness anxiety disorder** (formerly hypochondriasis), **conversion disorder (functional neurological symptom disorder)**, factitious disorder, and psychological factors affecting a medical condition.
3. **The key geriatric rule. New-onset somatoform disorder after 60 years is rare.** A first presentation in old age must trigger a search for **occult organic disease, depression, anxiety, or early dementia**. **Depression is the commonest cause of medically unexplained somatic complaints in the elderly** ("masked depression").
4. **Clinical features. Multiple, shifting complaints** — pain, fatigue, dizziness, gastrointestinal symptoms; repeated consultations and investigations; **doctor-shopping**; poor response to reassurance. Conversion presents with **functional weakness, non-epileptic attacks, gait disorder (astasia–abasia), or aphonia**; **Hoover's sign** is a positive rule-in sign.
5. **Assessment. Proportionate but time-limited investigation**; screen for depression (**GDS-15**), anxiety, and cognition (MoCA); complete medication review; explore **bereavement, loneliness, elder abuse and role loss**. Diagnose functional disorder on **positive clinical signs**, not by exclusion alone.

6. **Management.** One **named coordinating physician**; **regular scheduled (not symptom-triggered) appointments**; **avoid repeat investigations and iatrogenic harm**; validate that the symptoms are real. **CBT is first-line**; treat comorbid depression/anxiety with **SSRIs**; graded **physiotherapy** for functional neurological disorder. **Avoid benzodiazepines and opioids.**

Latest Update — 1 Point

7. **ICD-11 and FND practice (2023–2025).** ICD-11 has replaced the somatoform group with **Bodily Distress Disorder**, graded mild / moderate / severe. **Functional Neurological Disorder is now a rule-in diagnosis** made on positive clinical signs, no longer a diagnosis of exclusion. Current evidence and UK/NICE-aligned FND guidance support **specialist physiotherapy plus CBT** as core treatment, with **clear explanation of the diagnosis itself being therapeutic**.

If asked for 15 marks, add: *detailed DSM-5 versus ICD-11 criteria comparison; epidemiology in Indian primary care; the biopsychosocial model with predisposing / precipitating / perpetuating factors; a table of positive rule-in signs for FND (Hoover's, hip-abductor, tremor entrainment, give-way weakness); prognosis and predictors of poor outcome; and the role of liaison psychiatry services.*

Section D — Psychotic Disorders in Late Life

Three genuinely distinct questions that overlap in content: the broad differential, the schizophrenia-spectrum diagnosis, and delusional disorder. Each answer is written to its own pivot so that the three do not repeat one another.

Question 15. Late-life psychotic disorders [7 marks]

Pivot: the differential diagnosis and classification of psychosis in old age as a whole. Question 16 pivots to late-onset schizophrenia and Question 17 to delusional disorder.

Textbook Core — 6 Points

1. **Definition and burden.** Psychosis is the presence of **delusions and/or hallucinations** with loss of contact with reality. There is an **increased incidence of psychotic symptoms in older adults compared with younger adults**; the lifetime risk of psychosis is about **23%**, and roughly **15% of older adults experience psychotic phenomena**.
2. **The single most important rule. Approximately 60% of new-onset psychosis in late life is secondary** — due to a medical, neurological or drug cause. **Primary psychosis is a diagnosis of exclusion. Older adults presenting with psychotic symptoms for the first time need a thorough evaluation before any psychiatric label is applied.**
3. **Classification — the "six D's" of late-life psychosis. Delirium** · **Disease** (medical/neurological — hypothyroidism, B12 deficiency, hyponatraemia, cancer, temporal lobe lesions, epilepsy) · **Drugs** (levodopa and dopamine agonists, steroids, anticholinergics, alcohol/substance use and withdrawal) · **Dementia** (Alzheimer's, and especially **DLB** and Parkinson's disease dementia) · **Depression** (psychotic depression with nihilistic/guilt delusions) · **Delusional disorder** and late-onset schizophrenia.
4. **Clinical clues that point to the cause. Fluctuating attention and altered consciousness → delirium. Recurrent, well-formed complex visual hallucinations of people, children or animals with parkinsonism, fluctuating cognition and REM sleep behaviour disorder → DLB**, where **misidentification delusions are the commonest type** and **severe neuroleptic sensitivity** may occur. **Nihilistic and guilt delusions with mood disturbance → psychotic depression. Well-systematised non-bizarre persecutory delusions with preserved function → delusional disorder. Purely visual hallucinations with intact insight in visual loss → Charles Bonnet syndrome** (not psychosis, needs no antipsychotic).
5. **Evaluation.** History plus **informant history**; full drug and alcohol review; cognitive testing (**MMSE/MoCA**); **audiometry and vision assessment**. Investigations: **CBC, sodium and potassium (mEq/L), calcium (mg/dL), glucose (mg/dL), renal and liver function, TSH, vitamin B12, VDRL/HIV, urine culture, ECG**, and **CT or MRI brain**. **If the workup is negative, refer to a psychiatrist to diagnose late-onset schizophrenia or delusional disorder.**
6. **Management principles. Treat the cause first** — stop or reduce the offending drug, correct the metabolic abnormality, treat the infection, treat the depression. **Environmental and psychosocial interventions are first-line** — correct sensory deficits with hearing aids and cataract surgery, reduce isolation, simplify the environment, and educate carers. **Antipsychotics are used judiciously, as needed**: **atypical agents at one-quarter to one-third of adult doses**, with **quetiapine or clozapine preferred in DLB and Parkinson's disease**, and **pimavanserin** for Parkinson's disease psychosis. Note the **boxed warning of increased stroke and mortality in dementia-related psychosis**.

Latest Update — 1 Point

7. **Xanomeline-trospium (KarXT), 2024–25.** The first **muscarinic M1/M4 receptor agonist antipsychotic**, FDA-approved in September 2024 for schizophrenia. Because it is **non-dopaminergic** it causes **no extrapyramidal symptoms, no prolactin elevation and no weight gain** — the 2025 *International Psychogeriatrics* overview of late-onset psychoses identifies it as the **most promising emerging therapy for late-life psychosis**, with trials in dementia-related psychosis ongoing.

```
APPROACH TO NEW-ONSET PSYCHOSIS IN AN OLDER ADULT
   New delusions and/or hallucinations after age 60
                |
   Is attention/consciousness fluctuating?  -- YES --> DELIRIUM: find & treat cause
                | NO
   Drug review (levodopa, steroids, anticholinergics, alcohol) -- positive --> withdraw/reduce
                |
   Metabolic + infective screen; B12, TSH, Na, Ca; neuroimaging -- positive --> treat DISEASE
                |
   Cognitive testing + informant history -- impaired --> DEMENTIA (AD / DLB / PDD)
                |
   Mood assessment -- depressed --> PSYCHOTIC DEPRESSION (antidepressant + antipsychotic or ECT)
                |
   Workup negative --> refer psychiatry: DELUSIONAL DISORDER or LATE-ONSET SCHIZOPHRENIA
```

Question 16. Late-onset schizophrenia [7 marks]

Pivot: the schizophrenia spectrum of late life — LOS and VLOSLP — and how it differs from early-onset disease.

Textbook Core — 6 Points

1. **Definitions and terminology (International Late-Onset Schizophrenia Group consensus). Late-onset schizophrenia (LOS)** = onset between **40 and 59 years**. **Very late-onset schizophrenia-like psychosis (VLOSLP)** = onset at **60 years or above**. The older European term for the latter is **late paraphrenia**. Both require the exclusion of dementia, delirium, mood disorder and organic causes.
2. **Epidemiology.** Onset of schizophrenia occurs **after 50 years in about 13% of hospitalised patients with schizophrenia, after 60 in 7%, and after 70 in 3%**. There is a **marked female predominance** in late-onset cases, in contrast to the male predominance of early-onset disease.
3. **Risk factors. Female sex**, **sensory impairment — particularly deafness and visual loss**, **social isolation and living alone**, never-married or childless status, **premorbid paranoid or schizoid personality traits**, migrant status, and a weaker family history of schizophrenia than in early-onset disease.
4. **Clinical features — how it differs from early-onset schizophrenia. Persecutory delusions dominate**, often with **partition delusions** (the belief that people, objects or radiation pass through walls, floors or ceilings) — highly characteristic. **Multimodal hallucinations** (auditory, visual, tactile, olfactory) are common. In contrast, **formal thought disorder, negative symptoms and affective flattening are rare**, and **premorbid function, personality and occupational history are relatively well preserved**. Cognitive impairment is milder and non-progressive.
5. **Differential diagnosis and workup.** Exclude **delirium, dementia (especially DLB and Alzheimer's disease), psychotic depression, mania, delusional disorder, drug-induced psychosis and Charles Bonnet syndrome**. Investigate as for any first-episode late-life psychosis: **CBC, sodium (mEq/L), calcium (mg/dL), glucose (mg/dL), renal and liver function, TSH, vitamin B12, VDRL/HIV, urine culture, ECG, cognitive testing, audiometry, visual assessment and neuroimaging**.
6. **Management. Engagement and trust are the first therapeutic task**, as insight is absent and patients resist the suggestion that they need treatment. **Correct sensory deficits — hearing aids and cataract surgery — and reduce social isolation. Atypical antipsychotics are preferred over conventional agents**, at **much lower doses than in younger patients**. Combine with **psychosocial rehabilitation — social skills training, cognitive remediation, supported accommodation** — plus **assertive community treatment and case management, which greatly increase success**. **Optimally control vascular risk factors** (obesity, hypertension, diabetes, dyslipidaemia), which are highly prevalent. Adherence is poor — consider **long-acting injectables**. Patients typically remain **stable but symptomatic and functionally impaired**, with **higher mortality than earlier-onset schizophrenia**, though **sustained remission can occur even in chronic illness**.

Latest Update — 1 Point

7. **The ATLAS trial remains the only randomised, placebo-controlled evidence in VLOSLP** (*Lancet Psychiatry*) — **amisulpride 100 mg daily was superior to placebo** in reducing psychosis symptoms over **12 and 24 weeks**, with a large effect size and good tolerability despite a modest increase in extrapyramidal symptoms. Reviews through **2025** continue to cite it as the standard, while the **2023 AGS Beers Criteria** permit antipsychotic use where the indication is **schizophrenia or bipolar disorder**, and **xanomeline–trospium (2024)** offers a non-dopaminergic alternative under study.

Current Practice Update

Aspect	Textbook	Current recommendation	Exam approach
Drug evidence	Atypicals preferred; no trial data specified	**Amisulpride 100 mg/day (ATLAS)** is the only RCT-supported regimen in VLOSLP	Name ATLAS and the dose — it distinguishes a good answer
First-line step	Drugs plus psychosocial rehabilitation	**Sensory correction and reduction of isolation before drugs**	Lead with hearing aids and cataract surgery

Question 17. Delusional disorder in the elderly [15 marks]

Pivot: delusional disorder as a discrete diagnosis, with its subtypes and its distinction from the wider late-life psychosis differential in Question 15.

Textbook Core — 10 Points

1. **Definition.** Persistent **non-bizarre delusions lasting ≥1 month**, without prominent hallucinations, disorganised speech or negative symptoms. **Personality and functioning are relatively preserved.**

2. **Terminology and burden.** Suspiciousness and paranoia affect **4–6%** of older people. Onset at **≥60 years** with schizophrenia-like features is termed **very late-onset schizophrenia-like psychosis (VLOSLP)**; the older term is **late paraphrenia**.
3. **Risk factors. Female sex**, **social isolation** and living alone, never-married status, **sensory impairment (deafness, poor vision)**, migrant status, premorbid **paranoid or schizoid personality**, and family history.
4. **Subtypes. Persecutory** (commonest), **jealous** (Othello), **somatic** (delusional infestation / Ekbom syndrome), **erotomanic** (de Clérambault), grandiose, and mixed.
5. **Clinical features.** Well-systematised, plausible delusions; **partition delusions** (belief that people or objects pass through walls, floors or ceilings) are characteristic of VLOSLP. Hallucinations, if present, are secondary. **Insight is absent** but daily function is maintained.
6. **Differential diagnosis. Delirium**, **dementia** (especially DLB and Alzheimer's disease), **psychotic depression**, drug-induced psychosis (**levodopa**, steroids, anticholinergics), alcohol/substance use, hypothyroidism, **vitamin B12 deficiency**, and temporal lobe lesions.
7. **Evaluation.** First-episode psychosis in old age **always needs a full workup**: CBC, sodium (mEq/L), calcium (mg/dL), glucose (mg/dL), renal and liver function, TSH, B12, VDRL/HIV, urine culture, complete drug review, cognitive testing (MMSE/MoCA), **audiometry and vision assessment**, and **neuroimaging (CT/MRI brain)**.
8. **Non-pharmacological management (first-line).** Patient engagement and trust-building; **correct sensory deficits — hearing aids, cataract surgery**; reduce isolation through day care and community services; supportive psychotherapy and **CBT for psychosis**; carer education. In India — **NPHCE day-care centres, Elder Line 14567, Tele-MANAS 14416**.
9. **Pharmacological management. Atypical antipsychotics preferred over conventional agents. Start low, go slow** — roughly one-quarter to one-third of adult doses: **risperidone 0.25–1 mg**, **olanzapine 2.5–5 mg**, **quetiapine 12.5–50 mg** (drug of choice if parkinsonism or DLB), **amisulpride 50–100 mg**.
10. **Adverse effects, monitoring, prognosis.** Watch for **EPS, tardive dyskinesia, falls, orthostatic hypotension, QTc prolongation, metabolic syndrome**, and the **boxed warning of increased stroke and mortality in dementia-related psychosis**. Adherence is poor — consider **long-acting injectables**. Course is chronic but function is preserved; VLOSLP carries **higher mortality** than earlier-onset schizophrenia.

Latest Updates — 2 Points

11. **Xanomeline–trospium (KarXT, 2024–25).** The first **muscarinic M1/M4 agonist antipsychotic**, FDA-approved in September 2024 for schizophrenia. Being **non-dopaminergic**, it causes **no EPS, no prolactin rise and no weight gain** — highlighted in the 2025 *International Psychogeriatrics* overview of late-onset psychoses as the most promising emerging option for older adults; trials in dementia-related psychosis are ongoing.
12. **Antipsychotic evidence base reaffirmed.** The **ATLAS trial** (Lancet Psychiatry) remains the only placebo-controlled RCT in VLOSLP — **amisulpride 100 mg/day** was superior to placebo over 12 and 24 weeks and well tolerated. The **2023 AGS Beers Criteria** continue to classify antipsychotics as potentially inappropriate in older adults **unless** the indication is schizophrenia, bipolar disorder, or short-term antiemesis.

Current Practice Update

Aspect	Textbook	Current recommendation	Exam approach
Drug class	Atypical antipsychotics preferred over conventional	Same, but with explicit boxed-warning caution; muscarinic agents (KarXT) emerging as non-dopaminergic alternative	Write "atypical, lowest dose, shortest duration" and add KarXT as the newest development
First-line step	Pharmacological + psychosocial rehabilitation	**Sensory correction and reduction of social isolation are first-line**; drugs added if distress or risk persists	Lead with non-pharmacological measures — examiners look for this

Section E — Grief, Personality and Behaviour

Bereavement, the personality questions — normal ageing, disorder and severe self-neglect — and the practical management of agitation.

Question 18. Grief, bereavement and complicated grief [7 marks]

Textbook Core — 6 Points

1. **Definitions. Bereavement** is the objective fact of loss; **grief** is the emotional response; **mourning** is its social and cultural expression. **Anticipatory grief** begins from the moment a life-limiting illness is diagnosed — for an elderly couple this marks the start of a painful parting.
2. **Normal grief.** Waves of **yearning, sadness, disbelief and searching behaviour**, sometimes with **transient hallucinations of the deceased** (culturally normal). Functioning gradually returns over weeks to months. **No active treatment is required.**
3. **Complicated (prolonged) grief.** Characterised by **extreme traumatic distress, numbness, disbelief, bitterness, feeling that part of oneself has died, and assuming the symptoms of the deceased**, with **symptoms enduring beyond 6 months** and causing functional impairment.
4. **Warning signals / risk factors. Sudden or unexpected death**, **traumatic death**, **loss of a child**, and **loss of a partner with an ambivalent relationship** ("can't live with, can't live without"). Also — lack of social support, female sex, older age, previous psychiatric illness, and death in an ICU.
5. **Consequences. Decline in health, increased healthcare utilisation and increased mortality** — highest in the first 6 months. Bereaved elders are at risk of **depression, cardiac events (including takotsubo cardiomyopathy), intercurrent infection, and suicide**. In India, **widowhood also brings loss of income, social status and property rights** — link to the **MWPSC Act 2007** and Elder Line **14567**.
6. **Management.** Normal grief needs **support, not medication** — family, faith and community, support groups, and practical help. Complicated grief responds to **grief-specific psychotherapy (complicated grief therapy), brief dynamic psychotherapy, and crisis intervention**. **Antidepressants are for comorbid major depression, not for grief itself.** Hospice programmes provide bereavement follow-up for about **one year**.

Latest Update — 1 Point

7. **Prolonged Grief Disorder is now a formal diagnosis** — in **DSM-5-TR (2022)** requiring **≥12 months** since the death in adults, and in **ICD-11** requiring **≥6 months**. The **Lancet 2025 Seminar on prolonged grief disorder** and RCTs in *JAMA Psychiatry* (Bryant 2024; Rosner 2025) confirm **grief-specific CBT / Prolonged Grief Therapy as first-line**, superior to interpersonal and present-centred therapy, with **antidepressants adding no benefit over psychotherapy alone**.

Current Practice Update

Aspect	Textbook	Current recommendation	Exam approach
Name and duration	"Complicated bereavement", symptoms enduring 6 months	**Prolonged Grief Disorder** — DSM-5-TR ≥12 months, ICD-11 ≥6 months	Write both names and both durations, attributing each correctly
Drug treatment	Antidepressants "may be considered" for complicated bereavement	Psychotherapy is first-line; antidepressants only for comorbid major depression	State psychotherapy first, then the antidepressant caveat

If asked for 15 marks, add: *Kübler-Ross and dual-process models of grief; full DSM-5-TR criteria list; differentiating grief from major depression (self-esteem preserved, waves vs pervasive low mood, suicidal thoughts of joining vs worthlessness); Inventory of Complicated Grief and PG-13-R as screening tools; grief in dementia caregivers; and cultural/Indian mourning practices as protective factors.*

Question 19. Age-associated personality changes [7 marks]

Pivot: normal, non-pathological ageing of personality, trait accentuation, and organic personality change. Personality disorder proper is Question 20.

Textbook Core — 6 Points

1. **Normal ageing of personality.** Personality is **largely stable across the lifespan**; the popular idea that people inevitably become rigid or cantankerous with age is not supported. Longitudinal studies show a **"maturation" pattern** — a modest **rise in agreeableness and conscientiousness and a fall in neuroticism, extraversion and openness to experience**. There is a shift towards **introversion, cautiousness, increased interiority and reminiscence**, with **better emotional regulation and improved positivity of affect**. **These are trends, not pathology.**

2. **Accentuation of traits.** Pre-existing traits are commonly **exaggerated rather than replaced** under the stress of ageing — losses, bereavement, retirement, illness, disability, sensory loss and dependence. A previously thrifty person becomes hoarding; a private person becomes reclusive; an anxious person becomes preoccupied with health. **Personality disorders in old age follow the same pattern — Cluster A becomes more prominent with isolation, Cluster B tends to attenuate, and Cluster C persists or worsens.**
3. **The critical clinical rule. A genuine change in personality beginning in old age is never normal ageing and is never a personality disorder — it is organic until proved otherwise.** A personality disorder by definition has its **onset by adolescence or early adulthood**.
4. **Causes of pathological personality change. Frontotemporal dementia — the prototype**, with **disinhibition, apathy, loss of empathy, perseverative and stereotyped behaviour, and hyperorality**, in a patient with relatively preserved memory. Also **Alzheimer's disease and other dementias**, **frontal lobe stroke, tumour or subdural haematoma**, **traumatic brain injury**, **normal pressure hydrocephalus**, **Parkinson's and Huntington's disease**, **epilepsy**, **hypothyroidism, B12 deficiency, neurosyphilis and HIV**, **alcohol-related brain damage**, and drugs — **steroids, dopamine agonists (impulse-control disorders, punding), benzodiazepines and anticholinergics**.
5. **Assessment.** The diagnosis rests on an **informant history establishing the premorbid personality and the timeline of change** — the patient often has no insight. Add **full neurological examination**, **cognitive testing with emphasis on executive and frontal function (Frontal Assessment Battery, clock drawing, verbal fluency)**, **drug and alcohol review**, screening for depression and mania, and investigations — **CBC, sodium (mEq/L), calcium (mg/dL), glucose (mg/dL), renal and liver function, TSH, vitamin B12, VDRL/HIV**, with **MRI brain** and, where indicated, EEG.
6. **Management. Treat the underlying cause** where reversible. Otherwise the approach is behavioural — a **consistent, coordinated team plan with clear boundaries**, **modification of the environment and of carer approach**, and **carer education and support**, since these behaviours rapidly drain family and staff resources. **Psychotropic medications generally do not reduce aggressive behaviour or mood lability due to personality**; use drugs only for a comorbid disorder or a crisis, and **avoid benzodiazepines**. Address **capacity, financial vulnerability, driving and safeguarding**, and hold **regular team meetings to prevent staff splitting and to manage negative counter-transference**.

Latest Update — 1 Point

7. **ICD-11 dimensional model of personality (in force since 2022, widely adopted 2023–25).** The categorical subtypes have been **abolished** in favour of a **single Personality Disorder diagnosis graded mild, moderate or severe**, described by **five trait domains — negative affectivity, detachment, dissociality, disinhibition and anankastia** — plus a **borderline pattern qualifier**. ICD-11 also retains **"personality change due to another medical condition"** as a separate, organic category. This dimensional framework fits older adults far better than the cluster system, since it captures **degree of severity and trait accentuation** rather than forcing an all-or-none categorical label; DSM-5-TR retains a parallel **Alternative Model in Section III**.

Question 20. Personality disorders in old age [7 marks]

Pivot: the diagnosable personality disorders, their clusters and their ageing course. Normal age-related personality change is Question 19.

Textbook Core — 6 Points

1. **Definition.** An **enduring, pervasive and inflexible pattern** of inner experience and behaviour that deviates markedly from cultural expectation, with **onset by adolescence or early adulthood**, causing distress or impairment.
2. **Prevalence.** About **10% of community-dwelling elderly** and up to **one-third of older psychiatric patients**. It is **under-diagnosed** because diagnostic criteria were validated in younger adults and depend on **work and relationship domains that are irrelevant to a retired, widowed person**.
3. **Clusters and their ageing course. Cluster A** (paranoid, schizoid, schizotypal) — often **becomes more prominent** with isolation and sensory loss. **Cluster B** (antisocial, borderline, histrionic, narcissistic) — impulsive traits tend to **"burn out" or attenuate**. **Cluster C** (obsessive-compulsive, avoidant, dependent) — **persists or worsens**; obsessive-compulsive and avoidant types are the commonest in old age.
4. **Age-specific presentation.** Expressed through **illness behaviour rather than work or sexual life** — **treatment refusal and non-adherence**, **staff splitting in care homes**, repeated crises, hoarding disorder, and **severe self-neglect (Diogenes syndrome)**, querulous or demanding behaviour, and carer burnout.
5. **Differential diagnosis — the critical point.** A **personality change that begins in old age is never a personality disorder**. It is **organic until proved otherwise** — **frontotemporal dementia**, stroke, head injury, tumour, hypothyroidism, alcohol, or medication effect. Also exclude depression, mania and psychosis.
6. **Management.** A **consistent, coordinated team approach** with a single agreed care plan and clear behavioural boundaries. **Psychotherapy (adapted CBT, DBT, schema-focused therapy) has evidence in older adults.** Treat comorbid depression, anxiety and substance misuse. **Medication only for comorbid disorder or crisis**; **avoid benzodiazepines**. Support and supervise family carers and care-home staff.

Latest Update — 1 Point

7. **ICD-11 dimensional model (in force since 2022, widely adopted 2023–25).** The categorical subtypes have been **abolished** and replaced by a **single diagnosis of Personality Disorder graded mild / moderate / severe**, described by **five trait domains — negative affectivity, detachment, dissociality, disinhibition and anankastia** — plus a **borderline pattern qualifier**. This dimensional approach fits older adults far better than the cluster system, and DSM-5-TR retains a parallel **Alternative Model (Section III)**.

If asked for 15 marks, add: *full list of the ten DSM categories with one-line descriptors; theories of trait stability versus change with ageing (maturation vs heterotypic continuity); assessment instruments (SIDP-IV, Gerontological Personality Disorder Scale, informant history); interaction of personality disorder with physical illness outcomes and rehabilitation; medico-legal aspects of self-neglect and capacity; and a worked care-plan example for Diogenes syndrome.*

Question 21. Diogenes syndrome [7 marks]

Textbook Core — 6 Points

1. **Definition.** Diogenes syndrome, also called **senile squalor syndrome** or **severe social breakdown syndrome**, is characterised by the tetrad of **extreme self-neglect, domestic squalor, social withdrawal and refusal of help**, usually accompanied by **hoarding (syllogomania)** and a **striking lack of shame or concern about the situation**. The name is a misnomer — the philosopher Diogenes was an ascetic who rejected possessions, whereas these patients accumulate them.
2. **Epidemiology and typical profile.** Incidence of about **0.5 per 1000 per year in those over 60**. Typically an **older person living alone**, often **widowed or never married**, with **above-average intelligence and a good previous educational and occupational record**, and frequently **not poor** — cases are often discovered to have substantial savings. Both sexes are affected. Presentation is usually precipitated by **a crisis — a fall, a fire, complaints from neighbours, or an emergency admission**.
3. **Classification. Primary (about half)** — no identifiable psychiatric disorder, arising from lifelong personality traits under stress. **Secondary** — associated with an underlying disorder, most often **frontotemporal dementia or other dementia**, **alcohol misuse**, **depression**, **schizophrenia and delusional disorder**, **obsessive-compulsive disorder and hoarding disorder**, and **mania**. Underlying **frontal-executive dysfunction** is the common thread.
4. **Predisposing factors. Premorbid personality** — **paranoid, schizoid, obsessive-compulsive and avoidant traits**, aloofness, suspiciousness, stubbornness and independence; personality disorder is the underlying cause of much provocative and hostile behaviour in older adults. Add **social isolation, bereavement or loss of a supporting relative, sensory impairment, chronic physical illness and poverty of social network**. A **precipitating loss** commonly triggers the decompensation.
5. **Clinical consequences and assessment. Malnutrition, vitamin deficiencies, anaemia, dehydration, hypothermia, infestation and skin infection, pressure ulcers, untreated chronic disease, falls and fire risk.** Assessment requires **a home visit** — the diagnosis cannot be made in clinic. Perform a **comprehensive geriatric assessment** with **cognitive testing including frontal-executive tasks, depression screening (GDS-15), alcohol history, nutritional assessment, and capacity assessment**; investigate with **CBC, albumin, vitamin B12 and folate, TSH, calcium (mg/dL), glucose (mg/dL), renal and liver function, and neuroimaging where cognitive impairment is suspected**.
6. **Management. Slow, patient engagement and trust-building** — **refusal of help is part of the syndrome**, and confrontation or forced clearance triggers withdrawal and a rapid return to squalor. Use a **multi-agency approach** — geriatrician, old age psychiatry, general practitioner, social work, community nursing, public health and environmental services, with **graded cleaning rather than a single mass clearance**. **Treat the treatable** — nutrition, infection, depression, alcohol misuse, psychosis. **Assess decision-making capacity formally**: if capacity is retained, an unwise choice must be respected while support continues; if capacity is absent, act in the person's best interests, using safeguarding and, in India, the framework of the **MWPSC Act 2007**, the **Mental Healthcare Act 2017** and **Elder Line 14567**. **Mortality is high — around 45% at five years** — and **relapse after cleaning is the rule without long-term follow-up**.

Latest Update — 1 Point

7. **Hoarding disorder as an independent diagnosis and current terminology (2023–2025). Hoarding disorder is now a distinct diagnosis in DSM-5 and ICD-11** within the **obsessive-compulsive and related disorders** group, separate from OCD, and defined by **persistent difficulty discarding possessions due to a perceived need to save them, with resulting congestion of living areas**, with an **"with poor or absent insight/delusional beliefs" specifier**. Prevalence rises with age and is around **6% in older adults**. Contemporary literature increasingly favours the neutral terms **"severe domestic squalor"** and **"self-neglect"** over the eponym, and recommends **specialised CBT for hoarding (with motivational interviewing and skills training rather than forced clearance)**, delivered alongside **multi-agency safeguarding**, as the treatment with the best evidence.

Question 22. Non-pharmacological management of the agitated older individual [7 marks]

Textbook Core — 6 Points

1. **Scale and rationale. Agitation and aggressive behaviours will be seen at some point in almost all individuals with dementia**, and behavioural and psychological symptoms of dementia are the **commonest psychiatric problem in nursing homes**. Non-drug management is first-line because **no medication is FDA-approved for treating the neuropsychiatric symptoms of dementia**, and all antipsychotics carry a **boxed warning of increased stroke and mortality** in this population.
2. **Step 1 — search for and treat the medical cause. A medical workup often identifies one or more causes.** Look for **pain** (the single most important and most missed), **infection, especially urinary tract infection**, **dehydration**, **constipation and urinary retention**, hypoxia, hypoglycaemia, electrolyte disturbance, **delirium**, sensory deprivation, and **drug effects — anticholinergic medications**, benzodiazepines, opioids, steroids and dopamine agonists. **Treating pain alone significantly reduces agitation in nursing-home residents (Husebø cluster RCT).**
3. **Step 2 — identify the trigger. Environmental triggers** (excessive noise, poor lighting, clutter, overheating, unfamiliar surroundings, over- or under-stimulation); **psychosocial triggers** (boredom, loneliness, loss of routine); and **interpersonal triggers** (a counter-therapeutic approach by family or professional carers — rushing, confrontation, arguing, task-focused care). **Once these modifiable causes are addressed, the behaviour often improves sufficiently that no further intervention is needed.**
4. **Step 3 — the structured frameworks. DICE — Describe the behaviour, Investigate causes, Create a plan, Evaluate the outcome. ABC analysis — Antecedent, Behaviour, Consequence**, recorded in a behaviour diary over several days to reveal the pattern. **TREA — Treatment Routes for Exploring Agitation**, matching the intervention to the unmet need behind the behaviour. Multiple sources of information may be needed in long-term care, **including video recordings of the behaviour**.
5. **Step 4 — specific evidence-based interventions. Person-centred, individually tailored activity** based on the person's life history and preferences; **music therapy**; **massage and therapeutic touch**; **animal-assisted therapy**; **multisensory stimulation (Snoezelen)**; **bright light therapy and sleep-wake regulation for sundowning**; **exercise and outdoor activity**; **reminiscence therapy**; **doll and simulated-presence therapy**; and **aromatherapy**. Network meta-analytic evidence ranks **massage therapy, animal-assisted intervention and personally tailored intervention** as producing the most substantial reductions in agitation.
6. **Step 5 — communication, environment and staff.** Approach from the front, at eye level, **one carer at a time**; **speak slowly with short simple sentences, one instruction at a time**; **do not argue, correct or confront — validate the emotion and redirect**; **offer choice and allow time**. Modify the environment — **consistent daily routine, familiar objects, adequate lighting, reduced noise, safe wandering space, visible clocks and calendars**. **Education and training of all healthcare providers working in long-term care homes is key**, together with **carer support to prevent burnout**. **Reserve antipsychotics for severe, distressing or dangerous behaviour that persists after these measures, at the lowest dose, with a defined review and withdrawal date.**

Latest Update — 1 Point

7. **International Psychogeriatric Association consensus definition of agitation in cognitive disorders and the 2023–2025 evidence base.** The IPA provisional consensus definition requires **excessive motor activity, verbal aggression or physical aggression causing excess distress or disability, beyond that attributable to another disorder** — giving a standard outcome definition for trials. Recent network meta-analyses and umbrella reviews (through 2025) confirm that **personally tailored activity, massage and touch therapy, animal-assisted intervention, music-based therapy and exercise outperform usual care**, and that **non-pharmacological interventions should be tried first and continued even when a drug is added**. Where a drug is unavoidable, **brexpiprazole (FDA-approved May 2023)** is now the only agent licensed for agitation in Alzheimer's dementia, but it retains the class mortality boxed warning.

```
DICE PATHWAY FOR THE AGITATED OLDER PERSON
 D  DESCRIBE   - what exactly happens, when, where, who is present, how often
 I  INVESTIGATE- pain? infection? constipation/retention? dehydration? drugs?
                 delirium? sensory loss? boredom? carer approach? environment?
 C  CREATE     - treat the cause + tailored activity, music, touch, exercise, light,
                 routine, communication technique, carer training, safe environment
 E  EVALUATE   - re-rate behaviour (Cohen-Mansfield Agitation Inventory), adjust plan
                 -> only if severe/dangerous and persisting: lowest-dose antipsychotic
                    with consent, monitoring, and a fixed review/withdrawal date
```

Section F — Substance Use Disorders

Alcohol, prescribed sedatives, tobacco and the wider substance-misuse question.

Question 23. Alcoholism in the elderly [7 marks]

Textbook Core — 6 Points

1. **Definition and classification.** Alcohol use disorder is a problematic pattern of use causing clinically significant impairment or distress. In older adults it is classified by **onset**: **early-onset ("survivors")**, about two-thirds, with lifelong drinking, more physical damage and more personality and social disruption; and **late-onset ("reactors")**, about one-third, beginning after 60 in response to **bereavement, retirement, loneliness, pain or physical illness**, with better prognosis and better treatment response.
2. **Altered pharmacokinetics — why safe limits are lower. Reduced total body water and lean body mass** raise the blood alcohol concentration for any given intake; **reduced hepatic metabolism and first-pass gastric alcohol dehydrogenase** prolong exposure; and there is **increased CNS sensitivity**. **The same drink produces a substantially higher blood level and greater impairment than in a younger adult**, so recommended limits are lower — no more than about **one standard drink per day and two on any occasion** above 65 years.
3. **Why it is missed.** Older people **present with physical problems rather than with a complaint of drinking**; symptoms are attributed to ageing or dementia — **falls, confusion, incontinence, self-neglect, malnutrition, poor diabetic or hypertensive control, insomnia and depression**. There is **stigma and under-reporting**, an absence of the usual work and legal markers in a retired person, and screening tools validated in the young. Clinicians simply do not ask.
4. **Consequences in old age. Falls, fractures and head injury (including chronic subdural haematoma)**; **cognitive impairment, alcohol-related brain damage and Wernicke–Korsakoff syndrome**; **peripheral neuropathy and cerebellar degeneration**; **liver disease, pancreatitis, gastritis and GI bleeding**; **hypertension, cardiomyopathy and atrial fibrillation ("holiday heart")**; **malnutrition and thiamine, folate and B12 deficiency**; **osteoporosis**; **cancer**; **depression and increased suicide risk**; and **drug interactions** with benzodiazepines, opioids, warfarin, paracetamol, metformin and antihypertensives. **A prior history of alcoholism predisposes to later benzodiazepine misuse.**
5. **Screening and assessment. Ask every older patient** — quantity, frequency, pattern, and the timeline of onset. Tools: the **CAGE questionnaire** and the **Short Michigan Alcohol Screening Test — Geriatric version (SMAST-G)**, which is specifically validated for older adults, plus AUDIT-C. Corroborate with an **informant**. Investigations: **CBC with MCV, liver function including GGT, blood sugar (mg/dL), electrolytes (mEq/L), magnesium, albumin, INR, vitamin B12 and folate**, and **CT head where there has been a fall or confusion**. **Screening for alcohol misuse, depression and delirium is recommended for all elderly people attending the emergency department.**
6. **Management. Detoxification** where dependence exists — **ideally as an inpatient in frail older adults**, using **CIWA-Ar-guided short-acting benzodiazepines such as lorazepam** (safer where liver function is impaired), with **parenteral thiamine before any glucose load** to prevent Wernicke's encephalopathy; **withdrawal is more likely to be complicated by confusion and delirium tremens than in younger adults**. Then **rehabilitation delivered concurrently, not consecutively, across physical, psychological and social domains**. **Age-specific, non-confrontational programmes that promote self-esteem, tolerance and peer relationships achieve roughly twice the abstinence of confrontational programmes**, and improve retention. Add **brief interventions and motivational interviewing**, treat **comorbid depression, pain and insomnia**, rebuild the social network, and consider **relapse-prevention pharmacotherapy — naltrexone or acamprosate**, with **disulfiram generally avoided in older adults**. Outcomes in older patients are **at least as good as in younger patients**.

Latest Update — 1 Point

7. **No safe level of alcohol for cancer risk (2023–2025).** The **WHO statement in *The Lancet Public Health* (2023)** concluded that **no level of alcohol consumption is safe for health**, since alcohol is a **Group 1 carcinogen**, and the **US Surgeon General's Advisory of January 2025** identified alcohol as the **third leading preventable cause of cancer** and called for cancer warning labels. This has removed the older "cardioprotective" framing based on J-shaped curves now attributed to **abstainer bias**. For older adults this reinforces **lower recommended limits, routine screening at every contact, and brief intervention**, and aligns with the **2023 AGS Beers Criteria** caution against combining alcohol with benzodiazepines, opioids or gabapentinoids.

Question 24. Benzodiazepine dependence and withdrawal in the older adult [7 marks]

Textbook Core — 6 Points

1. **Scale of the problem.** Benzodiazepines are among the most prescribed drugs in old age — about **10% of community-dwelling over-65s**, and up to **25% of nursing-home residents**. Roughly **70% of users continue two to four years later**, and **women are twice as likely as men** to be taking one.

2. **Risk factors for dependence. Female sex**, increasing age, **duration of use beyond 4 weeks**, **short-acting high-potency agents (alprazolam, lorazepam)**, insomnia, **comorbid anxiety and depressive disorders**, bereavement, poor physical health, institutionalisation, and **multiple prescribers**.
3. **Harms in the elderly. Falls and hip fracture**, **cognitive impairment and delirium**, daytime sedation, road traffic accidents, **respiratory depression (especially with opioids)**, **paradoxical excitement**, and dependence. Listed as **potentially inappropriate for all older adults in the AGS Beers Criteria**.
4. **Detection.** Watch for **drug-seeking behaviour** — early repeat prescription requests, requests for dose escalation, and obtaining medication from multiple sources. The **Severity of Dependence Scale** (5 items, each scored 0–3) is validated in older adults — a **total score ≥6 indicates problematic use** (sensitivity 97.9%, specificity 94.2%).
5. **Withdrawal syndrome. Rebound insomnia and anxiety**, tremor, sweating, palpitations, **perceptual hypersensitivity**, depersonalisation, and in severe cases **confusion and delirium, psychosis, and seizures**. **Withdrawal in the elderly is far more likely to be complicated by confusion** than in younger adults.
6. **Management. Convert to a long-acting benzodiazepine (diazepam) and taper gradually over months. Avoid rapid detoxification** — it causes breakthrough symptoms and may precipitate convulsions; if unavoidable, conduct it **as an inpatient**. Add **sleep hygiene education, relaxation training and CBT-I**; treat comorbid depression and anxiety. A simple GP letter urging reduction cut total use by **one-third at six months**. **Never stop abruptly.**

Latest Update — 1 Point

7. **ASAM Joint Clinical Practice Guideline on Benzodiazepine Tapering (2025, *J Gen Intern Med*).** Recommends that clinicians **taper benzodiazepines in most older adults unless there is a compelling reason to continue**; use **gradual, symptom-based (hyperbolic) reductions of about 5–10% every 2–4 weeks** with shared decision-making; **forced or abrupt discontinuation is not recommended**; and it formally recognises **Benzodiazepine-Induced Neurological Dysfunction (BIND)** as a protracted withdrawal state.

Current Practice Update

Aspect	Textbook	Current recommendation	Exam approach
Taper rate	"Gradual reduction over months", rate unspecified	**5–10% dose reduction every 2–4 weeks**, symptom-guided, no fixed end-date	Quote the numeric taper rate and credit the ASAM 2025 guideline
Consent	Physician-driven detoxification	**Shared decision-making; no forced taper**; monitor if the patient declines	Add one line on shared decision-making — it is a marked point now

```
TAPER ALGORITHM
Long-term BZD user (>4 weeks) in an older adult
        |
   Assess: SDS score, indication, falls/cognition, opioid co-prescription
        |
   Shared decision-making + psychoeducation  ---> declines ---> monitor, re-offer later
        |
   Convert to equivalent DIAZEPAM dose (long-acting)
        |
   Reduce by 5-10% every 2-4 weeks, symptom-guided
        |
   Add CBT-I / sleep hygiene / relaxation; treat depression & anxiety
        |
   Severe dependence, seizure risk or failure ---> INPATIENT taper
```

If asked for 15 marks, add: *pharmacology of benzodiazepines and altered pharmacokinetics in ageing (increased volume of distribution, prolonged half-life, active metabolites); a diazepam-equivalence table; Z-drugs as an equivalent problem; benzodiazepines and dementia risk evidence; STOPP/START and Beers criteria wording; deprescribing tools (EMPOWER trial, D-PRESCRIBE); and the psychology of deprescribing.*

Question 25. Nicotine dependence and smoking cessation [7 marks]

Textbook Core — 6 Points

1. **It is never too late to quit.** Epidemiological data show that **even among people in their 70s, stopping smoking reduces mortality towards that of non-smokers within a short enough time to justify actively encouraging quitting**. **Benefits for vascular and lung disease are rapid.**
2. **Additional geriatric gains.** Slower decline in **FEV1**, better wound and fracture healing, reduced risk of **stroke, myocardial infarction, cancer, cataract, macular degeneration, osteoporosis and dementia**, and reduced **fire and burn risk** in frail or oxygen-dependent patients.
3. **Assessment of dependence.** Pack-years, the **Fagerström Test for Nicotine Dependence**, and above all the **time to the first cigarette of the day (<30 minutes indicates high dependence)**; exhaled CO; previous quit attempts and reasons for relapse; readiness to change. Screen for **depression, alcohol use and COPD**.

4. **Behavioural framework.** The **5 A's — Ask, Advise, Assess, Assist, Arrange**, with the **5 R's** (Relevance, Risks, Rewards, Roadblocks, Repetition) for the unmotivated. **Brief physician advice alone raises quit rates**; combine with counselling and quitlines. In India — the **National Tobacco Control Programme (NTCP)**, **Tobacco Cessation Centres**, **COTPA 2003**, and the **national quitline 1800-11-2356**.
5. **Pharmacotherapy. Nicotine replacement therapy** — combination of a long-acting patch with a short-acting gum/lozenge is most effective. **Varenicline** — nicotinic partial agonist, the **most effective single agent**; dose-reduce if eGFR <30; causes nausea and vivid dreams. **Bupropion SR** — **lowers the seizure threshold**, contraindicated in seizure and eating disorders, and interacts through **CYP2D6**.
6. **Geriatric precautions.** Nicotine patches need caution in **unstable angina, recent MI and arrhythmia**. Critically, **stopping smoking removes CYP1A2 induction** — plasma levels of **clozapine, olanzapine, theophylline and the effect of warfarin rise**, so doses must be reviewed and reduced. Combine cessation with **pulmonary and cardiac rehabilitation**.

Latest Update — 1 Point

7. **Cytisinicline (cytisine) — the first new cessation drug in two decades.** A plant-derived **nicotinic acetylcholine receptor partial agonist**. The Phase 3 **ORCA-2 and ORCA-3 trials (JAMA 2023; JAMA Internal Medicine 2024)** showed significantly higher biochemically confirmed abstinence versus placebo, sustained to 24 weeks, with **fewer gastrointestinal adverse effects than varenicline**. The **NICE 2025 evidence review** and **ICER 2025** report rate its efficacy as comparable to varenicline; it is already inexpensive and available in several countries, although US approval was delayed in 2026 by a manufacturing-related Complete Response Letter.

Source note: *the uploaded textbooks cover the rationale and epidemiology of cessation in old age but do not detail pharmacotherapy protocols; points 3–6 follow standard tobacco-dependence guidance (NICE NG209 / USPHS 5A framework) integrated with geriatric prescribing principles.*

If asked for 15 marks, add: *neurobiology of nicotine dependence (α4β2 receptor upregulation); a full timeline of health benefits after quitting (20 min → 15 years); detailed NRT dosing by cigarettes/day; e-cigarettes and the current evidence and controversy; smokeless tobacco (khaini, gutkha, zarda) and its dominance in older Indians; COTPA 2003 and the 2019 e-cigarette ban; and relapse prevention.*

Question 26. Substance misuse other than alcohol in older adults [7 marks]

Pivot: prescribed, over-the-counter and illicit substances other than alcohol. Alcohol is Question 23 and benzodiazepines Question 24.

Textbook Core — 6 Points

1. **Three broad categories (textbook classification). (a) Medications** — prescribed (**benzodiazepines, opioids, Z-drugs, anticholinergics**) and non-prescribed / over-the-counter (codeine, antihistamines, laxatives, decongestants); **(b) socially sanctioned psychoactive substances** — tobacco, caffeine; **(c) illicit substances** — cannabis, opioids, cocaine.
2. **Changing epidemiology.** There is a **growing trend of substance misuse in the elderly**, driven by a **generation of lifetime drug users now entering old age**. **Prescribed benzodiazepine and opioid misuse remains far commoner than illicit drug use.** In India, traditional patterns include **opium and doda-post** use in Rajasthan and Punjab, and cannabis (**bhang, ganja**) in older men.
3. **Why it is missed.** Older people **rarely present complaining of substance misuse** — they present with associated physical problems. Symptoms are wrongly attributed to ageing or dementia (**falls, confusion, incontinence, self-neglect, weight loss**). Add **stigma**, screening tools validated in the young, and the absence of work or legal markers in a retired person.
4. **Age-specific vulnerability. Reduced hepatic and renal clearance**, **reduced lean body mass and total body water**, **increased CNS sensitivity**, and **polypharmacy with drug interactions** — so **much smaller quantities produce toxicity and withdrawal** than in younger adults.
5. **Assessment. Sensitive but persistent enquiry** plus an **informant history**, supported by **standardised screening tools**. Review every prescribed and over-the-counter drug, look for **multiple prescribers and early repeat requests**, and **actively treat comorbid depression, pain and insomnia**, which perpetuate the disorder. Examine social factors — housing and a social network of substance users.
6. **Management.** A **multistage, integrated physical + psychological + social package delivered concurrently, not consecutively**, in a form acceptable to older people. **Detoxification** where physical dependence exists. **Age-specific, non-confrontational, supportive group programmes retain older patients better** than mixed-age confrontational programmes — one study found **confrontational programmes achieved half the abstinence** of those promoting self-esteem, tolerance and peer relationships. Follow with rehabilitation, aftercare and relapse prevention.

Latest Update — 1 Point

7. **Cannabis is the fastest-growing substance of misuse in adults aged ≥65 (2023–2025).** Recent national survey data show a sharp rise in past-year use, and current reviews link it in older adults to **falls, orthostatic hypotension, tachyarrhythmia, cognitive impairment and cannabis-induced psychosis**, compounded by drug interactions and unregulated cannabidiol products. In parallel, the **2023 AGS Beers Criteria** strengthened the caution against combining **opioids with benzodiazepines or gabapentinoids** in older adults.

If asked for 15 marks, add: *screening instruments (ASSIST, DAST, Severity of Dependence Scale); detailed opioid misuse and opioid use disorder management with buprenorphine/methadone in the elderly; withdrawal syndromes by substance; Indian data from the NDDTC-AIIMS "Magnitude of Substance Use in India" report and the Nasha Mukt Bharat Abhiyaan; the interface with elder abuse and financial exploitation; and prevention through rational prescribing and deprescribing.*

Section G — Treatments and Therapeutics

The three therapeutic modalities asked as stand-alone notes.

Question 27. Atypical antipsychotics — their uses and side effects in the elderly [7 marks]

Textbook Core — 6 Points

1. **Definition and mechanism.** Second-generation ("atypical") antipsychotics act by **combined dopamine D2 and serotonin 5-HT2A antagonism**, with **looser, more transient D2 binding** than conventional agents. This gives them a **lower risk of extrapyramidal symptoms and tardive dyskinesia**, at the cost of a **higher metabolic burden**. **Atypical antipsychotics are preferred over conventional antipsychotics in older adults.**
2. **Evidence-based indications. Schizophrenia and late-onset schizophrenia / VLOSLP** (amisulpride 100 mg has RCT support); **delusional disorder and late-life psychosis**; **acute mania in bipolar disorder** (quetiapine, aripiprazole, risperidone at one-third to one-half adult dose); **augmentation of antidepressants in treatment-resistant depression**; **psychotic depression** combined with an antidepressant; **Parkinson's disease psychosis** (quetiapine, clozapine, or pimavanserin); and **severe, distressing or dangerous agitation and psychosis in dementia after non-drug measures have failed**.
3. **Choosing the agent. Quetiapine** — the drug of choice where **parkinsonism or DLB** is present; sedating, causes orthostatic hypotension. **Risperidone 0.25–1 mg** — most evidence in dementia-related psychosis, but dose-dependent EPS and hyperprolactinaemia. **Olanzapine 2.5–5 mg** — sedating, most metabolic burden, strongly anticholinergic. **Aripiprazole 2–5 mg** — partial D2 agonist, least metabolic effect, may cause akathisia. **Clozapine** — reserved for treatment-resistant schizophrenia and Parkinson's disease psychosis; requires **mandatory neutrophil monitoring**.
4. **Adverse effects — the geriatric list. Metabolic** — weight gain, hyperglycaemia, dyslipidaemia (worst with olanzapine and clozapine). **Cardiovascular** — **orthostatic hypotension and falls**, **QTc prolongation and torsades**. **Neurological** — EPS, **tardive dyskinesia (risk rises with age)**, **neuroleptic malignant syndrome**, sedation, **worsening of cognition**. **Anticholinergic** — dry mouth, constipation, urinary retention, delirium (olanzapine, clozapine). **Endocrine** — hyperprolactinaemia (risperidone). **Others** — aspiration pneumonia, venous thromboembolism, **agranulocytosis with clozapine**, and **severe neuroleptic sensitivity in DLB** (sedation, worsening parkinsonism, autonomic instability — may be irreversible).
5. **The boxed warning — must be written. All antipsychotics, typical and atypical, carry a boxed warning of increased mortality and cerebrovascular events when used for behavioural symptoms of dementia** (roughly a 1.6–1.7 fold increase in death, mainly cardiac and infective). **No medication is FDA-approved for treating the neuropsychiatric symptoms of dementia.** Use therefore requires **documented failure of non-pharmacological measures, informed consent from the patient or nominated representative, the lowest effective dose, and a defined review and withdrawal date**.
6. **Safe prescribing rules. Start at one-quarter to one-third of the adult dose and titrate slowly.** Baseline and periodic monitoring of **weight and waist circumference, fasting glucose (mg/dL), lipid profile, ECG for QTc, blood pressure lying and standing, prolactin if symptomatic, and an EPS/tardive dyskinesia examination (AIMS)**. **Review for deprescribing every 3 months** — most patients with dementia can be withdrawn without symptom recurrence. Avoid combining with other QT-prolonging or anticholinergic drugs. Note that **antipsychotics such as quetiapine should generally not be used for anxiety disorders, as the risks outweigh the benefits**. The **AGS Beers Criteria list antipsychotics as potentially inappropriate** unless the indication is schizophrenia, bipolar disorder or short-term antiemesis.

Latest Update — 1 Point

7. **Brexpiprazole (FDA-approved May 2023) — the first drug ever approved for agitation associated with dementia due to Alzheimer's disease**, at 2–3 mg daily. It remains subject to the **class boxed warning on mortality in dementia-related psychosis**, effect sizes are modest, and it must still follow rather than replace non-pharmacological management. Alongside this, **xanomeline–trospium (KarXT, 2024)** introduces a **non-dopaminergic muscarinic mechanism** with no EPS, prolactin rise or weight gain — potentially a major advance for older adults, with dementia-related psychosis trials ongoing.

Quick-Reference Comparison

Drug	Geriatric dose	Best used for	Watch for
Quetiapine	12.5–50 mg	Parkinsonism, DLB, Parkinson's disease psychosis	Sedation, orthostatic hypotension, falls
Risperidone	0.25–1 mg	Dementia-related psychosis (most evidence), mania	Dose-dependent EPS, hyperprolactinaemia, stroke risk
Olanzapine	2.5–5 mg	Mania, psychosis where sedation is wanted	Weight gain, hyperglycaemia, anticholinergic delirium
Aripiprazole	2–5 mg	Antidepressant augmentation, mania, OABD	Akathisia, insomnia

Amisulpride	50–100 mg	VLOSLP (ATLAS trial evidence)	EPS, hyperprolactinaemia, renal accumulation
Clozapine	6.25–25 mg	Treatment-resistant schizophrenia, PD psychosis	**Agranulocytosis**, myocarditis, seizures, sialorrhoea

Question 28. Electroconvulsive therapy in older adults [7 marks]

Pivot: ECT as a complete stand-alone answer — technique, consent, adverse effects and continuation treatment. Its role specifically within depression is also covered in Question 2.

Textbook Core — 6 Points

1. **Definition and utilisation.** ECT is the delivery of a **brief electric current to the brain under general anaesthesia and muscle relaxation to produce a generalised cerebral seizure**, given in a course of typically **6–12 treatments, two to three times per week**. **Older adults constitute more than one-half of all patients who receive ECT for depression**, yet it remains **under-utilised despite its relative safety and high effectiveness**.
2. **Indications. Severe depression with somatic complications, severe suicidality, catatonia, and refusal of food and fluids — here immediate initiation of ECT is indicated. Psychotic depression. Pharmacotherapy-resistant depression.** Also **refractory mania**, **severe and treatment-resistant persistent agitation and aggression in dementia**, and neuroleptic malignant syndrome. **ECT should be considered a first-line option in all patients with severe depressive symptoms, especially where psychotic symptoms are present** — it is not a last resort.
3. **Efficacy and the age effect. ECT is probably more effective in more severe depression in older people than in younger adults**, and remission rates in psychotic depression exceed 70–80%. **The efficacy of ECT does not diminish with advancing age, but the seizure threshold increases**, so higher stimulus doses are needed. **Older age is a predictor of better response to ECT. ECT rapidly relieves expressed suicidal intent.**
4. **Pre-ECT assessment.** Confirm the indication and document **informed consent** — from the patient if capacity is present, otherwise from the nominated representative under the **Mental Healthcare Act 2017**, which **prohibits ECT without anaesthesia and muscle relaxants** and bars its use in minors without Mental Health Review Board permission. Assess **cardiovascular and respiratory fitness, cervical spine and dentition, and cognition (baseline MMSE/MoCA)**; obtain **CBC, electrolytes (mEq/L), glucose (mg/dL), renal and liver function, ECG, and chest X-ray**; review drugs — **stop or reduce benzodiazepines and anticonvulsants** (they raise the seizure threshold), continue antihypertensives, and withhold lithium.
5. **Adverse effects. ECT has remarkably few side effects in older age, with reversible cognitive side effects the most common feature** — **post-ictal confusion, anterograde amnesia and retrograde amnesia**, usually resolving within weeks, though patchy autobiographical memory gaps may persist. Others: **transient hypertension, tachycardia and arrhythmia**, headache, myalgia, nausea, prolonged seizure, and anaesthetic risk. **There is no absolute age limit and no absolute contraindication**; raised intracranial pressure, recent myocardial infarction, recent stroke, unstable aneurysm and severe cardiac disease are **relative** contraindications requiring specialist assessment.
6. **Technique, continuation and alternatives. Right unilateral electrode placement** reduces cognitive effects compared with bitemporal, at the cost of needing a higher stimulus dose; **brief and ultrabrief pulse widths** further limit memory impairment. **Relapse is high after a successful course**, so give **continuation pharmacotherapy (antidepressant with or without lithium) or continuation/maintenance ECT**. **Transcranial magnetic stimulation** is a non-invasive alternative for depression resistant to at least one antidepressant, approved also for OCD, and carries **lower risks than antidepressants**, but should be used judiciously where there is seizure risk after stroke or traumatic brain injury.

Latest Update — 1 Point

7. **ELEKT-D trial (*NEJM* 2023).** In **treatment-resistant major depression without psychosis**, **subanaesthetic intravenous ketamine was non-inferior to ECT** with fewer cognitive adverse effects — providing an option where ECT is refused, unavailable or contraindicated. **ECT nevertheless remains superior and first-line in psychotic depression, catatonia and acute suicidality**, all of which were excluded from that trial. Current practice increasingly favours **right unilateral ultrabrief-pulse ECT**, which achieves **comparable efficacy with substantially less cognitive impairment** in older adults.

Exam one-liners: *ECT = brief current under anaesthesia + muscle relaxant → generalised seizure · >50% of ECT recipients for depression are older adults · efficacy undiminished by age but seizure threshold rises · older age predicts better response · commonest adverse effect is reversible cognitive impairment · no absolute contraindication · MHCA 2017 prohibits unmodified ECT · relapse is high — always give continuation treatment.*

Question 29. Cognitive behavioural therapy [7 marks]

Textbook Core — 6 Points

1. **Definition and model.** CBT is an **active, structured, time-limited, goal-directed psychotherapy that aims to change the thinking and behaviour that maintain a disorder**. It rests on the **cognitive model** — a situation triggers an **automatic thought**, which drives **emotion, physiological arousal and behaviour**; these are underpinned by **intermediate beliefs (rules and assumptions)** and **core beliefs (schemas)**. Changing the cognition changes the emotion and the behaviour.
2. **Structure of treatment.** Typically **8–20 weekly sessions of 45–60 minutes**, individual or group, delivered with a **collaborative, agenda-setting style** and **homework between sessions**. Core techniques: **psychoeducation**, **thought records and identification of cognitive distortions** (catastrophising, all-or-nothing thinking, overgeneralisation, mind-reading), **Socratic questioning and cognitive restructuring**, **behavioural activation and activity scheduling**, **graded exposure with response prevention**, **problem-solving**, **relaxation training**, and **relapse prevention**.
3. **Evidence base and indications in older adults. CBT for depression carries Level A evidence and is effective when compared with no treatment. Psychotherapy is as effective in older adults as in younger adults**, and **many older adults prefer counselling to psychopharmacological intervention**. Indications: **depression** (especially mild to moderate), **anxiety disorders and GAD**, **phobias including fear of falling**, **panic disorder**, **OCD (as exposure and response prevention)**, **PTSD**, **insomnia (CBT-I, the first-line treatment for chronic insomnia at any age)**, **chronic non-cancer pain**, **somatic symptom disorder**, **caregiver stress**, and as psychological support during **rational deprescribing**.
4. **Problem-solving therapy — the key geriatric variant. Individual CBT, particularly problem-solving therapy, is the recommended psychological intervention for late-life depression**, and it is **especially effective where executive dysfunction is present** — the group least likely to respond to antidepressants. Its seven steps: define the problem → set a realistic goal → generate solutions → evaluate the options → choose and plan → implement → review the outcome.
5. **Adaptations required in older adults. Psychotherapy may need to be modified to accommodate cognitive and sensory deficits.** Practical adjustments: **slower pace and shorter, more frequent sessions**; **written summaries, large print and audio recordings**; **hearing aids and spectacles in place, quiet well-lit room**; **repetition and memory aids for homework**; **involvement of family or carer**; **home visits or telephone/video delivery** where mobility is limited. Content must be **dynamic and sensitive to the existential issues of loss, dependency and change of status, yet fully aware of opportunities for growth and vigour**, with the **focus best directed at functional outcomes**.
6. **Limitations and related therapies.** CBT requires **some capacity for abstraction, insight and memory**, so it is of limited use in **moderate to severe dementia, delirium, and active psychosis**, and needs motivation and regular attendance. Availability of trained therapists is a real constraint in India. Related and complementary approaches include **interpersonal therapy, reminiscence and life review, behavioural activation, mindfulness-based interventions, dialectical behaviour therapy (personality disorders), and family therapy**. **Psychoeducation should be a routine part of treatment and, whenever feasible, should include family members** — this prevents the unrealistic expectation of rapid, complete recovery that leads to premature termination.

Latest Update — 1 Point

7. **Digital and remotely delivered CBT (2023–2025).** Systematic reviews and randomised trials now show that **internet-delivered and telephone/video CBT are effective in older adults for depression, anxiety and insomnia**, with effect sizes approaching face-to-face therapy when therapist support is included — directly addressing the shortage of trained therapists and the mobility and transport barriers that limit uptake in this age group. Alongside this, **mindfulness-based stress reduction was shown to be non-inferior to escitalopram for anxiety disorders (Hoge et al., *JAMA Psychiatry* 2023)**, and **CBT-I remains the guideline-endorsed first-line treatment for chronic insomnia in older adults**, in preference to any hypnotic.

Section H — Sexuality in Later Life

Question 30. Sexual dysfunction and its psychiatric associations in the elderly [7 marks]

Textbook Core — 6 Points

1. **Normal ageing changes. Men** — slower erection, reduced rigidity, **longer refractory period**, reduced ejaculatory volume. **Women** — vaginal dryness, atrophy, dyspareunia, reduced elasticity (**genitourinary syndrome of menopause, GSM**). **Sexual desire persists into late life** — ageing alone does not abolish sexual interest.
2. **Classification. Desire** disorders (hypoactive sexual desire), **arousal** disorders (**erectile dysfunction**, female arousal disorder), **orgasmic** disorders, and **sexual pain** disorders (dyspareunia, vaginismus).
3. **Organic causes. Diabetes**, cardiovascular and peripheral vascular disease, hypertension, **prostate surgery and pelvic radiotherapy**, neurological disease (**Parkinson's disease**, stroke, neuropathy), **hypogonadism** (testosterone in ng/dL), hyperprolactinaemia, CKD, arthritis, and obesity. **Erectile dysfunction is an early marker of endothelial dysfunction and predicts future cardiovascular events.**
4. **Drug causes. SSRIs and SNRIs** (commonest — delayed orgasm, reduced desire), **antipsychotics** (hyperprolactinaemia), **beta-blockers, thiazides, spironolactone, finasteride**, anticholinergics, opioids, and alcohol.
5. **Psychiatric associations. Depression** — a **bidirectional relationship** (both a cause and a consequence, and its drug treatment worsens it further); **anxiety and performance anxiety**; **dementia** — disinhibition, **inappropriate sexual behaviour**, and difficult **capacity-to-consent** questions in care homes; alcohol misuse; and **bereavement and widowhood — lack of a partner is the commonest reason for sexual inactivity in older women**.
6. **Management.** Use the **PLISSIT model** (Permission → Limited Information → Specific Suggestions → Intensive Therapy). Optimise chronic disease; **review and switch the offending drug** (e.g. SSRI → bupropion or mirtazapine). **PDE-5 inhibitors (sildenafil, tadalafil) are first-line for ED** — **absolutely contraindicated with nitrates**, caution with alpha-blockers. **Low-dose vaginal oestrogen ± lubricants** for GSM. Couple and psychosexual counselling. **Testosterone only in biochemically confirmed hypogonadism.**

Latest Update — 1 Point

7. **2023–2025 guidance.** AUA/EAU reaffirm **PDE-5 inhibitors as first-line for ED at any age**, with cardiovascular risk stratification before resuming sexual activity (**Princeton consensus** low / intermediate / high-risk categories). Menopause society guidance (**NAMS / IMS 2023–24**) endorses **low-dose vaginal oestrogen, prasterone (intravaginal DHEA) and ospemifene** as safe and effective for GSM — including in most breast cancer survivors after discussion with the oncologist — as **systemic absorption is negligible**.

If asked for 15 marks, add: *physiology of the sexual response cycle and its age-related changes; detailed history-taking and examination framework; investigations (fasting glucose, lipids, testosterone ng/dL, prolactin, TSH, PSA ng/mL); management of inappropriate sexual behaviour in dementia (behavioural first, then SSRI, antiandrogen as last resort); capacity and consent in institutional settings; sexuality in LGBT+ elders; and Indian cultural attitudes and the near-absence of sexual history-taking in geriatric practice.*

Section I — Law, Ethics and Capacity

The two statutory and ethical questions that recur in every paper.

Question 31. Mental Healthcare Act 2017 — provisions relevant to geriatric psychiatry [15 marks]

Textbook Core — 10 Points

1. **Background and scope. Act No. 10 of 2017**, assented on **7 April 2017** and in force from **29 May 2018**. It **repeals the Mental Health Act 1987** and was enacted to fulfil India's obligations under the **UN Convention on the Rights of Persons with Disabilities (UNCRPD)**. The shift is from a **custodial to a rights-based model** — mental healthcare is now a **legal right, not a discretionary service**.
2. **Definition of mental illness — Section 2(s).** A **substantial disorder of thinking, mood, perception, orientation or memory** that **grossly impairs judgement, behaviour, capacity to recognise reality, or the ability to meet the ordinary demands of life**; it **includes conditions associated with alcohol and drug abuse** but **excludes mental retardation**. **Dementia and delirium therefore fall within the Act.**
3. **Presumption of capacity — Section 4. Every person is presumed to have the capacity** to make mental healthcare decisions unless proved otherwise. Capacity means the ability to **understand the information, appreciate the reasonably foreseeable consequences, and communicate the decision**. **A diagnosis of dementia alone does not remove capacity**, which is decision-specific.
4. **Advance Directive — Sections 5 to 13.** Any adult with capacity may make a **written advance directive** stating **how they wish to be, and not to be, cared for and treated**, and may register it with the **Mental Health Review Board (MHRB)**. It may be revoked or amended at any time and can be challenged before the MHRB. **Of particular value in early dementia and recurrent bipolar disorder.**
5. **Nominated Representative — Sections 14 to 17.** Every person has the right to **appoint a Nominated Representative (NR)** in writing. If none is appointed, a statutory order of default applies (relative, caregiver, or a person appointed by the MHRB). The NR **receives information, supports the person, and gives consent where capacity is lacking** — a key provision in advanced dementia.
6. **Right to access mental healthcare — Section 18.** A right to **affordable, good-quality, geographically accessible** services run or funded by government — including **outpatient and inpatient care, half-way homes, sheltered accommodation and community-based rehabilitation**, with explicit mention of provision for **older persons**.
7. **Community living and protection from abandonment — Sections 19 to 20.** The right to **live in and be part of the community and not to be segregated**. Crucially, **no person may be kept in a mental health establishment merely because the family is absent or unwilling** — the government must provide **supported accommodation**. This is directly relevant to **abandoned elders**, alongside the **MWPSC Act 2007**.
8. **Other rights — Sections 20 to 28.** Protection from **cruel, inhuman and degrading treatment**; equality and non-discrimination; right to information, **confidentiality**, legal aid, and to make complaints. **Chaining is prohibited**; **seclusion and solitary confinement are prohibited**; **physical restraint only as a last resort** with documentation and NR intimation.
9. **Admission, treatment and ECT. Independent admission (Sec 86)** for those with capacity who consent. **Supported admission (Sec 89 — up to 30 days; Sec 90 — beyond 30 days)** for those lacking capacity who pose a risk of harm to self or others, or are unable to care for themselves — requires **two independent mental health professionals** and MHRB oversight. **ECT is prohibited without muscle relaxants and anaesthesia**, and is **banned for minors without MHRB permission**. **Psychosurgery requires informed consent plus MHRB approval.**
10. **Insurance parity and decriminalisation of suicide. Section 21(4)** — every insurer must provide medical insurance for mental illness **on the same basis as for physical illness**. **Section 115** — a person who attempts suicide is **presumed to be under severe stress, shall not be prosecuted**, and the government must provide care and rehabilitation. This matters greatly given the **high suicide rate in older Indians**. Institutional framework: **Central and State Mental Health Authorities and Mental Health Review Boards**.

Latest Updates — 2 Points

11. **Ayushman Bharat Vay Vandana (from late 2024).** All citizens **aged 70 years and above** are now entitled to **AB-PMJAY cover of ₹5 lakh per family per year irrespective of income**, with **mental health packages included**. This operationalises the Section 18 right of access and the Section 21(4) parity requirement specifically for older adults, and is the single most quotable Indian development in this area.
12. **Implementation gap and delivery route (2024–25 reviews).** Studies in *Indian Journal of Psychological Medicine* and *Lancet Psychiatry* document that **Section 21(4) insurance parity remains poorly implemented**; the current enforcement instrument is the **IRDAI directive requiring all policies to cover mental illness without exclusion**. Service delivery now runs mainly through the **District Mental Health Programme under the NMHP**, **Tele-MANAS (14416)**, and **Ayushman Arogya Mandirs** for community-level screening.

Exam one-liners: *Act No. 10 of 2017 · assented 7 April 2017 · effective 29 May 2018 · repeals MHA 1987 · Sec 2(s) definition · Sec 4 capacity*

· Sec 5 advance directive · Sec 14 nominated representative · Sec 18 right to access · Sec 21(4) insurance parity · Sec 86 independent admission · Sec 89/90 supported admission · Sec 95 prohibited procedures (ECT without anaesthesia, chaining) · Sec 115 decriminalisation of suicide.

Question 32. Capacity assessment and consent in the cognitively impaired patient [15 marks]

Textbook Core — 10 Points

1. **Capacity versus competence. Capacity** is a **clinical judgement** about a person's ability to make a **specific decision at a specific time**. **Competence is a legal term** implying that a person has been declared competent or incompetent **by a court of law**. **Clinicians assess capacity; courts declare competence.**
2. **Ethical basis.** The four principles — **autonomy, beneficence, non-maleficence and justice**, with **fidelity**. **Valid consent requires three things: it must be informed, voluntary, and given by a person with capacity.** Autonomy does not mean "I want, therefore I get" — but it does mean "you may not do things to me without my permission".
3. **The four elements of decisional capacity. (i) Understanding** the disclosed information; **(ii) Appreciation** of its significance for one's own situation; **(iii) Reasoning** — the ability to weigh risks and benefits; **(iv) Communicating a Choice** clearly and consistently.
4. **Core principles. Capacity is presumed until proved otherwise.** It is **decision-specific and time-specific**. **An unwise decision is not evidence of incapacity** — if a patient with capacity refuses treatment, that refusal stands and the duty of care continues without prejudice. **A diagnosis of dementia does not by itself establish incapacity**; deficits are patchy, and a person may decide well with appropriate prompting.
5. **Sliding scale of capacity.** The **standard required rises with the risk of the decision**. A patient may have capacity to consent to a blood test but not to major surgery or a Phase 1 trial. **Match the complexity of the decision to the patient's cognitive capacity** — "stage the ageing as carefully as you stage the disease".
6. **How to assess.** Give information in **simple language, in the patient's own language**, in a **quiet setting**, with **hearing aids and spectacles in place**, at the **patient's best time of day**, with written material in **large type and good contrast**. Ask the patient to **paraphrase the information back**, and **re-check consistency after an interval**.
7. **Cognitive tests and their limits. No cognitive screening test determines decision-making capacity. MMSE and MoCA correlate poorly**; tests of **executive function — the Clock Drawing Test and the Executive Interview — correlate better**. Structured instruments: **MacArthur Competence Assessment Tool for Treatment (MacCAT-T)** is the reference standard, and the **Aid to Capacity Evaluation (ACE)** is the practical bedside tool recommended for hospitalised older adults.
8. **Optimise capacity before declaring it absent.** Treat reversible contributors first — **delirium, infection, pain, hypoxia, hypoglycaemia, constipation, urinary retention, depression, sedatives and anticholinergic drugs, and sensory deprivation**. **Incapacity is common in hospitalised older adults and is under-recognised by physicians.**
9. **When capacity is lacking — the hierarchy.** Follow, in order: **(1) a valid advance directive → (2) the appointed surrogate / nominated representative / power of attorney → (3) a best-interests decision by the lead clinician**, or an application for guardianship where the law requires. **Best interests must weigh the person's past and present wishes, beliefs and values**, not only medical benefit. **Personhood persists even when capacity does not.**
10. **Documentation and common pitfalls.** Record the **specific decision, the information given, the patient's responses under each of the four elements, the conclusion, and who was involved**. Pitfalls to avoid — equating a low MMSE with incapacity; assessing "global" instead of decision-specific capacity; accepting the family's view over the patient's; **infantilising the patient by directing questions to relatives**; and **failing to reassess once delirium has resolved**.

Latest Updates — 2 Points

11. **Indian statutory framework.** The **Mental Healthcare Act 2017, Section 4** creates a **statutory presumption of capacity** with a three-part test (**understand → appreciate consequences → communicate**), and provides for **Advance Directives (Sec 5)** and **Nominated Representatives (Sec 14)**. The **Rights of Persons with Disabilities Act 2016** similarly favours **supported over substituted decision-making**, replacing the older guardianship-based approach.
12. **International direction (2023–2025).** Guidance continues to shift from **substituted to supported decision-making** under **UNCRPD Article 12**. The **2024 Lancet Commission on dementia prevention, intervention and care** reinforces **early advance care planning while capacity is retained**, and current practice recommends **structured instruments (MacCAT-T / ACE) rather than unaided clinical impression** for high-stakes decisions.

```
CAPACITY ASSESSMENT ALGORITHM
Specific decision required
        |
Presume capacity  ->  Optimise: treat delirium/pain/depression, hearing aid, glasses,
        |                          quiet room, simple language, best time of day
Assess the FOUR elements:  Understand -> Appreciate -> Reason -> Communicate a Choice
        |
   ALL PRESENT?
      |        \
     YES        NO
      |          \
  Patient decides  Advance Directive?  --yes--> follow it
  (even if unwise)        | no
                  Nominated Representative / surrogate / POA?  --yes--> they decide
                         | no
                  BEST-INTERESTS decision by lead clinician (+ guardianship if required)
                         |
                  DOCUMENT everything; REASSESS when the reversible cause clears
```

Appendix. Consolidated Night-Before Master Sheet [all 32 questions]

One line of core content and one latest update for every topic in this compilation, merged from all three source sets.

Topic	Must-write core	Must-write latest update
Psychogeriatrics		
Delusional disorder	Non-bizarre delusion ≥1 month, function preserved; partition delusions in VLOSLP; sensory loss + isolation are key risks; atypicals at ¼–⅓ dose	Xanomeline–trospium (KarXT) FDA 2024, first muscarinic antipsychotic; ATLAS — amisulpride 100 mg
Bipolar in late life	Late-onset mania = secondary mania until proved otherwise; irritability not euphoria; GERI-BD — lithium ≈ divalproex	ISBD OABD: lithium 0.4–0.8 mEq/L (60–79 y), 0.4–0.7 (≥80 y)
Somatoform / conversion	New onset after 60 is rare — exclude organic disease and depression; Hoover's sign; CBT first-line; avoid repeat investigations	ICD-11 Bodily Distress Disorder; FND is a rule-in diagnosis
Grief	Complicated grief ≥6 months; risk = sudden/traumatic death, ambivalent relationship; psychotherapy first-line	Prolonged Grief Disorder — DSM-5-TR ≥12 months, ICD-11 ≥6 months
Personality disorders	Cluster A prominent, B burns out, C persists; new-onset change = organic (FTD) until proved otherwise; Diogenes syndrome	ICD-11 — single dimensional diagnosis, severity + 5 trait domains
Sexual dysfunction	Desire persists in old age; ED predicts CVD; SSRIs commonest drug cause; PLISSIT model; PDE-5i first-line	Vaginal oestrogen / prasterone / ospemifene safe for GSM (NAMS-IMS)
Benzodiazepine dependence	10% community, 25% nursing home; SDS score ≥6; convert to diazepam and taper over months; withdrawal → confusion, seizures	ASAM 2025 — taper most older adults, 5–10% every 2–4 weeks, no forced taper; BIND
Nicotine dependence	Quitting benefits even in the 70s; 5 A's; combination NRT / varenicline / bupropion; cessation raises clozapine and olanzapine levels	Cytisinicline — ORCA-2/ORCA-3, efficacy like varenicline, fewer GI effects
Substance misuse	Three categories — medications, sanctioned substances, illicit; prescribed BZD/opioid misuse dominates; non-confrontational age-specific programmes work better	Cannabis fastest-rising in ≥65; Beers 2023 opioid + BZD/gabapentinoid caution
MHCA 2017	Sec 4 capacity, Sec 5 advance directive, Sec 14 NR, Sec 18 access, Sec 21(4) insurance, Sec 89/90 supported admission, Sec 115 suicide	Ayushman Bharat Vay Vandana — ₹5 lakh cover for all aged ≥70, mental health included
Capacity and consent	Capacity is clinical, competence is legal; four elements; decision-specific; unwise ≠ incapable; MacCAT-T / ACE; optimise before declaring absent	Supported over substituted decision-making (UNCRPD Art 12); 2024 Lancet Commission — early ACP
GDS	Yesavage 1982; somatic items excluded; GDS-15 cut-off ≥5; GDS-30 cut-off ≥11; invalid if MMSE <15 → Cornell Scale	USPSTF 2023 Grade B — screen all adults where follow-up exists
Mood, Anxiety & Suicide		
Depression — prevalence	MDD 1–5% community; symptoms 8–16%; higher in primary care, hospital and LTC; LASI ~8% in Indians ≥60	USPSTF 2023 Grade B — screen all adults including ≥65
Depression — presentation	"Depression without sadness"; somatic complaints; psychomotor symptoms; nihilistic delusions; anxious distress specifier; bereavement exclusion removed	Lancet Commission 2024 — depression a modifiable dementia risk factor
Depression — treatment	Stepped care; SSRI first-line at half dose; 6–12 weeks for full effect; ≥1 year after first recurrence, lifelong after second	OPTIMUM 2023 — aripiprazole augmentation, remission 28% vs 19%, fewer falls
Antidepressants	Sertraline/escitalopram first; avoid TCAs and paroxetine; hyponatraemia, falls, GI bleeding, QTc; mirtazapine if	Hyperbolic tapering (Maudsley 2024); withdrawal is not relapse

	anorexia + insomnia	
ECT	First-line if severe, psychotic, catatonic, suicidal or not eating; efficacy undiminished with age; seizure threshold rises; older age predicts better response; reversible cognitive effects only	ELEKT-D 2023 — ketamine non-inferior in non-psychotic TRD; ultrabrief right unilateral ECT
Suicide	WHO at age 75: men 50/100,000, women 16/100,000; attempt-to-completion 4:1 vs 200:1; 20–50% see the PCP within a week; depression strongest risk factor, self-harm strongest predictor	India NSPS 2022 — 10% reduction by 2030; NCRB elderly suicides up 40% from 2019 to 2022
Anxiety (general)	Not normal ageing; most prevalent yet most under-treated; agoraphobia and panic > phobias > GAD; SSRIs first-line, up to 6 weeks; no antipsychotics; benzodiazepines short-term only	USPSTF 2023 — Grade B for 19–64, **I-statement for ≥65**
GAD	Half lifelong, half onset within last 5 years; health-centred worry; buspirone an option; exclude medical, drug and withdrawal causes	MBSR non-inferior to escitalopram (JAMA Psychiatry 2023); Beers 2023
Phobias	Agoraphobia commonest; **fear of falling** is the geriatric-specific phobia, rare in the young; vicious cycle of activity restriction; graded exposure + CBT; avoid benzodiazepines	World Falls Guidelines — assess "concern about falling" in every falls assessment
OCD	DSM-5 own chapter; new onset after 60 is organic (basal ganglia, FTD); Y-BOCS; ERP first-line; SSRIs need higher dose and 10–12 weeks	TMS approved for OCD; deep TMS (H7 coil) for refractory cases
Mood disorders overall	DSM-5 split into depressive and bipolar; persistent depressive disorder ≥2 years; secondary mania; apathy is not depression	ISBD lithium 0.4–0.8 mEq/L (60–79 y), 0.4–0.7 (≥80 y)
Psychosis, Therapeutics, Personality & Alcohol		
Late-life psychosis	~60% of new-onset psychosis in late life is secondary; the **six D's** — delirium, disease, drugs, dementia, depression, delusional disorder; treat the cause, correct sensory loss, then low-dose atypical	Xanomeline–trospium (KarXT), FDA 2024 — first muscarinic antipsychotic
Late-onset schizophrenia	LOS 40–59 y, VLOSLP ≥60 y (late paraphrenia); female predominance; deafness and isolation; **partition delusions**; negative symptoms and thought disorder rare	ATLAS — amisulpride 100 mg/day, the only placebo-controlled RCT in VLOSLP
Atypical antipsychotics	D2 + 5-HT2A; quarter to a third of adult dose; quetiapine in DLB/PD; boxed warning of stroke and death in dementia; monitor weight, glucose, lipids, QTc, EPS; review every 3 months	Brexpiprazole, FDA May 2023 — first drug approved for agitation in Alzheimer's dementia
ECT	>50% of recipients are older adults; first-line if severe, psychotic, catatonic, suicidal or not eating; efficacy undiminished by age but seizure threshold rises; older age predicts better response; reversible cognitive effects; no absolute contraindication	ELEKT-D 2023 — ketamine non-inferior in non-psychotic TRD; ultrabrief right unilateral ECT
CBT	Structured, time-limited, 8–20 sessions; thought records, behavioural activation, graded exposure, homework; **problem-solving therapy** best where executive dysfunction; adapt for sensory and cognitive deficits	Digital and remote CBT effective in older adults; CBT-I first-line for chronic insomnia
Agitation — non-drug	Almost all people with dementia become agitated; look first for **pain, infection, constipation, retention, dehydration, drugs, delirium**; DICE and ABC; tailored activity, music, massage, animal-assisted, light therapy; carer training	IPA consensus definition of agitation; 2023–25 network meta-analyses favour tailored activity, massage, animal-assisted intervention
Personality change	Normal ageing = maturation, not rigidity; traits are accentuated under stress; **new personality change in old age is organic until proved otherwise** — FTD is the prototype; drugs do not help	ICD-11 — single dimensional diagnosis with severity + 5 trait domains; "personality change due to another medical condition" retained separately
Diogenes syndrome	Tetrad — extreme self-neglect, domestic squalor, social withdrawal, refusal of help, with hoarding and no shame; half are primary; premorbid paranoid/schizoid/OC traits; **home visit essential**; ~45% five-year mortality	Hoarding disorder now a distinct DSM-5/ICD-11 diagnosis; specialised CBT, not forced clearance
Alcoholism in the elderly	Early-onset "survivors" two-thirds vs late-onset "reactors" one-third; reduced body water and hepatic metabolism → higher levels; CAGE and **SMAST-G**; inpatient detox with lorazepam and thiamine; non-confrontational programmes double abstinence	WHO 2023 — no safe level; US Surgeon General 2025 — alcohol the third leading preventable cause of cancer
OP poisoning essay	DUMBBELS; decontaminate and protect staff; **atropine by**	AHA 2023 poisoning guidelines; India NSPS 2022 —

	dose doubling to clear chest, dry axillae, HR >80; pralidoxime early (benefit debated); watch intermediate syndrome at 24–96 h and OPIDN at 2–3 weeks; psychiatric assessment before discharge	pesticide restriction as the leading pillar

Cross-Cutting Themes the Examiner Rewards

1. **"Organic until proved otherwise"** — applies to late-onset psychosis, late-onset mania, new personality change, and new somatoform presentation. Write it every time.
2. **"Start low, go slow"** plus explicit numbers — antipsychotics at ¼–⅓ adult dose, mood stabilisers 30–50% lower, lithium 0.4–0.8 mEq/L.
3. **Non-pharmacological measures come first** — sensory correction, reducing isolation, CBT, psychoeducation, carer support.
4. **Deprescribing is treatment** — benzodiazepines, anticholinergics, opioids; cite Beers and STOPP/START.
5. **Indian context in every answer** — MHCA 2017, NPHCE, AB-PMJAY / Vay Vandana, Tele-MANAS 14416, Elder Line 14567, MWPSC Act 2007, NTCP quitline 1800-11-2356, LASI data.

Five Lines That Earn Marks in Every Answer in This Module

1. **"Asking about suicide does not increase risk — most patients are relieved and it strengthens trust."**
2. **"Antidepressants are just as effective in older people as in younger adults"** — tolerability, not efficacy, drives drug choice.
3. **"ECT is not a last resort — it is first-line in severe, psychotic, catatonic or acutely suicidal depression, and older age predicts a better response."**
4. **"Benzodiazepines: short-acting, short-term, and only for incapacitating symptoms"** — cognitive impairment, falls and fractures otherwise.
5. **"New-onset mania, OCD or personality change in old age is organic until proved otherwise."**

Cross-Module Differentiation Grid — How the Overlapping Questions Differ

Question	Pivot point — what the answer must emphasise
Q1 — Depression, full essay	Prevalence figures → atypical presentation → subtypes → management → **ECT as a dedicated section**
Q2 — Treatment and management	**Stepped-care model**, psychotherapy vs drugs, resistant depression pathway, duration of maintenance
Q3 — Antidepressant medication	**Pure pharmacology** — class-by-class, adverse effects, contraindications, augmentation, tapering
Q4 — Clinical vignette	**Safety first** → exclude organic cause of weight loss → drug table with side effects → structured follow-up schedule
Q10 — Mood disorders in late life	**Classification and the whole spectrum** — depressive + bipolar + secondary/organic + apathy and mimics

Six Lines That Earn Marks Across This Whole Module

1. **"About 60% of new-onset psychosis in late life is secondary — primary psychosis is a diagnosis of exclusion."**
2. **"Atypical antipsychotics are preferred over conventional agents — but all carry a boxed warning of increased stroke and death in dementia, and no drug is approved for the neuropsychiatric symptoms of dementia."**
3. **"ECT is not a last resort — older age predicts a better response, and its efficacy does not diminish with age."**
4. **"For agitation, look for pain, infection, constipation and drugs before reaching for any prescription."**
5. **"A personality change that begins in old age is organic until proved otherwise."**
6. **"In organophosphate poisoning, the endpoint of atropinisation is a clear chest and dry axillae — not the pupils."**

MD GERIATRIC MEDICINE — UNIVERSITY EXAMINATION

Compiled Question Bank

Renal · Fluid–Electrolyte · Diabetes · Endocrine · Metabolic Bone
Crystal Arthropathy · Environmental & Systemic Disorders

36 Questions · Mark-wise High-Yield Answers

Primary source: Pathy's Principles and Practice of Geriatric Medicine, 6th edition · Kane's Essentials of Clinical Geriatrics
Latest evidence: 2023–present international and Indian guidelines · Indian conventional units throughout

How to Use This Document

ANSWER ARCHITECTURE

Every answer follows the same four-block structure, so you always know where to look:

1. **Textbook Core** — numbered high-yield points drawn from the uploaded textbooks, with the keywords an examiner scans for in bold.
2. **Latest Update (2023–present)** — named trials, guideline changes and regulatory approvals, cited inline.
3. **Current Practice Update** — a comparison table, included *only* where a genuine conflict exists between the textbook position and current evidence, with an explicit exam-approach line.
4. **Recall aids** — exam one-liners, memory anchors, answer skeletons and named examiner traps.

Recurring examination questions carry a red flag in the header and receive additional scaffolding.

POINT-COUNT CONVENTION

Marks	Textbook core points	Latest updates	Total points
20-mark essay	14	3	**17**
15-mark essay	10	2	**12**
7-mark note	6	1	**7**
6-mark note	5	1	**6**

OVERLAPPING QUESTION PAIRS — READ BEFORE YOU REVISE

Several questions in this bank cover neighbouring ground. Each has been given a **distinct pivot** so that the answers do not repeat one another. Learn the pivot, not just the topic.

Pair	First question's pivot	Second question's pivot
Q3 vs Q4 & Q5 Fluid/electrolytes vs hyponatraemia and SIADH	**Q3** — the whole spectrum, grounded in ageing renal physiology	**Q4** — the diagnostic algorithm and correction rules for hyponatraemia of any cause; **Q5** — SIADH itself: criteria, exclusions, therapeutic ladder
Q10 vs Q12 AKI vs starting dialysis	**Q10** — treatment of the acute injury	**Q12** — the decision to start renal replacement therapy
Q12 vs Q13 Starting RRT vs modality	**Q12** — *when* to start (AEIOU, symptom-driven criteria)	**Q13** — *which* modality: PD, HD or conservative management
Q18 vs Q19 Drug selection vs drug class	**Q18** — case-based agent selection by comorbidity profile	**Q19** — pharmacology, outcome trials and geriatric cautions of the two classes
Q25, Q26, Q27 Hypothyroid cluster	**Q25** — when a raised TSH with normal T4 should and should not be treated	**Q26** — practical management in a cardiac patient; **Q27** — the disease itself, its antibodies, associations and complications
Q32 vs Q33 Gout cluster	**Q32** — diagnosis and treatment of the acute attack	**Q33** — the whole disease, weighted to urate-lowering therapy and treat-to-target

A NOTE ON SOURCE COVERAGE

Every answer is grounded in the uploaded textbooks as the primary source. **One topic is an exception:** chronic fatigue syndrome / myalgic encephalomyelitis (Question 36) is **not covered adequately in the uploaded textbooks**. Pathy's addresses *cancer-related fatigue* and refers to *idiopathic chronic fatigue in older adults* being linked to impaired skeletal-muscle mitochondrial content and biogenesis signalling; Kane's addresses fatigue as a non-specific presentation. Those elements are used where they apply, and the diagnostic frameworks are taken from the **IOM/NAM 2015 criteria**, the **Fukuda 1994 CDC criteria** and **NICE NG206 (2021)**, each clearly named in the text.

Contents

Part I — Renal Physiology and Assessment

1. Glomerular filtration rate *(6 marks)* 6

2. Renal biopsy — indications in the older adult *(7 marks)* 8

Part II — Fluid, Electrolyte and Acid–Base Disorders

3. Fluid and electrolyte disturbances in older patients and their management *(20 marks)* 9

4. Hyponatraemia *(7 marks · **recurring**)* 12

5. SIADH — syndrome of inappropriate antidiuretic hormone secretion *(20 marks)* 15

6. Hypokalaemia and hypomagnesaemia *(7 marks)* 18

7. A known diabetic on oral hypoglycaemic agents attends for routine review; serum potassium is 5.7 mEq/L. Investigation and treatment *(7 marks · **recurring**)* 19

8. Hypercalcaemia — causes, evaluation and management, including PTH-independent hypercalcaemia *(7 marks · **recurring**)* 21

9. Metabolic acidosis and its evaluation *(7 marks)* 25

Part III — Kidney Disease

10. Management of acute kidney injury *(7 marks · **recurring**)* 27

11. Chronic kidney disease — etiopathogenesis, diagnosis and management, with conservative management in the elderly *(15 marks · **recurring**)* 28

12. Criteria for initiating renal replacement therapy, with special emphasis on the elderly in our country *(7 marks · **recurring**)* 31

13. Peritoneal dialysis versus haemodialysis in the very old *(7 marks)* 33

14. Acute glomerulonephritis *(7 marks)* 34

15. Nephrotic syndrome and membranous nephropathy in the elderly *(15 marks)* 36

16. Interstitial nephritis *(7 marks)* 37

17. Diabetic nephropathy and its staging *(15 marks)* 39

Part IV — Diabetes Mellitus

18. A 65-year-old newly diagnosed diabetic with HbA1c 10%, hypertension, dyslipidaemia, osteoarthritis and BMI 35 refuses to adhere to diet and exercise. Which antidiabetic to start, and what to add *(7 marks)* 42

19. SGLT2 inhibitors and GLP-1 receptor agonists in the older adult *(15 marks)* 45

20. Alpha-glucosidase inhibitors *(6 marks)* 47

21. Newer insulins — onset, duration and advantages *(7 marks)* 48

22. Hypoglycaemia — evaluation and management *(15 marks)* 50

23. Diabetic ketoacidosis and hyperosmolar hyperglycaemic state *(15 marks)* 52

24. Diabetic foot syndrome *(15 marks)* 55

Part V — Thyroid Disease

25. Subclinical hypothyroidism in the elderly *(7 marks · **recurring**)* 58

26. A 70-year-old on treatment for cardiac failure has TSH 50 µIU/mL with suppressed T3 and T4, constipation, cold intolerance and considerable weight gain. Treatment, follow-up and specific instructions *(7 marks)* 61

27. Hashimoto's thyroiditis *(7 marks)* 63

28. Hyperthyroidism in the elderly — presentation, investigation and treatment *(7 marks)* 65

29. Solitary thyroid nodule and thyroid malignancy *(7 marks)* 67

Part VI — Endocrine and Metabolic Bone Disease

30. Testosterone replacement and andropause *(7 marks)* 69

31. Osteomalacia and vitamin D deficiency *(15 marks)* 71

Part VII — Crystal Arthropathy

32. A 65-year-old man presents with acute onset of severe pain in the right big toe for two days following a binge of alcohol. Most probable diagnosis and management *(7 marks)* 74

33. Management of gout *(7 marks)* 76

34. Pseudogout *(7 marks)* 79

Part VIII — Environmental and Systemic Disorders

35. Heat stroke — clinical features, diagnostic criteria, differential diagnosis and treatment *(7 marks)* 82

36. Chronic fatigue syndrome — diagnostic criteria, clinical manifestations, predisposing factors, course, prognosis and treatment *(15 marks)* 85

Night-Before Master Sheet — All 36 Questions 90

PART I

Renal Physiology and Assessment

Q1 · Q2

QUESTION 1 · RENAL PHYSIOLOGY

Glomerular filtration rate

6-Mark Note — 5 textbook points + 1 latest update = 6 points

TEXTBOOK CORE — 5 POINTS

1. **Definition and the trajectory with age.** GFR is the volume of plasma filtered by the glomeruli per unit time, expressed as **mL/min/1.73 m^2**. It **peaks at approximately 140 mL/min/1.73 m^2 in the third decade** and then **declines at about 8 mL/min/1.73 m^2 per decade**. Effective renal plasma flow falls **proportionally more — about 10% per decade, from 600 mL/min in youth to 300 mL/min by age 80** — so the **filtration fraction (GFR/ERPF) actually increases in the elderly**. Importantly, **in approximately one-third of older adults GFR does not decrease with age at all**.
2. **Why serum creatinine misleads in old age.** The fall in creatinine clearance is **accompanied by a concomitant decrease in creatinine production owing to senile sarcopenia, so serum creatinine does not increase despite the progressive decrease in GFR**. Healthy older people additionally show **net creatinine tubular reabsorption**, so measured creatinine clearance **underestimates** true glomerular filtration. The consequence to quote: **a serum creatinine of 1 mg/dL reflects a GFR of 120 mL/min in a 20-year-old but only 60 mL/min in an 80-year-old.**
3. **The estimating equations. Cockcroft–Gault:** creatinine clearance = **(140 – age) × body weight ÷ (72 × serum creatinine)**, and **15% lower in women**. **MDRD** and **CKD-EPI** are the two other standard formulae. Regarding **cystatin C** as a GFR marker, it has been demonstrated that **in elderly people with glomerular filtration below 60 mL/min it is not superior to that calculated by the Cockcroft–Gault and MDRD formulae**. Keller's simple approximation is **GFR = 130 – age (mL/min)** for people between 25 and 100 years, though this has not been validated against cimetidine creatinine clearance.
4. **Choosing the right equation — a favourite examination point. Cockcroft–Gault underestimates GFR in people over 80**, while **MDRD has the advantage of not requiring the patient's weight**. **CKD-EPI tends to be the most accurate choice at higher levels of GFR and is recommended for estimating creatinine clearance in adults without known chronic kidney disease**, and for risk stratification before percutaneous coronary intervention. **However, when dosing medications based on a patient's GFR, the Cockcroft–Gault equation remains the standard of care and should be used.**
5. **Consequences of senile hypofiltration. (a) Senile hypofiltration and diastolic cardiac failure predispose healthy old people to cardiac failure and to lung congestion after a saline load. (b) The dose of every prescribed drug must be adjusted to the estimated GFR in the elderly. (c) Senile hypofiltration differs**

from chronic kidney disease because the glomerular filtration value is **stable over 6–12 months** and there is **no haematuria and no significant proteinuria (>0.30 g/day)** — a distinction that prevents the over-labelling of healthy older people as having CKD.

Equation	Needs	Strength	Limitation / best use
Cockcroft–Gault	Age, weight, creatinine, sex	Remains the **standard of care for drug dosing**	Underestimates GFR in those over 80; distorted by obesity and oedema
MDRD	Age, creatinine, sex	No weight required	Poor correlation with measured clearance; inaccurate at high GFR
CKD-EPI	Age, creatinine, sex	Most accurate at higher GFR; recommended in adults without known CKD	Still creatinine-dependent, so distorted by sarcopenia
Cystatin C	Cystatin C ± creatinine	Independent of muscle mass	Not superior to CG or MDRD in the elderly below 60 mL/min; cost and availability

LATEST UPDATE (2023–PRESENT) — 1 POINT

6. **KDIGO 2024 CKD guideline — combined creatinine–cystatin C estimation becomes the preferred standard.** The guideline recommends **using a validated GFR estimating equation to derive GFR from serum filtration markers rather than relying on the markers alone**; that **in adults at risk of CKD, creatinine-based eGFR should be used, and if cystatin C is available the GFR category should be estimated from the combination of creatinine and cystatin C (eGFRcr-cys)**; and specifically that **eGFRcr-cys should be used in clinical situations where creatinine-based eGFR is less accurate and GFR affects clinical decision-making** — which describes the **sarcopenic, malnourished, amputee, cirrhotic or very old patient** exactly. The equations are now **race-free**. Two further points of practical relevance: **point-of-care testing for creatinine and urine albumin may be used where laboratory access is limited** — directly applicable to rural India — and **measured GFR using an exogenous filtration marker remains the reference standard** when a more accurate assessment is needed for clinical decision-making.

EXAM ONE-LINERS

- GFR peaks at **140** in the third decade, then falls **8 mL/min/1.73 m² per decade**.
- Filtration fraction **rises** with age, because renal plasma flow falls faster than GFR.
- Creatinine **1 mg/dL** = GFR **120** at 20 years, **60** at 80 years.
- **Cockcroft–Gault for drug dosing; CKD-EPI for staging; eGFRcr-cys when creatinine is unreliable.**
- **One-third of older adults show no age-related decline in GFR** — ageing is not uniform.
- Senile hypofiltration = **stable over 6–12 months, no haematuria, proteinuria <0.30 g/day**.

QUESTION 2 · NEPHROLOGY

Renal biopsy — indications in the older adult

7-Mark Answer — 6 textbook points + 1 latest update = 7 points

TEXTBOOK CORE — 6 POINTS

1. **Age alone is not a contraindication.** The **indications and the incidence of complications of renal biopsy are the same** for older and younger adults. However, **when a complication does occur in an older adult it is generally more serious** — so patient selection and post-procedure monitoring matter more.
2. **Main indications. Nephrotic syndrome** (accounts for **50% of biopsy indications** in the elderly), **unexplained acute kidney injury**, suspected **rapidly progressive glomerulonephritis**, unexplained CKD with **normal-sized kidneys**, non-nephrotic proteinuria with haematuria, and suspected systemic disease — **vasculitis, myeloma, amyloidosis, lupus**.
3. **The yield is high and clinically useful.** Although older adults formed only 23% of the biopsy series, they were **over-represented relative to their share of the population**, and **primary glomerulonephritis was the most frequent biopsy-proven renal disease** in this group — more frequent than in younger adults.
4. **A biopsy finds treatable disease that is easily missed. Crescentic glomerulonephritis peaks at 60–79 years** and presents as rapidly progressive renal failure; **renal vasculitis** is probably the commonest primary cause of renal failure in older adults. Both respond to steroids and cyclophosphamide.
5. **Biopsy also drives the search for a secondary cause.** About **20% of membranous nephropathy in the elderly is secondary** to an underlying malignancy (chiefly lung and colon adenocarcinoma) or to drugs; minimal change disease may be caused by **NSAIDs** or associated with **lymphoma**. The histological diagnosis therefore triggers a malignancy and drug review.
6. **Contraindications and precautions.** Uncontrolled hypertension, bleeding diathesis or continuing anticoagulation/antiplatelet therapy, solitary kidney (relative), **small echogenic kidneys**, active urinary tract infection, large cysts or tumour, and an uncooperative patient. Before biopsy: control BP, stop antiplatelets and anticoagulants, check haemoglobin, platelets and coagulation, and ultrasound for kidney size and number. Afterwards watch for **haematuria, perinephric haematoma and arteriovenous fistula**.

LATEST UPDATE (2023–PRESENT) — 1 POINT

7. **KDIGO Glomerular Diseases guideline — the "biopsy-free" exception, with an important geriatric caveat.** In a patient with **nephrotic syndrome, a positive anti-PLA2R antibody and normal kidney function**, biopsy is **not required** to diagnose membranous nephropathy. But in older adults, membranous nephropathy can **co-exist with renal amyloidosis or diabetic nephropathy** — conditions with entirely different treatment and prognosis — so biopsy should still be performed when the clinical picture is atypical, when renal function is declining, or when a plasma-cell dyscrasia is suspected.

EXAM ONE-LINERS

- Commonest indication for renal biopsy in the elderly = **nephrotic syndrome (50%)**.
- Same indications, same complication rate — but **more serious complications** when they occur.
- Small echogenic kidneys = biopsy will show **only scarring** — do not biopsy.
- Elderly + AKI + haematuria + systemic symptoms = **biopsy for vasculitis, do not delay**.

PART II

Fluid, Electrolyte and Acid–Base Disorders

Q3 · Q4 · Q5 · Q6 · Q7 · Q8 · Q9

QUESTION 3 · FLUID, ELECTROLYTE & ACID–BASE

Fluid and electrolyte disturbances in older patients and their management

20-Mark Essay — 14 textbook points + 3 latest updates = 17 points

TEXTBOOK CORE — 14 POINTS

1. **The ageing kidney is the substrate for every disturbance.** GFR peaks at about **140 mL/min/1.73 m^2 in the third decade** and then falls by roughly **8 mL/min/1.73 m^2 per decade**; effective renal plasma flow falls proportionally more (10% per decade), so the **filtration fraction rises**. Add **aglomerular circulation**, **medullary hypotonicity**, **tubular dysfunction** and **tubular frailty** — the ageing kidney has narrowed margins in every direction.
2. **Body water is reduced, and the loss is intracellular.** Total body water is lower in older people and lower in elderly women than elderly men. Because **the reduction takes place in the intracellular compartment**, **hypovolaemia always represents a pathological state in older adults** — it is never a normal age change.
3. **Thirst is blunted.** When healthy, active older volunteers were water-restricted for 24 hours, their **threshold for thirst was increased and water intake reduced** compared with younger controls. Contributors: dryness of the mouth, decreased taste, altered mental capacity or cortical cerebral dysfunction, **reduced sensitivity of both osmoreceptors and baroreceptors**, and a lower concentration of **angiotensin**, a powerful generator of thirst.
4. **Urinary concentrating ability is impaired.** Maximum urinary concentration remains normal until about the third decade, then falls by about **30 mOsm/kg per decade**. Mechanisms: aglomerular circulation, defective sodium reabsorption in the ascending limb of Henle, reduced distal urea reabsorption, and **decreased responsiveness of the collecting tubule epithelium to antidiuretic hormone** — which is why **plasma vasopressin levels are higher in older adults**.
5. **Urinary diluting ability is also impaired.** Minimum urine concentration is only **92 mOsm/kg in the elderly versus 52 mOsm/kg in the young**, and **maximum free water clearance falls from 16.2 to 5.9 mL/min**. **Clinical consequence:** older people develop **dehydration and hypernatraemia under water restriction**, and **volume overload and hyponatraemia under a water load**.
6. **Sodium is wasted and conserved slowly.** The **24-hour urinary sodium output and fractional excretion of sodium are significantly greater** in old and very old people. Clinically this produces **slow adaptation to salt restriction or salt loss**, orthostatic hypotension, and a tendency to hypovolaemia during illness, fasting or bowel preparation.

7. **Potassium — depleted stores but a tendency to retain. Total body potassium is lower** (reduced muscle mass, poor intake), yet **renal potassium excretion is significantly lower in the aged** because of **reduced serum aldosterone and reduced tubular response to aldosterone**, plus increased **H^+/K^+-ATPase activity in the intercalated cells** of the aged collecting duct. Result: serum potassium is usually normal, but older people develop **hypokalaemia more rapidly on diuretics** and are **predisposed to hyperkalaemia on NSAIDs, ACE inhibitors, beta blockers and potassium-sparing diuretics**.
8. **Urea and creatinine handling changes.** The **fractional excretion of urea is increased — 65% in the old versus 50% in the young** — contributing to **osmotic diuresis and nocturia**, medullary hypotonicity, and a **characteristically low serum urea in the healthy older person**. Healthy older people also show **net creatinine tubular reabsorption**, so creatinine clearance **underestimates** true glomerular filtration.
9. **Calcium, phosphate and magnesium.** Serum levels and fractional excretion are **similar in the healthy young, old and very old**. However, low dietary vitamin D, reduced sunlight exposure, decreased renal hydroxylation of vitamin D and low sex hormone levels, combined with **poor intestinal calcium absorption**, lead to **senile secondary hyperparathyroidism**. On volume overload, **renal magnesium excretion increases enough to significantly lower serum magnesium**.
10. **Acid–base.** The ageing kidney **handles an acute acid load poorly** — acidification is slower and recovery is slower — so metabolic acidosis is both commoner and more prolonged. **Type 4 renal tubular acidosis** (hyporeninaemic hypoaldosteronism) is common in the older diabetic and presents as a **normal anion gap acidosis with hyperkalaemia**.
11. **Dehydration is the commonest disturbance.** Causes: **reduced intake** — dementia, delirium, immobility, dysphagia, depression, fear of incontinence, restricted access to fluid, restraint; and **increased losses** — fever, diarrhoea, vomiting, diuretics, uncontrolled hyperglycaemia, hot weather. **Physical signs are unreliable in the elderly**: skin turgor and mucous membranes mislead. Use instead **serial daily weight, postural blood pressure, urine output, urea:creatinine ratio and serum osmolality**.
12. **Hyponatraemia — a serious and often overlooked problem of the older patient.** Three general causes: **(a)** decreased renal blood flow with a decreased ability to excrete a water load; **(b) diuretic administration leading to water intoxication** — a condition rapidly corrected by discontinuing the diuretic; and **(c)** excess vasopressin secretion. Older adults seem **more prone to develop SIADH**, and certain drugs such as **chlorpropamide and barbiturates** cause it more frequently in older individuals. **Elderly patients may manifest cognitive impairment with even mild hyponatraemia.**
13. **Hypernatraemia — the mirror image, with higher mortality.** Almost always due to **water deficit rather than salt excess**: the febrile, immobile, tube-fed or institutionalised patient who cannot access water. Correct the free water deficit **slowly (≤10–12 mEq/L per 24 hours)** to avoid cerebral oedema, preferably by the enteral route.
14. **Principles of management common to all these disturbances. (i)** Identify and treat the underlying cause. **(ii) Medication review is the single highest-yield intervention** — diuretics, SSRIs, carbamazepine, NSAIDs, ACE inhibitors, PPIs, laxatives. **(iii)** Correct **slowly**: rapid correction of serum sodium **should not exceed a 20 mEq/L rise during the first 48 hours (1 mEq/L/h)**, to avoid neurological damage and **central pontine myelinolysis**. **(iv)** Give fluid cautiously — **senile hypofiltration and diastolic cardiac failure predispose healthy old people to cardiac failure and lung congestion after a saline load**. **(v)** Prefer the oral route; use **subcutaneous hypodermoclysis** where venous access is difficult in the frail. **(vi)** Monitor daily weight, input–output and electrolytes; **adjust every drug dose to the estimated GFR**.

Age change	Physiological consequence	Clinical disturbance	Practical guard
↓ Thirst threshold, ↓ osmoreceptor sensitivity	Inadequate water intake for a given osmolality	Dehydration, hypernatraemia	Scheduled fluid rounds; do not rely on the patient asking
↓ Concentrating ability (−30 mOsm/kg per decade)	Obligatory water loss continues despite deficit	Hypernatraemia, nocturia	Daily weights; early enteral water
↓ Diluting ability (free water clearance 5.9 mL/min)	Cannot excrete a water load	Hyponatraemia	Avoid hypotonic IV fluids; review thiazides and SSRIs
↑ FENa, ↑ FEurea	Salt and urea wasting	Hypovolaemia, postural hypotension, low serum urea	Do not over-interpret a "normal" urea or a high FENa
↓ Aldosterone and tubular response	Impaired potassium excretion	Hyperkalaemia on RAS blockers and NSAIDs	Check K^+ 1–2 weeks after any RASi change
↓ Muscle mass, net creatinine reabsorption	Serum creatinine stays "normal"	Occult renal impairment, drug toxicity	Dose by estimated GFR, never by creatinine alone

LATEST UPDATES (2023–PRESENT) — 3 POINTS

15. **Correction limits have tightened, and re-lowering is now standard practice.** Current guidance caps sodium correction at **≤8–10 mEq/L in the first 24 hours and ≤18 mEq/L over 48 hours**, and at **≤6–8 mEq/L per 24 hours in those at high risk of osmotic demyelination** — malnutrition, alcohol use, hypokalaemia, liver disease and initial sodium below 105 mEq/L, all common in frail older patients. If the limit is breached, **active re-lowering with 5% dextrose ± desmopressin (the "DDAVP clamp")** is recommended, and proactive desmopressin co-administration from the outset is increasingly used to make correction predictable.
16. **SGLT2 inhibitors have entered electrolyte medicine.** In a randomised, double-blind, placebo-controlled crossover trial in outpatients with chronic SIAD (*JASN* 2023), four weeks of **empagliflozin 25 mg** added to fluid restriction raised median serum sodium by **4.1 mEq/L** versus placebo, with no hypoglycaemia, hypotension, overcorrection or AKI, and a small improvement in neurocognitive function. Separately, SGLT2 inhibitors **raise serum magnesium** — a useful incidental effect in the older patient on a diuretic or a proton-pump inhibitor.
17. **Potassium binders have changed the hyperkalaemia doctrine.** The **KDIGO 2024 CKD guideline** states that potassium binders **may facilitate essential use of RAS inhibitors and mineralocorticoid receptor antagonists**, that **binders should be considered before reducing the RAS inhibitor**, and that **RASi discontinuation should be the last resort**. **Sodium zirconium cyclosilicate** and **patiromer** are the modern agents; **sodium polystyrene sulphonate has been removed from acute guidance** because of weak evidence and the risk of colonic necrosis.

CURRENT PRACTICE UPDATE

Aspect	Textbook	Current recommendation
Sodium correction rate	Up to 20 mEq/L in 48 h (1 mEq/L/h)	≤8–10 mEq/L in 24 h; ≤6–8 mEq/L in high-risk patients; re-lower if exceeded
Hyperkalaemia on a RAS blocker	Reduce or stop the ACE inhibitor	Add a potassium binder and keep the RAS blocker; stopping is the last resort
Cation-exchange resin	Calcium/sodium polystyrene sulphonate	Replaced by SZC or patiromer; resin withdrawn from acute algorithms

Exam approach: Quote the textbook 20 mEq/48 h figure as the classical teaching, then state the modern 8–10 mEq/24 h cap. Writing both, and naming osmotic demyelination, is what separates a pass from a distinction here.

MEMORY ANCHORS & ESSAY SKELETON

Skeleton (20 marks, 4 paragraphs): (1) Ageing renal physiology — thirst, concentrating, diluting, sodium, potassium, urea, calcium, acid–base. (2) The specific disturbances — dehydration, hyponatraemia, hypernatraemia, hypo/hyperkalaemia, hypo/hypercalcaemia, acidosis. (3) Assessment — why signs mislead; what to measure. (4) Management principles — cause, drug review, slow correction, cautious volume, monitoring. Close with the three updates.

Numbers to memorise: GFR peak 140, −8 per decade · concentrating −30 mOsm/kg per decade · min urine osm 92 vs 52 · free water clearance 5.9 vs 16.2 mL/min · FEurea 65% vs 50% · creatinine 1 mg/dL = GFR 120 at 20 years, 60 at 80 years · Na correction ≤8–10 mEq/24 h.

EXAMINER TRAPS

- Listing electrolyte abnormalities without first explaining the **ageing physiology that causes them** — this is what the 20-mark format is testing.
- Assessing dehydration by **skin turgor** in an 85-year-old.
- Reassuring yourself with a **normal serum urea** — it is characteristically low in the healthy old because of urea wasting.
- Aggressive saline resuscitation without noting that **senile hypofiltration plus diastolic failure causes pulmonary congestion after a saline load**.
- Forgetting that **hypovolaemia in an older adult is always pathological**, never a normal age change.

QUESTION 4 · SODIUM & WATER

Hyponatraemia

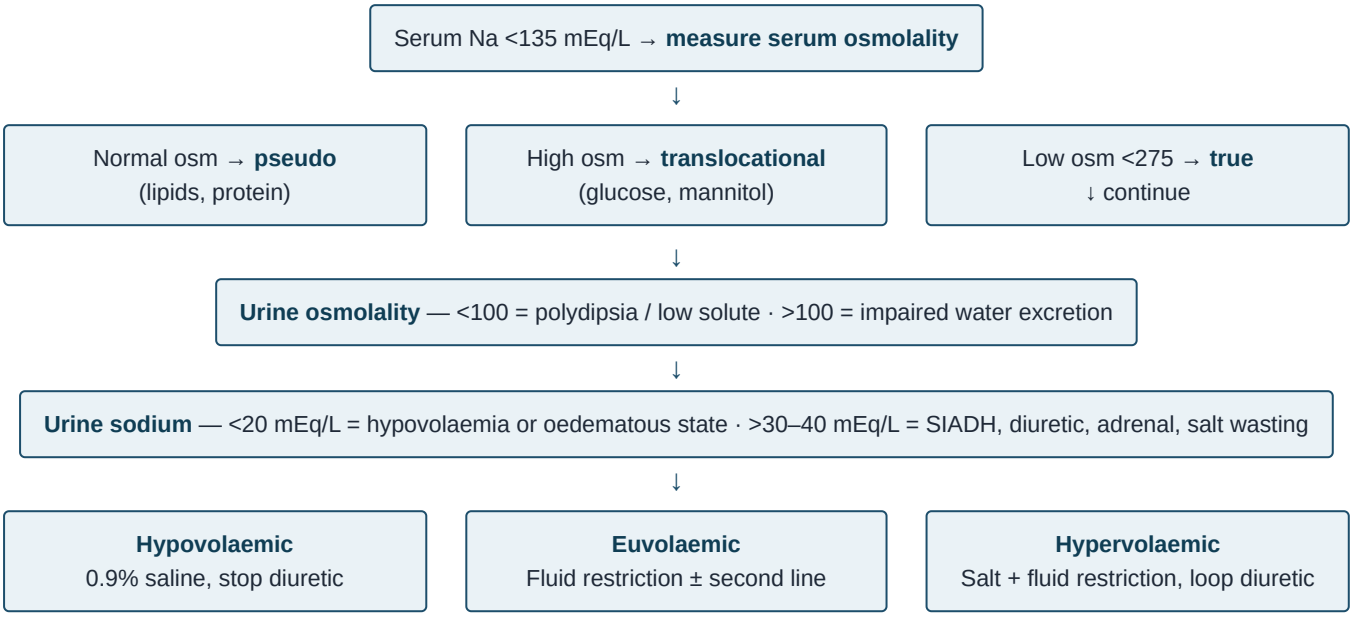
7-Mark Note — 6 textbook points + 1 latest update = 7 points

Recurring question — asked ×3. Extra scaffolding provided below.

TEXTBOOK CORE — 6 POINTS

1. **Definition, severity and significance.** Serum sodium **<135 mEq/L**; mild 130–134, moderate 125–129, **severe or profound <125 mEq/L**. It is the commonest electrolyte abnormality in hospitalised older adults and is described as **"a serious and often overlooked problem of the older patient"**. Critically, **elderly patients are more susceptible and may manifest cognitive impairment with even mild hyponatraemia**.

2. **Why older people are predisposed. Impaired water excretion** — minimum urine osmolality is only **92 mOsm/kg versus 52 in the young**, and maximum free water clearance falls from **16.2 to 5.9 mL/min**. In addition, infusion of hypertonic saline produces a **greater rise in plasma vasopressin in older than in younger persons**, and older adults are heavily exposed to the precipitating drugs.
3. **The three general causes. (a)** Decreased renal blood flow with a **decreased ability to excrete a water load**; **(b) diuretic administration leading to water intoxication** — a condition **rapidly corrected by discontinuing the diuretic**; and **(c) excess vasopressin secretion (SIADH)**, to which older adults are more prone. The drugs to interrogate: **thiazides** (the classic), **SSRIs and other antidepressants** (risk increases with age, female sex, low body weight and renal failure), **carbamazepine**, **chlorpropamide and barbiturates** (more frequent in older individuals), NSAIDs, PPIs, opioids and desmopressin.
4. **Step 1 of evaluation — is it true hypotonic hyponatraemia?** Measure **serum osmolality**. **Normal osmolality** = **pseudohyponatraemia** (severe hyperlipidaemia, hyperproteinaemia, paraproteinaemia). **High osmolality** = **translocational** hyponatraemia (hyperglycaemia, mannitol infusion). **Low osmolality (<275 mOsm/kg)** = true hypotonic hyponatraemia — proceed.
5. **Step 2 — urine osmolality, then urine sodium, then volume status. Urine osmolality <100 mOsm/kg** = appropriately dilute urine → **primary polydipsia** or **low solute intake** ("tea-and-toast" diet, beer potomania) — both common in the socially isolated older person. **Urine osmolality >100** = impaired water excretion → check **urine sodium**: **<20 mEq/L** suggests **hypovolaemia or an oedematous state** (heart failure, cirrhosis, nephrotic syndrome); **>30–40 mEq/L** suggests **SIADH, diuretic use, adrenal insufficiency or salt wasting**. Always also check **TSH and a 9 a.m. cortisol**.
6. **Step 3 — treat by volume status, and correct slowly. Hypovolaemic** → 0.9% saline and stop the diuretic. **Euvolaemic (SIADH)** → fluid restriction. **Hypervolaemic** → salt and fluid restriction with a loop diuretic; treat the heart or liver disease. **Water restriction and hypertonic saline are indicated when the patient is symptomatic or the sodium level is below 120 mEq/L**; in an emergency give **3% saline 100–150 mL over 10–20 minutes**, repeated up to three times until symptoms abate or sodium rises 4–6 mEq/L. **This rapid correction should not exceed a 20 mEq/L rise during the first 48 hours (1 mEq/L/h), to avoid neurological damage and central pontine myelinolysis.**



LATEST UPDATE (2023–PRESENT) — 1 POINT

7. **Correction limits have tightened, and deliberate re-lowering is now standard.** Current practice caps correction at **≤8–10 mEq/L in the first 24 hours and ≤18 mEq/L over 48 hours**, and at **≤6–8 mEq/L per 24 hours in patients at high risk of osmotic demyelination syndrome — malnutrition, alcohol use, hypokalaemia, liver disease, and initial sodium below 105 mEq/L**, a risk profile that describes a large share of frail older inpatients. If the cap is breached, **active re-lowering with 5% dextrose with or without desmopressin (the "DDAVP clamp")** is recommended, and **proactive desmopressin co-administration** from the outset is increasingly used to make the rise predictable. Note also the danger of **autocorrection**: once a hypovolaemic stimulus or a thiazide is removed, vasopressin switches off and the sodium can rise dangerously fast on its own — so recheck sodium **every 4–6 hours** in the first day.

CURRENT PRACTICE UPDATE

Aspect	Textbook	Current recommendation
Maximum correction	20 mEq/L over 48 h (1 mEq/L/h)	≤8–10 mEq/L in 24 h; ≤6–8 in high-risk patients
Overcorrection	Avoid it	Actively re-lower with 5% dextrose ± desmopressin
Emergency therapy	Hypertonic saline infusion	3% saline as 100–150 mL boluses, targeting a 4–6 mEq/L rise

Exam approach: Write the textbook 20 mEq/48 h rule *and* the modern 8–10 mEq/24 h cap, and name **osmotic demyelination syndrome** (the current term for central pontine myelinolysis). Both together is the full-mark answer.

MEMORY ANCHORS & 15-SECOND SKELETON

Skeleton: Define and grade → why the old are vulnerable (diluting defect + vasopressin + drugs) → three general causes → **serum osmolality** → **urine osmolality** → **urine sodium** → **volume status** → treat by volume compartment → correction rate and osmotic demyelination.

Numbers: 135 / 130 / 125 / 120 · urine osm cut-off **100** · urine Na cut-offs **20 and 30–40** · serum osm **275** · 3% saline **100–150 mL** boluses · target rise **4–6** · cap **8–10 per 24 h** · min urine osm **92 vs 52**.

EXAMINER TRAPS

- Diagnosing SIADH **without excluding hypothyroidism, adrenal insufficiency and diuretic use** — the commonest error in the whole topic.
- Skipping **serum osmolality** and treating a pseudohyponatraemia.
- Giving **0.9% saline to a patient with SIADH** — the urine is more concentrated than the infusate, so free water is retained and the sodium *falls further*.
- Forgetting the risk of **autocorrection** after the thiazide is stopped or volume is restored.
- Treating the number rather than asking **"is this patient symptomatic, and is it acute or chronic?"** — that question dictates everything.

QUESTION 5 · SODIUM & WATER

SIADH — syndrome of inappropriate antidiuretic hormone secretion

20-Mark Essay — 14 textbook points + 3 latest updates = 17 points

TEXTBOOK CORE — 14 POINTS

1. **Definition.** A **euvolaemic, hypotonic hyponatraemia** caused by **non-osmotic, non-physiological release of antidiuretic hormone (arginine vasopressin)**, with continued renal water reabsorption despite plasma hypo-osmolality. It is now often called **SIAD (syndrome of inappropriate antidiuresis)**, because a minority of cases are due to a **gain-of-function V2 receptor mutation** with undetectable circulating ADH.
2. **The four textbook diagnostic features. (i) Inappropriately high urine osmolality for the concomitant plasma hypo-osmolality**; **(ii) continued renal excretion of sodium**; **(iii) clinical normovolaemia**; and **(iv) normal renal, adrenal and thyroid function**. All four must be satisfied.
3. **Full Bartter–Schwartz criteria — essential.** Hypotonic hyponatraemia (Na <135 mEq/L, effective osmolality <275 mOsm/kg); **urine osmolality >100 mOsm/kg**; clinical euvolaemia with no oedema and no signs of volume depletion; **urine sodium >30–40 mEq/L** on normal salt and water intake; normal thyroid and adrenal function; and **no recent diuretic use**.
4. **Supplemental criteria that strengthen the diagnosis. Low serum uric acid (<4 mg/dL)** and **low blood urea** — both reflect dilution and increased fractional excretion; **fractional excretion of urate >12%**; fractional excretion of sodium >0.5%; **failure to correct with 0.9% saline**; and correction with fluid restriction. A **normal or high uric acid should make you doubt the diagnosis**.
5. **Pathophysiology.** Excess ADH drives **aquaporin-2 insertion into the collecting duct** → water retention → mild volume expansion → **suppression of aldosterone and release of natriuretic peptides** → **secondary natriuresis**. The patient therefore remains **clinically euvolaemic while the sodium falls**, and continues to excrete sodium in the urine. **Vasopressin escape** limits further water retention and prevents gross oedema — which is why SIADH does not cause oedema.
6. **Ageing itself is a predisposing factor.** Infusion of hypertonic saline leads to a **greater increase in plasma vasopressin in older compared with younger persons**, possibly through impaired baroreceptor input to the supraoptic nucleus. Combined with the reduced diluting capacity of the ageing kidney, this means **older adults seem more prone to develop SIADH** from any given trigger.
7. **Causes — central nervous system. Stroke, meningitis, subdural haematoma, tumour, intracranial metastases, haemorrhage, head injury and encephalitis.** Any acute intracranial event in an older adult should prompt a sodium check.
8. **Causes — pulmonary. Pneumonia** (a very common trigger in the elderly), **tuberculosis** (highly relevant in India), lung abscess, pulmonary metastases and infection, and positive-pressure ventilation.
9. **Causes — malignancy.** SIADH is a **paraneoplastic condition classically associated with small-cell carcinoma of the lung**; also head and neck cancers, thymoma, and others. In an older smoker with unexplained SIADH, **image the chest**.
10. **Causes — drugs, which are the commonest in geriatric practice. SSRIs and other antidepressants** — the risk of antidepressant-induced hyponatraemia **increases with age** and is associated with **female sex, low body weight, renal failure** and concurrent drugs that cause hyponatraemia. Also **carbamazepine and oxcarbazepine**, **chlorpropamide**, **barbiturates** (both of which **cause this syndrome more frequently in older individuals**), thiazides, antipsychotics, NSAIDs, opioids, PPIs, desmopressin, and the chemotherapeutic agents **vincristine, vinblastine, ifosfamide and cyclophosphamide**.

11. **Other causes. Pain, nausea, surgery and general anaesthesia** (all potent non-osmotic ADH stimuli — a frequent cause of postoperative hyponatraemia in the elderly), HIV infection, and **idiopathic SIAD of the elderly**, a recognised entity in which no cause is found and the hyponatraemia is chronic and recurring.
12. **Clinical features. Symptoms depend on the depth and the rate of development**, but **elderly patients are more susceptible and may manifest cognitive impairment with mild hyponatraemia**. **Acute** (<48 hours) — headache, nausea, vomiting, confusion, seizures, coma and respiratory arrest from cerebral oedema. **Chronic** — apparently "asymptomatic", but in reality associated with **gait instability, attention and memory deficits, falls, osteoporosis and fracture** (chronic hyponatraemia mobilises sodium from bone). **In an older adult, chronic hyponatraemia is almost never truly asymptomatic.**
13. **Differential diagnosis to exclude before you accept the label. Hypovolaemia**; **diuretic use, especially thiazides**; **hypothyroidism**; **adrenal insufficiency** — a 9 a.m. cortisol is mandatory, because glucocorticoid deficiency mimics SIADH exactly; **cerebral salt wasting** — which is **hypovolaemic with a high urine output and negative salt balance, and is treated with salt and fluid, the opposite of SIADH**; and low-solute states such as beer potomania and the tea-and-toast diet.
14. **Treatment. Treat the underlying disease — successful therapy for paraneoplastic SIADH requires treatment of the underlying disease by chemotherapy and/or radiotherapy**. **Acute measures are indicated in symptomatic patients with severe hyponatraemia (plasma sodium <125 mEq/L)**: the textbook protocol is to **initiate and maintain rapid diuresis with intravenous furosemide while restricting free water intake to 500–1000 mL per day, and to replace the sodium and potassium lost in the urine with 0.9% saline infusions with added potassium**; current emergency practice is **3% hypertonic saline 100–150 mL over 10–20 minutes, repeated up to three times, targeting a rise of 4–6 mEq/L** to abort seizures. **This rapid correction should not exceed a 20 mEq/L rise in serum sodium during the first 48 hours (1 mEq/L/h) to avoid neurological damage and central pontine myelinolysis.** For **chronic SIADH**: **fluid restriction to 500–1000 mL/day is first line**; then increased solute intake (salt and protein, or **oral urea 15–30 g/day**); a loop diuretic with salt tablets; **demeclocycline (demethylchlortetracycline) may be needed in resistant patients — it induces a partial nephrogenic diabetes insipidus and thus corrects the hyponatraemia, and serum creatinine and blood urea must be closely monitored**; and **vaptans (tolvaptan)** with strict inpatient sodium monitoring.

Feature	SIADH	Cerebral salt wasting	Adrenal insufficiency
Volume status	**Euvolaemic**	**Hypovolaemic**	Hypovolaemic
Urine sodium	>30–40 mEq/L	Very high	High
Urine output	Low / normal	**High (polyuria)**	Variable
Serum uric acid	**Low**	Low	Normal / high
Serum potassium	Normal	Normal / low	**High**
Response to 0.9% saline	**Worsens or no change**	**Improves**	Improves
Treatment	Fluid restriction	**Salt and fluid replacement**	Hydrocortisone

LATEST UPDATES (2023–PRESENT) — 3 POINTS

15. **Empagliflozin is emerging as a practical new option for chronic SIAD.** In a **randomised, double-blind, placebo-controlled crossover trial in outpatients with chronic SIAD (*JASN* 2023)**, four weeks of **empagliflozin 25 mg** added to fluid restriction of about 1500 mL/day **raised median serum sodium by 4.1 mEq/L versus placebo**, an effect achieved by the end of the first week and sustained thereafter. It was **well tolerated with no hypoglycaemia, hypotension, overcorrection or acute kidney injury**, and produced a small improvement in neurocognitive function, particularly when normonatraemia was restored. This followed an earlier inpatient randomised trial in 88 hospitalised patients (*JASN* 2020). The mechanism is **glucosuria-induced osmotic diuresis with increased electrolyte-free water excretion**. Its appeal in older adults is that it is **oral, inexpensive, and does not require the intensive monitoring or carry the overcorrection risk of vaptans**.
16. **Fluid restriction fails more often than is taught — predict failure and escalate early.** The recognised predictors of failure are **urine osmolality >500 mOsm/kg**, a **urine (Na + K) to serum Na ratio ≥1 (the Furst ratio)**, and a **24-hour urine volume <1500 mL**. Current practice is to identify these at the outset and **move to second-line therapy rather than persist with an ineffective restriction** — which in a frail older person also causes thirst, distress, constipation and delirium. Among second-line agents, **oral urea is increasingly favoured over vaptans** on grounds of cost, safety and the absence of overcorrection risk; palatability is the limiting factor.
17. **In geriatric practice, deprescribing is the definitive treatment.** The commonest reversible cause of SIADH in an older patient is **a drug — the SSRI, thiazide, carbamazepine or PPI**. A structured medication review using **STOPP/START and the AGS Beers criteria**, followed by stopping or substituting the culprit, corrects more cases than any pharmacological manoeuvre. The corresponding preventive standard is to **recheck serum sodium at 2 and 4 weeks after starting or changing any of these drugs in a person over 65** — a simple intervention that prevents a substantial proportion of hyponatraemic admissions.

CURRENT PRACTICE UPDATE

Aspect	Textbook	Current recommendation
Correction rate	Up to 20 mEq/L over 48 h (1 mEq/L/h)	≤8–10 mEq/L in 24 h; ≤6–8 in high-risk; re-lower if exceeded
Resistant SIADH	Demeclocycline	Oral urea, tolvaptan, or empagliflozin; demeclocycline now rarely used (nephrotoxicity, photosensitivity)
Emergency treatment	IV furosemide + fluid restriction + saline with potassium	3% saline 100–150 mL boluses targeting a 4–6 mEq/L rise
Terminology	SIADH / central pontine myelinolysis	SIAD / osmotic demyelination syndrome

Exam approach: Give the textbook protocol first — it is what the paper is set from — then add the modern correction cap and the second-line ladder. Naming **osmotic demyelination syndrome** and **empagliflozin** in the same answer signals current reading.

MEMORY ANCHORS & ESSAY SKELETON

Skeleton (20 marks, five blocks): (1) Define and give the four textbook features + Bartter–Schwartz criteria + supplemental criteria. (2) Pathophysiology — ADH → aquaporin-2 → volume expansion → secondary natriuresis → euvolaemic hyponatraemia, with vasopressin escape. (3) Causes in four groups — CNS, pulmonary, malignancy, drugs — plus pain/surgery and idiopathic SIAD of the elderly. (4) Clinical features acute versus chronic, and the differential (hypovolaemia, thyroid, adrenal, cerebral salt wasting). (5) Treatment ladder and correction rate. Close with the three updates.

Mnemonic for causes — "CNS · Chest · Cancer · Chemicals": the four Cs, then add pain/surgery and idiopathic.

Numbers: urine osm **>100** · urine Na **>30–40** · serum osm **<275** · uric acid **<4 mg/dL** · FEurate **>12%** · acute measures below Na **125** · fluid restriction **500–1000 mL** · urea **15–30 g/day** · empagliflozin **25 mg**, +**4.1 mEq/L** · cap **8–10 per 24 h**.

EXAMINER TRAPS

- Diagnosing SIADH **without documenting euvolaemia and without excluding hypothyroidism, adrenal insufficiency and diuretics**. State the exclusions explicitly — they carry marks.
- Giving **0.9% saline in SIADH**: when the urine osmolality exceeds that of the infusate, the salt is excreted and the free water is retained, so **the sodium falls further**.
- Confusing SIADH with **cerebral salt wasting** after a stroke — the entire treatment reverses. Volume status and urine output separate them.
- Forgetting **oedema is absent** in SIADH because of vasopressin escape.
- Omitting **drug causes** — in geriatric practice they are the majority, and the SSRI is the single commonest.

QUESTION 6 · FLUID, ELECTROLYTE & ACID–BASE

Hypokalaemia and hypomagnesaemia

7-Mark Answer — 6 textbook points + 1 latest update = 7 points

TEXTBOOK CORE — 6 POINTS

1. **Definitions and the ageing background. Hypokalaemia = serum K^+ <3.5 mEq/L** (severe <2.5); **hypomagnesaemia = serum Mg^{2+} <1.7 mg/dL. Total body potassium is lower in healthy older people**, and the potassium-controlling ATPases of the collecting tubule are impaired by ageing — so a given loss produces greater depletion and slower repletion than in the young.
2. **Causes of hypokalaemia in the elderly. Diuretics (loop and thiazide) are by far the commonest**; also GI losses (diarrhoea, vomiting, **laxative abuse**), poor dietary intake and malnutrition, primary or secondary hyperaldosteronism, and drugs — corticosteroids, insulin, **β_2-agonists**, amphotericin B. **Magnesium depletion** is an under-recognised cause.
3. **Causes of hypomagnesaemia in the elderly. Chronic proton-pump inhibitor use** (the classic geriatric cause), diuretics, alcohol excess, chronic diarrhoea and malabsorption, **aminoglycosides, cisplatin, amphotericin, calcineurin inhibitors**, and poorly controlled diabetes with osmotic diuresis.
4. **Clinical features.** Hypokalaemia — muscle weakness, cramps, **constipation and ileus**, polyuria (nephrogenic DI), rhabdomyolysis, and ECG changes: **flattened T waves, ST depression, prominent U waves, prolonged QU, ventricular ectopy**. Hypomagnesaemia — tremor, **tetany with positive Trousseau and Chvostek signs**, seizures, and **torsades de pointes**. In older adults, both frequently present **atypically as falls, generalised weakness or delirium**.

5. **The critical link — correct magnesium first.** Magnesium depletion causes **refractory hypokalaemia and refractory hypocalcaemia**: low intracellular magnesium **releases the inhibition of the ROMK channel** in the distal nephron, so potassium is wasted in the urine and **cannot be corrected until magnesium is replaced**. Hypomagnesaemia also impairs PTH secretion and end-organ response.
6. **Management.** Replace **magnesium first** — oral magnesium salts if mild; **IV magnesium sulphate 1–2 g** for symptomatic disease or torsades. Then potassium: **oral KCl is preferred** whenever the patient can take it; **IV KCl only for severe or symptomatic hypokalaemia**, at a maximum of **10 mEq/h peripherally (up to 20 mEq/h centrally with cardiac monitoring)**, and **never in dextrose** — dextrose stimulates insulin and drives potassium intracellularly, worsening the deficit. Finally, **review and deprescribe the culprit diuretic, laxative or PPI**.

LATEST UPDATE (2023–PRESENT) — 1 POINT

7. **Deprescribing, not escalating replacement, is the modern answer to drug-induced hypomagnesaemia.** Chronic PPI use is now firmly established as a cause of **hypomagnesaemia and of acute interstitial nephritis**, and PPIs have accordingly become a **major target of deprescribing strategies in older adults**. Current guidance in unexplained or recurrent hypomagnesaemia is to **stop the PPI** rather than keep increasing magnesium supplements. Note also that **SGLT2 inhibitors consistently raise serum magnesium**, an incidental benefit in the diabetic older patient on a diuretic.

EXAM ONE-LINERS

- **You cannot correct potassium until you correct magnesium** — the single highest-yield line in this answer.
- U waves = hypokalaemia; torsades = hypomagnesaemia.
- Never give IV potassium in dextrose; never exceed 10 mEq/h peripherally.
- Elderly + unexplained hypomagnesaemia = **look at the PPI**.
- Falls and delirium may be the **only** presenting features of severe electrolyte disturbance in the very old.

QUESTION 7 · POTASSIUM

A known diabetic on oral hypoglycaemic agents attends for routine review; serum potassium is 5.7 mEq/L. Investigation and treatment

7-Mark Note — 6 textbook points + 1 latest update = 7 points

TEXTBOOK CORE — 6 POINTS

1. **First — confirm the value and assess urgency simultaneously.** Exclude **pseudohyperkalaemia** by repeating with a properly taken sample: causes are a **tight or prolonged tourniquet, fist clenching, haemolysis, difficult venepuncture, delayed transport or prolonged storage**, and marked **thrombocytosis or leucocytosis**. At the same time perform a **12-lead ECG** — the presence of ECG change converts this from an outpatient problem into an emergency, and its absence permits an unhurried outpatient approach.
2. **Understand why this particular patient is at risk — the geriatric-diabetic substrate. Renal excretion of potassium is significantly lower in the aged**: there is **reduced serum aldosterone and a reduced tubular response to aldosterone**, together with **increased H^+/K^+-ATPase activity (potassium reabsorption) in the intercalated cells of the aged collecting duct**. Consequently **elderly people are predisposed to hyperkalaemia, particularly when treated with NSAIDs, ACE inhibitors, beta blockers and/or potassium-sparing diuretics**. In a diabetic, add **type 4 renal tubular acidosis (hyporeninaemic hypoaldosteronism)**, **diabetic nephropathy**, and **insulin deficiency**, since insulin drives potassium into cells.

3. **Take a targeted drug and diet history — the highest-yield step.** Drugs: **ACE inhibitors, ARBs, spironolactone or eplerenone, amiloride, beta blockers, NSAIDs, co-trimoxazole (trimethoprim acts like amiloride), heparin, digoxin, calcineurin inhibitors**. Diet and over-the-counter items: **"low-sodium" salt substitutes, which are potassium chloride** — a very common and frequently missed cause in Indian practice — plus **tender coconut water, banana, citrus, tomato, potato and dry fruits**. Also ask about **constipation** (the colon is an important route of potassium excretion in CKD) and about traditional or ayurvedic preparations.
4. **Investigate systematically.** Repeat serum potassium on a clean sample; **ECG**; **urea, creatinine and eGFR**; sodium; **serum bicarbonate** — a normal anion gap acidosis with hyperkalaemia points to **type 4 RTA**; blood glucose and HbA1c; **urine albumin–creatinine ratio**; calcium and magnesium; full blood count and platelet count (for pseudohyperkalaemia); creatine kinase if rhabdomyolysis is possible; and **9 a.m. cortisol with a short Synacthen test** if adrenal insufficiency is plausible. Where the cause remains unclear, a **urine potassium or transtubular potassium gradient** distinguishes impaired renal excretion from a transcellular shift.
5. **Know the ECG sequence and its limitations.** In order: **tall peaked T waves → flattening and loss of P waves → PR prolongation → widening of the QRS → sine wave → ventricular fibrillation or asystole**. Critically, **ECG changes correlate poorly with the potassium level in chronic hyperkalaemia** and may be entirely absent even at 6.5 mEq/L — so a normal ECG does not exclude danger, and a changing ECG demands action irrespective of the number.
6. **Treat this level — 5.7 mEq/L, asymptomatic, no ECG change — as moderate chronic hyperkalaemia, not an emergency.** This does **not** require calcium, insulin-dextrose or salbutamol. Do the following: **(a)** stop or reduce the culprit — **NSAIDs first, then reassess the potassium-sparing diuretic**, and **eliminate salt substitutes**; **(b)** dietary potassium restriction with dietitian input; **(c)** correct **metabolic acidosis with oral sodium bicarbonate if bicarbonate is below 22 mEq/L**; **(d)** improve glycaemic control; **(e)** add or increase a **loop or thiazide diuretic** if the patient is volume-replete; **(f)** treat constipation; **(g) recheck potassium in 3–7 days**. Reserve **emergency treatment for K^+ ≥6.5 mEq/L or any ECG change**: **10% calcium gluconate 10 mL IV** for membrane stabilisation (repeat as needed; it does not lower potassium), **10 units soluble insulin with 25% dextrose 50–100 mL**, **nebulised salbutamol 10–20 mg**, sodium bicarbonate if acidotic, and **dialysis if refractory or if renal failure is advanced**.

LATEST UPDATE (2023–PRESENT) — 1 POINT

7. **Potassium binders now allow prognostically important RAS blockade to be continued — a change of doctrine directly relevant to this patient.** The **KDIGO 2024 CKD guideline** states that in hyperkalaemia, **potassium binders may help facilitate essential use of RAS inhibitors and mineralocorticoid receptor antagonists**, that **potassium binders should be considered before reducing the RAS inhibitor**, and that **RASi discontinuation should be the last resort**. The modern agents are **sodium zirconium cyclosilicate (SZC)** and **patiromer**, both licensed for adults and both effective for chronic control and prevention of recurrence; the **UK Kidney Association 2023 guideline** broadened their use to **moderate as well as severe hyperkalaemia** and **removed calcium resonium (sodium polystyrene sulphonate) from the acute algorithm** because of weak evidence and the risk of colonic necrosis. For this diabetic patient, whose ACE inhibitor or ARB is renoprotective, the correct modern reasoning is therefore to **remove the avoidable contributors first (NSAID, salt substitute, dietary potassium), then add a binder — and only then, if all else fails, reduce the RAS blocker**.

CURRENT PRACTICE UPDATE

Aspect	Textbook	Current recommendation
Response to hyperkalaemia on a RASi	Reduce or stop the ACE inhibitor	Consider a potassium binder first; discontinuation is the last resort
Cation-exchange resin	Calcium / sodium polystyrene sulphonate	Replaced by SZC or patiromer; resin removed from acute guidance
Threshold for binder use	Severe hyperkalaemia only	Moderate as well as severe, when RASi preservation matters

Exam approach: This is a clinical-scenario question, so **structure the answer as confirm → why this patient → history and drugs → investigations → ECG → graded treatment**. Say explicitly that 5.7 mEq/L with a normal ECG is *not* an emergency — candidates who reflexively write "calcium gluconate, insulin-dextrose" lose marks for misjudging severity.

MEMORY ANCHORS

Causes mnemonic — "MACHINE": Medications (ACEi/ARB, spironolactone, NSAIDs, trimethoprim, beta blockers) · **A**cidosis · **C**ellular destruction (rhabdomyolysis, haemolysis, tumour lysis) · **H**ypoaldosteronism (type 4 RTA, Addison's) · **I**ntake (salt substitutes, diet) · **N**ephrons (renal failure) · **E**xcretion impaired.

Numbers: mild 5.5–5.9 · moderate 6.0–6.4 · **severe ≥6.5** or any ECG change · calcium gluconate 10% 10 mL · insulin 10 units + 25% dextrose 50–100 mL · salbutamol 10–20 mg nebulised · bicarbonate target ≥22 mEq/L · recheck in **3–7 days**.

EXAMINER TRAPS

- Treating 5.7 mEq/L as a crash call. **Judge severity by symptoms and ECG, not by the number alone.**
- Forgetting to **repeat the sample** — pseudohyperkalaemia is the commonest cause of a spuriously abnormal outpatient potassium.
- Missing the **salt substitute** — ask about it by name; patients do not consider it a medicine.
- Thinking **calcium gluconate lowers potassium**. It stabilises the myocardium and buys 30–60 minutes; nothing more.
- Stopping the ACE inhibitor reflexively and losing its renal and cardiac protection.
- Overlooking **type 4 RTA** — check the bicarbonate in every diabetic with hyperkalaemia.

QUESTION 8 · CALCIUM

Hypercalcaemia — causes, evaluation and management, including PTH-independent hypercalcaemia

7-Mark Note — 6 textbook points + 1 latest update = 7 points

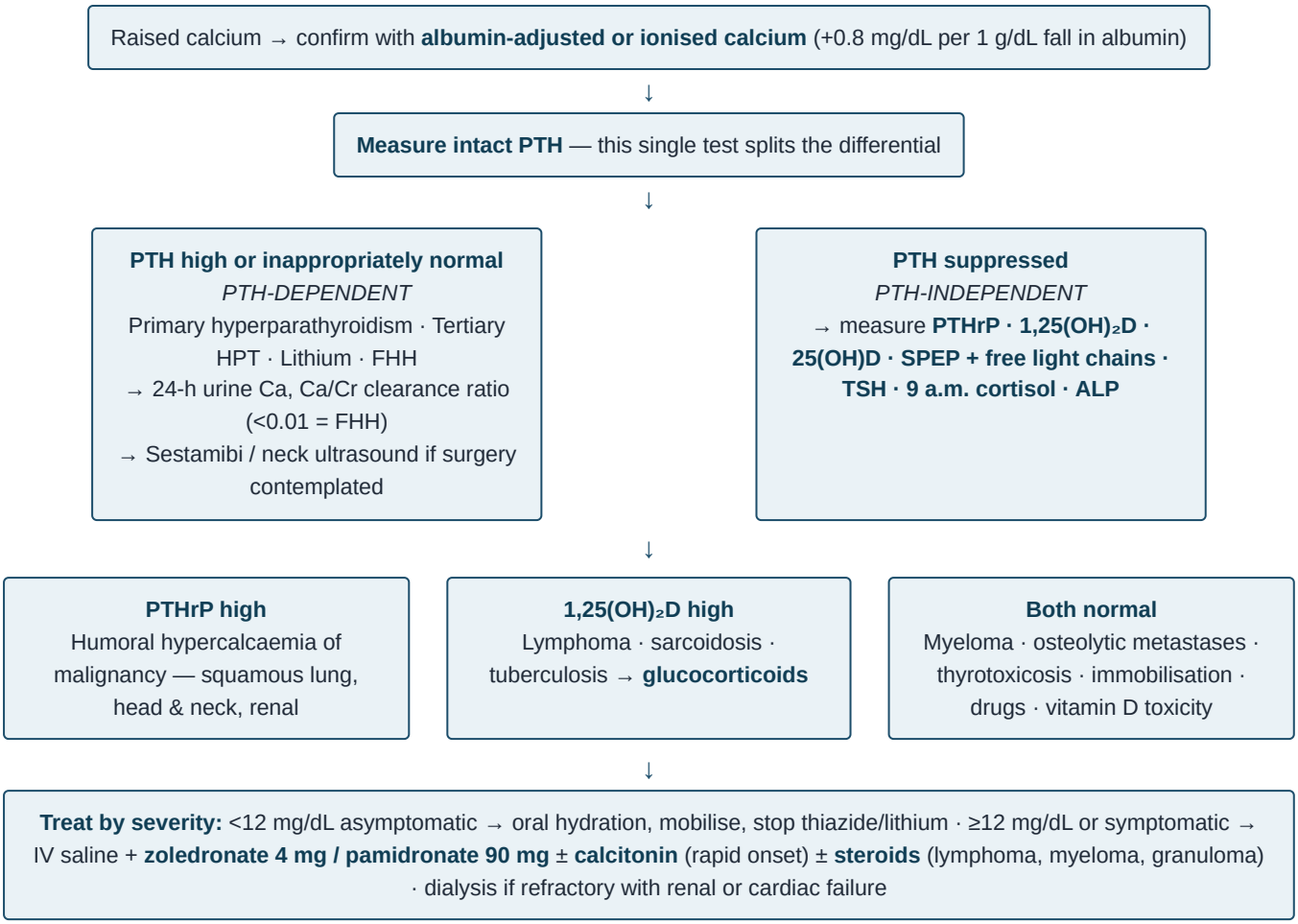
Recurring question — asked ×4. Extra scaffolding and full evaluation flow chart provided below.

TEXTBOOK CORE — 6 POINTS

1. **Definition and thresholds. Hypercalcaemia is defined by an elevation of calcium above 10.6 mg/dL, and values above 11.0 mg/dL should be considered an indication to initiate treatment.** This threshold applies **only when the albumin level is normal**. If there is any doubt about the validity of the total serum calcium,

measuring ionised calcium or calculating the albumin-adjusted calcium is essential. Correction: **add 0.8 mg/dL to the measured calcium for every 1 g/dL fall in albumin below 4 g/dL** — vital in the malnourished older patient, in whom true hypercalcaemia is otherwise missed.

2. **Two causes account for 90%. Primary hyperparathyroidism** — the commonest cause in **outpatients**, typically detected incidentally on routine biochemistry, and **PTH-dependent**. **Malignancy** — the commonest cause in **hospital inpatients**, usually with the cancer already overt, and **PTH-independent**. Other PTH-dependent causes: **tertiary hyperparathyroidism** in long-standing CKD, **lithium**, and **familial hypocalciuric hypercalcaemia**.
3. **PTH-independent causes — the part of the question most often under-answered. (a) Malignancy** by three mechanisms: **humoral hypercalcaemia via PTHrP** (squamous carcinoma of lung and head and neck, renal, bladder, ovarian), **osteolytic metastases and local cytokines** (breast cancer, **multiple myeloma**), and **excess 1,25-dihydroxyvitamin D production by lymphoma**. **(b) Granulomatous disease** with extrarenal 1α-hydroxylase activity — **sarcoidosis and tuberculosis** (the latter of major importance in India), plus fungal infection. **(c) Drugs** — **thiazides, lithium, vitamin D or vitamin A toxicity, calcium-alkali (milk–alkali) syndrome from over-the-counter antacids and supplements, teriparatide, and tamoxifen flare**. **(d) Endocrine** — **thyrotoxicosis**, adrenal insufficiency, phaeochromocytoma, acromegaly. **(e) Immobilisation** in a patient with high bone turnover — Paget's disease, myeloma, recent fracture — **highly relevant in the bed-bound older patient**. **(f)** Recovery phase of rhabdomyolysis-associated AKI, and parenteral nutrition.
4. **Clinical features and the geriatric presentation.** The classical formulation is **"bones, stones, abdominal groans and psychic moans"** — **bone** pain and fracture; **renal** stones, nephrocalcinosis and **nephrogenic diabetes insipidus with polyuria, dehydration and prerenal AKI**; **gastrointestinal** anorexia, nausea, vomiting, **constipation**, peptic ulceration and pancreatitis; **neuropsychiatric** fatigue, depression, confusion, stupor and coma. **In the older adult the presentation is typically delirium, constipation, dehydration and falls**, and it is routinely misattributed to dementia, infection or drugs. The **ECG shows a short QT interval**. **Serum calcium belongs in the confusion screen of every older patient.**
5. **Evaluation — one measurement decides the whole pathway.** Confirm with an **albumin-adjusted or ionised calcium**, then measure **intact PTH**. If PTH is **high or inappropriately normal**, the hypercalcaemia is **PTH-dependent** — primary or tertiary hyperparathyroidism, lithium, or familial hypocalciuric hypercalcaemia, which is distinguished by a **24-hour urinary calcium and a calcium/creatinine clearance ratio below 0.01**. If PTH is **suppressed**, the hypercalcaemia is **PTH-independent** — measure **PTHrP, 25(OH)D and $1,25(OH)_2D$, serum protein electrophoresis and free light chains, TSH, 9 a.m. cortisol, alkaline phosphatase**, and image for malignancy or granulomatous disease. Always also review every drug and supplement.
6. **Management by severity. Asymptomatic patients with minimally elevated calcium (below about 12 mg/dL) may be treated as outpatients**, with encouragement to adopt **oral hydration, mobilisation, and elimination of drugs contributing to hypercalcaemia — thiazides and lithium**. **Patients who are symptomatic, or whose calcium exceeds about 12 mg/dL, should be considered for inpatient management** using **volume expansion with saline infusion, corticosteroids and bisphosphonates**: **(i) Rehydration** generally has **mild and transient effects on calcium levels**; the volume of saline depends on the extent of hypovolaemia, with **particular care for the patient's cardiac and renal function**. **(ii) Bisphosphonates have supplanted all other drugs except calcitonin**, which remains useful in severe refractory hypercalcaemia because of its **rapid onset — 2 to 4 hours, versus 72 hours for bisphosphonates**. Recommended doses: **pamidronate 90 mg IV over 2–4 hours** or **zoledronate 4 mg IV over 15 minutes**; in patients with pre-existing renal disease **no change in dosage, infusion time or interval is required**. **(iii) Glucocorticoids** are helpful in hypercalcaemia caused by **lymphoma, myeloma and breast cancer**, and may be of some value in other malignancies, at **10–100 mg per day of prednisone equivalent**. Also: stop calcium and vitamin D supplements, mobilise the patient, **avoid loop diuretics unless the patient is volume-overloaded**, and consider **haemodialysis** in severe hypercalcaemia with renal or cardiac failure.



LATEST UPDATE (2023–PRESENT) — 1 POINT

7. **Endocrine Society Clinical Practice Guideline on the Treatment of Hypercalcaemia of Malignancy in Adults — the first evidence-based guideline in this area, co-sponsored by ASBMR and the European Society of Endocrinology.** Its key positions: **all adults with hypercalcaemia of malignancy should be treated with denosumab or an intravenous bisphosphonate** rather than left untreated; **denosumab is suggested in preference to an intravenous bisphosphonate as first-line therapy**; and **denosumab is specifically suggested for recurrent or refractory hypercalcaemia in patients previously treated with an intravenous bisphosphonate**. In **severe or symptomatic** disease, **calcitonin should be combined with the bisphosphonate or denosumab rather than the antiresorptive used alone**. For hypercalcaemia from tumours producing **high calcitriol, such as lymphoma**, in patients already on glucocorticoids but still severely hypercalcaemic, **add an IV bisphosphonate or denosumab**. In **parathyroid carcinoma**, use either a **calcimimetic (cinacalcet)** or an IV bisphosphonate/denosumab. **Two geriatric practice points:** denosumab requires **no renal dose adjustment and can be used at low eGFR** where zoledronate is problematic — a real advantage in the older patient with CKD — but it carries a genuine risk of **prolonged, severe hypocalcaemia in advanced CKD**, so calcium and vitamin D must be monitored, and its effect **cannot be reversed** once given.

CURRENT PRACTICE UPDATE

Aspect	Textbook	Current recommendation
First-line antiresorptive in malignancy	Bisphosphonates have supplanted all other drugs	Denosumab suggested *in preference to* IV bisphosphonate as first line
Refractory / recurrent disease	Calcitonin for rapid onset	Denosumab after prior IV bisphosphonate; calcitonin *combined* with the antiresorptive in severe disease
Renal impairment	No dose change needed for bisphosphonate	Denosumab preferred at low eGFR — but watch for prolonged hypocalcaemia

Exam approach: Reproduce the textbook doses (pamidronate 90 mg, zoledronate 4 mg, calcitonin onset 2–4 h versus 72 h) — they are frequently asked verbatim — then add the denosumab-first recommendation in a single sentence.

MEMORY ANCHORS & 15-SECOND SKELETON

Skeleton: Define with the albumin caveat → 90% = hyperparathyroidism (outpatient) or malignancy (inpatient) → list PTH-independent causes in six groups → bones/stones/groans/moans, but *delirium and falls in the elderly* → **correct for albumin, then measure PTH** → branch → treat by severity with saline, bisphosphonate, calcitonin, steroids.

PTH-independent mnemonic — "GRAND MIT": Granulomatous (sarcoid, TB) · **R**habdomyolysis recovery · **A**drenal insufficiency · **N**eoplasia (PTHrP, osteolytic, calcitriol) · **D**rugs (thiazide, lithium, vitamins A and D, milk–alkali) · **M**yeloma · **I**mmobilisation · **T**hyrotoxicosis.

Numbers: 10.6 mg/dL defines it · **11.0** mg/dL is the treatment threshold · **12** mg/dL separates outpatient from inpatient · albumin correction **+0.8 per 1 g/dL** · pamidronate **90 mg over 2–4 h** · zoledronate **4 mg over 15 min** · calcitonin onset **2–4 h** versus bisphosphonate **72 h** · prednisone **10–100 mg/day** · FHH Ca/Cr clearance ratio **<0.01**.

EXAMINER TRAPS

- Not **correcting for albumin** — in a hypoalbuminaemic older patient a "normal" total calcium can conceal significant hypercalcaemia.
- Ordering a scan before measuring **PTH**. PTH is the branch point of the entire flow chart, and the examiner is looking for it.
- Giving a **loop diuretic** to "promote calciuresis" in a dehydrated patient — this worsens hypovolaemia and hypercalcaemia. Rehydrate first; furosemide only for fluid overload.
- Forgetting that **rehydration alone has only mild and transient effects** — the definitive agent is the antiresorptive.
- Omitting **tuberculosis** from the granulomatous causes in an Indian examination.
- Missing **immobilisation hypercalcaemia** in a bed-bound patient with Paget's disease or myeloma.

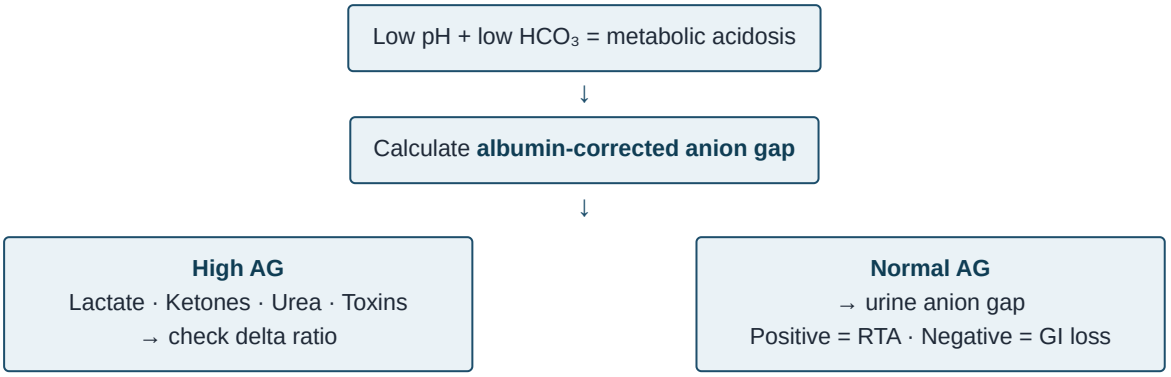
QUESTION 9 · FLUID, ELECTROLYTE & ACID–BASE

Metabolic acidosis and its evaluation

7-Mark Answer — 6 textbook points + 1 latest update = 7 points

TEXTBOOK CORE — 6 POINTS

1. **Definition and the ageing kidney.** Metabolic acidosis is a **primary fall in serum bicarbonate (<22 mEq/L)** with a low arterial pH (<7.35). The **ageing kidney handles an acute acid load poorly** — older people acidify the urine more slowly and take longer to recover, so acidosis is both commoner and more prolonged in old age.
2. **First step — the anion gap. AG = Na^+ – (Cl^- + HCO_3^-)**, normal **8–12 mEq/L**. Always **correct for albumin**: add 2.5 mEq/L to the measured gap for every 1 g/dL fall in serum albumin below 4 g/dL — otherwise a high-gap acidosis is missed in the malnourished older patient.
3. **High anion gap causes (GOLD MARK): G**lycols (ethylene glycol, propylene glycol), **O**xoproline (chronic paracetamol), **L**-lactate (sepsis, shock, **metformin**), **D**-lactate, **M**ethanol, **A**spirin, **R**enal failure (uraemia), **K**etoacidosis (diabetic, alcoholic, starvation).
4. **Normal anion gap (hyperchloraemic) causes: diarrhoea, renal tubular acidosis** — of which **type 4 (hyporeninaemic hypoaldosteronism) is the classic one in the older diabetic with CKD** — ureteric diversion, excessive saline, and drugs (**acetazolamide, spironolactone, trimethoprim, amiloride**).
5. **Stepwise evaluation.** ABG/VBG with simultaneous electrolytes → confirm the acidosis → calculate **albumin-corrected anion gap** → if high, calculate the **delta ratio** ($\Delta AG/\Delta HCO_3$) to detect a mixed disorder → if normal, calculate the **urine anion gap** ($U_{Na} + U_K - U_{Cl}$): a **positive** gap means **RTA**, a **negative** gap means **GI bicarbonate loss** → finally check respiratory compensation with **Winter's formula** (expected $pCO_2 = 1.5 \times HCO_3 + 8 \pm 2$).
6. **Management.** Treat the underlying cause — fluids and antibiotics for sepsis, insulin and fluids for ketoacidosis, dialysis for severe uraemia or toxic alcohols. **Sodium bicarbonate** is reserved for severe acidaemia (pH <7.1) or chronic CKD acidosis. In older adults, remember that bicarbonate carries a **sodium load that can precipitate volume overload and heart failure**, and rapid correction can cause hypokalaemia and hypocalcaemia.



LATEST UPDATE (2023–PRESENT) — 1 POINT

7. **Treating chronic metabolic acidosis of CKD is now standard renoprotective care.** Current KDIGO-aligned practice is to give **oral sodium bicarbonate to maintain serum bicarbonate ≥22 mEq/L** in CKD, because chronic acidosis accelerates GFR decline and drives **muscle protein catabolism, sarcopenia and bone demineralisation** — all of which are especially damaging in the frail older patient. The hydrochloric-acid–binding polymer **veverimer**, developed as a sodium-free alternative, **did not meet its primary endpoint in the VALOR-CKD trial**, so **sodium bicarbonate remains the treatment of choice**, with careful attention to volume status and blood pressure in older adults.

MEMORY ANCHORS

GOLD MARK for high-gap · **USED CARP** for normal-gap (Ureteric diversion, Saline, Endocrine/Addison, Diarrhoea, Carbonic anhydrase inhibitors, Ammonium chloride, RTA, Pancreatic fistula) · **Winter's = 1.5 × HCO_3 + 8 ± 2** · albumin correction **2.5 per 1 g/dL**.

EXAMINER TRAPS

- Forgetting to **correct the anion gap for albumin** in a malnourished 80-year-old — the commonest reason a high-gap acidosis is called "normal gap".
- Calling type 4 RTA rare — it is **common** in older diabetics on ACE inhibitors, spironolactone or trimethoprim, and presents as **hyperkalaemia with a normal-gap acidosis**.
- Giving bicarbonate for lactic acidosis of sepsis — treat the sepsis, not the number.

PART III

Kidney Disease

Q10 · Q11 · Q12 · Q13 · Q14 · Q15 · Q16 · Q17

QUESTION 10 · KIDNEY DISEASE

Management of acute kidney injury

7-Mark Note — 6 textbook points + 1 latest update = 7 points

Recurring question — asked ×2. Extra scaffolding provided below.

TEXTBOOK CORE — 6 POINTS

1. **Recognise and stage first.** AKI is defined by a rise in serum creatinine of **≥0.3 mg/dL within 48 hours**, or **≥1.5 times baseline within 7 days**, or urine output **<0.5 mL/kg/h for 6 hours**. In older adults the incidence is higher because of systemic illness, polypharmacy and the renal ageing process itself, and it is **frequently multifactorial**.
2. **Classify the cause, because two of the three categories are reversible. Prerenal — dehydration is the main cause in the elderly**, plus haemorrhage and cardiogenic or septic shock. **Renal** — acute tubular necrosis from persistence of prerenal causes and/or **nephrotoxins such as aminoglycosides**, rapidly progressive glomerular damage, and acute interstitial nephritis. **Postrenal** — stones, tumour, stricture and **prostatic hypertrophy**. **Prerenal and postrenal causes are of particular importance because their early identification and treatment may prevent or reverse the injury.**
3. **Immediate bedside assessment.** Assess volume status; **catheterise or bladder-scan to exclude retention**; send urinalysis and microscopy for casts and cells; arrange **renal ultrasound within 24 hours if obstruction is possible**; and **review every single drug**, prescribed and over-the-counter.
4. **The five therapeutic priorities. (i) Restore renal perfusion** with judicious fluid, guided by clinical reassessment — but give it cautiously, because **senile hypofiltration and diastolic cardiac failure predispose older people to cardiac failure and lung congestion after a saline load**. **(ii) Relieve obstruction** promptly. **(iii) Stop nephrotoxins** — NSAIDs, ACE inhibitors and ARBs, diuretics, aminoglycosides, contrast, PPIs. **(iv) Treat sepsis** with appropriately dosed antibiotics. **(v) Correct the consequences** — hyperkalaemia, acidosis, fluid overload — and **re-dose every drug to the current GFR**.
5. **Interpret the urinary indices with geriatric caution.** The urinary indices used for diagnosing AKI — **urinary sodium, fractional excretion of sodium and fractional excretion of urea — may be slightly different in older adults from those accepted for younger people**, because the aged kidney wastes sodium and urea at baseline. A higher-than-expected value therefore **does not exclude prerenal AKI**. Importantly, **age per se is not an important determinant of survival in AKI**.
6. **Dialysis, recovery and prevention. Temporary or permanent dialysis may sometimes be needed.** Although AKI is treated similarly in older and younger patients, older patients are **more vulnerable to dialysis-related complications** — haemodynamic instability, bleeding and disequilibrium syndrome. **If the kidney does not**

recover after approximately three months, the condition remains as chronic kidney disease. Because treatment options are limited, **prevention is paramount**: maintain adequate extracellular volume, avoid nephrotoxins, and **tailor drug dosage to the GFR**.

LATEST UPDATE (2023–PRESENT) — 1 POINT

7. **There is still no drug that treats AKI — but three practice points have changed. (a) Fluid choice: balanced crystalloids are preferred to 0.9% saline**, which causes hyperchloraemic acidosis; and there is **no role for low-dose dopamine, mannitol, or loop diuretics to "convert" oliguric to non-oliguric AKI** — diuretics are for volume overload only. **(b) Timing of dialysis:** the **STARRT-AKI trial** showed that **accelerated initiation of renal replacement therapy gave no mortality benefit and left more patients dialysis-dependent at 90 days**, supporting a watchful, **indication-driven** rather than number-driven strategy. **(c) Systems of care:** electronic **AKI alerts**, **nephrotoxin stewardship**, and **structured post-AKI follow-up** — because survivors of AKI carry a markedly elevated risk of subsequent CKD, recurrent AKI and cardiovascular death, and older survivors are the group least likely to be followed up.

MEMORY ANCHORS

Management mnemonic — "STOP AKI": Sepsis treated · **T**oxins stopped · **O**ptimise perfusion and blood pressure · **P**revent and relieve obstruction · then **A**ssess for **K**idney replacement **I**ndications.

Numbers: creatinine +0.3 mg/dL in 48 h · 1.5× baseline in 7 days · urine output <0.5 mL/kg/h × 6 h · ultrasound within 24 h · no recovery by **3 months** = CKD.

EXAMINER TRAPS

- Forgetting the **bladder**. In an older man, palpate and scan before you do anything clever.
- Using **FENa mechanically** in an 85-year-old — the ageing kidney wastes sodium at baseline.
- Giving furosemide to "improve urine output" — it improves the chart, not the kidney.
- Fluid-loading a frail patient with diastolic dysfunction into pulmonary oedema.
- Discharging without a **post-AKI follow-up plan** or restarting the ACE inhibitor without a repeat creatinine and potassium.

QUESTION 11 · KIDNEY DISEASE

Chronic kidney disease — etiopathogenesis, diagnosis and management, with conservative management in the elderly

15-Mark Essay — 10 textbook points + 2 latest updates = 12 points

Recurring question — asked ×2. Extra scaffolding provided below.

TEXTBOOK CORE — 10 POINTS

1. **Definition and burden.** CKD is a syndrome characterised by **progressive and generally irreversible deterioration of renal function due to a reduction in nephron mass**, present for more than three months with health implications. It is **predominantly a disease of older adults** — its incidence rises steadily with age and it is **at least ten times more common at 75 years and above than between 15 and 45 years**.
2. **Causes in old age.** The leading causes are **hypertension and nephrosclerosis, diabetes mellitus, and obstructive uropathy**. Two causes deserve special emphasis because they are potentially reversible: **vascular disease of the main renal arteries** and **prostatic hypertrophy**. In as many as **one-third of older patients no**

specific cause can be identified. Also consider amyloidosis, myeloma and chronic interstitial nephritis from long-term NSAID use.

3. **Etiopathogenesis — the mechanisms of progression. (a) Glomerular hyperfiltration** in surviving nephrons — initially compensatory but ultimately **accelerating glomerular sclerosis**. **(b) Retention of uraemic toxins** — polyamines, guanidines, middle molecules and hormonal peptides. **(c)** A **high parathyroid hormone level is itself a significant uraemic toxin**. **(d) Deficiency of erythropoietin and 1,25-dihydroxycholecalciferol** → anaemia and hypocalcaemia. **(e) Phosphate retention** → secondary hyperparathyroidism and **renal osteodystrophy**. **(f) Derangement of the renin–angiotensin–aldosterone system** → hypertension, which in turn accelerates nephron loss.
4. **Distinguish CKD from senile hypofiltration — a uniquely geriatric step.** In older adults, **senile hypofiltration differs from CKD** because the glomerular filtration value is **stable over 6–12 months**, and there is **no haematuria and no significant proteinuria (>0.30 g/day)**. Labelling every low eGFR in an 85-year-old as "CKD" causes unnecessary alarm, referral and treatment.
5. **Clinical features.** When creatinine clearance falls to about **25 mL/min** the full picture of uraemia appears: a **yellow-brown pallor**, pruritus, asthenia, anorexia and vomiting; cardiovascular — hypertension, heart failure, pulmonary oedema, coronary disease, arrhythmias and pericarditis; neurological — **polyneuropathy, clonus, asterixis and uraemic coma**; endocrine — **secondary hyperparathyroidism and hyperprolactinaemia**; plus **impaired cellular immunity** and **clotting abnormalities** from platelet dysfunction.
6. **Diagnosis and staging.** Stage with **eGFR (G1–G5) and albuminuria (A1–A3)**. Investigations: urinalysis and sediment, **urine albumin–creatinine ratio**, serum creatinine and electrolytes, bicarbonate, calcium, phosphate, **intact PTH**, haemoglobin and iron studies, and **renal ultrasound** for kidney size, symmetry and obstruction. Always **exclude reversible contributors first** — obstruction, volume depletion, infection, hypercalcaemia and nephrotoxic drugs.
7. **Management — slowing progression. Blood pressure control**, **RAS blockade with a single agent** for proteinuric disease (accept a creatinine rise of up to 30%; monitor potassium), **glycaemic control** individualised to frailty, **statin therapy**, smoking cessation, moderate protein intake with adequate calories to prevent sarcopenia, and **avoidance of nephrotoxins** — NSAIDs, aminoglycosides, iodinated contrast, and high-dose PPIs.
8. **Management — treating the complications. Anaemia** — iron repletion then an erythropoiesis-stimulating agent, targeting haemoglobin 10–11.5 g/dL, not normal. **CKD–mineral and bone disorder** — dietary phosphate restriction, phosphate binders, active vitamin D analogues, control of PTH. **Metabolic acidosis** — oral sodium bicarbonate to keep serum bicarbonate **≥22 mEq/L**, which also protects muscle mass. **Hyperkalaemia**, volume overload, vaccination, and **dose adjustment of every drug to the estimated GFR**.
9. **When to consider renal replacement.** Textbook threshold: when **GFR falls below 10 mL/min** it becomes necessary to start replacement therapy — dialysis or transplantation. **Age itself does not constitute a contraindication** to either. In practice the decision is **symptom-driven**, and access should be planned well in advance (see Question 12).
10. **Conservative kidney management (CKM) in the elderly — the second half of this question.** CKM is an **active, planned, non-dialytic pathway — not "no treatment"**. Its components are: **(i) symptom control** — pruritus, nausea, restless legs, breathlessness, fatigue and pain; **(ii) medical management** of anaemia, acidosis, fluid balance and blood pressure; **(iii) dietary modification** without inducing malnutrition; **(iv) advance care planning** and documentation of ceilings of care; **(v) psychosocial, spiritual and carer support**; and **(vi) integration with palliative care**. It is most appropriate where the predictors of poor dialysis outcome are present — **poor nutritional status, dementia, gait disorders, impaired ADLs and IADLs, and late referral**. The supporting figures: median survival on haemodialysis is **15.6 months at 80–84 years and 11.6 months at 85–89 years**, and **elective withdrawal accounts for 38% of deaths** in the oldest dialysis patients — evidence that for the frail, dependent, multimorbid very old, dialysis may add little survival while costing function and time at home.

LATEST UPDATES (2023–PRESENT) — 2 POINTS

11. **KDIGO 2024 CKD guideline — the shift from an eGFR-based to a risk-based approach.** Key changes: stage CKD preferably with the **race-free eGFRcr-cys equation** (creatinine plus cystatin C) where cystatin C is available, and use eGFRcr-cys whenever creatinine-based eGFR is likely to be inaccurate; in **CKD G3–G5, use an externally validated risk equation such as the Kidney Failure Risk Equation** to estimate absolute risk of kidney failure and guide referral; **SGLT2 inhibitors for adults with CKD regardless of diabetes** — eGFR ≥20 with ACR ≥200 mg/g, or heart failure irrespective of albuminuria (Level 1A), and suggested for eGFR 20–45 with ACR <200 mg/g; **non-steroidal MRA (finerenone)** for type 2 diabetes with eGFR >25, normal potassium and persistent albuminuria on maximally tolerated RASi; **statins for all adults over 50 with CKD G1–G5** not on dialysis; **urate-lowering therapy only if symptomatic**; and **point-of-care creatinine and albumin testing** where laboratory access is limited.
12. **Indian context.** India carries a heavy and partly distinct CKD burden, including **CKD of unknown aetiology (CKDu)** in agricultural belts of Andhra Pradesh, Odisha and Tamil Nadu, linked to heat stress, dehydration and agrochemical exposure. The **Pradhan Mantri National Dialysis Programme** under the National Health Mission provides free haemodialysis at district hospitals in public–private partnership mode, with an explicit policy push to expand **peritoneal dialysis**; **AB-PMJAY** and the **Ayushman Vay Vandana Card** (all citizens aged 70 and above) cover dialysis packages. Early detection depends on opportunistic screening for diabetes and hypertension through **NPCDCS** at health and wellness centres.

CURRENT PRACTICE UPDATE

Aspect	Textbook	Current recommendation
Staging tool	Creatinine-based eGFR	Race-free eGFRcr-cys preferred; KFRE for risk in G3–G5
Disease-modifying therapy	RAS blockade, BP and glycaemic control	Add SGLT2i for all CKD regardless of diabetes; finerenone in diabetic CKD
Non-dialytic pathway	Implicit / not named	Conservative kidney management is a named, structured, guideline-endorsed option

Exam approach: Give the textbook mechanisms of progression in full — that is the "etiopathogenesis" the question asks for — then compress diagnosis, and reserve real space for **conservative management**, which is the half of the question candidates routinely under-answer.

MEMORY ANCHORS & 15-SECOND SKELETON

Skeleton: Define → causes in the old (HTN, DM, obstruction; ⅓ unknown; renal artery disease and prostate are reversible) → six mechanisms of progression → *distinguish senile hypofiltration* → uraemia at CrCl 25 → stage by G and A → slow progression + treat complications → RRT below GFR 10 → **conservative kidney management in six components** with the survival figures.

Mnemonic for the six mechanisms — "HUP–PEP": **H**yperfiltration · **U**raemic toxins · **P**TH as a toxin · **P**hosphate retention · **E**PO and vitamin D deficiency · **P**ressure (RAAS/hypertension).

Numbers: 10× commoner at ≥75 · one-third no cause found · uraemia at CrCl 25 mL/min · RRT below GFR 10 · HCO_3 ≥22 mEq/L · Hb target 10–11.5 g/dL · HD median survival 15.6 and 11.6 months · withdrawal 38%.

EXAMINER TRAPS

- Writing a generic adult CKD answer with no **geriatric content** — the marks are in senile hypofiltration, reversible causes, and conservative management.
- Presenting CKM as "withdrawing care". State explicitly that it is an **active, planned pathway** with six named components.
- Missing **prostatic obstruction and renal artery disease** — the two potentially reversible causes the textbook singles out in the elderly.
- Targeting a **normal haemoglobin** with an ESA.

QUESTION 12 · KIDNEY DISEASE

Criteria for initiating renal replacement therapy, with special emphasis on the elderly in our country

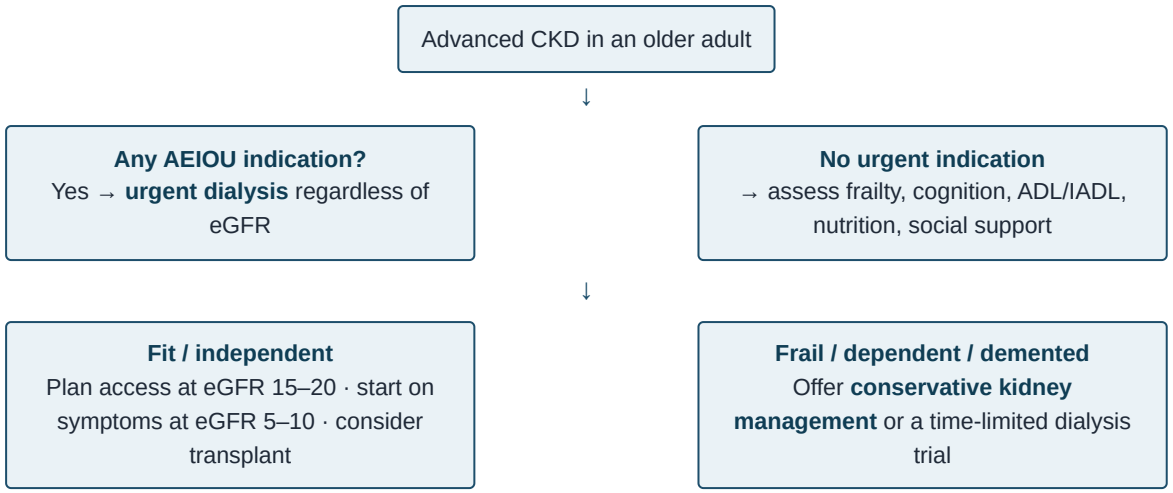
7-Mark Note — 6 textbook points + 1 latest update = 7 points

Recurring question — asked ×2. Extra scaffolding provided below.

TEXTBOOK CORE — 6 POINTS

1. **The textbook threshold.** As CKD progresses and creatinine clearance falls to about **25 mL/min** the full picture of uraemia appears; when the **GFR falls below 10 mL/min it is necessary to start replacement therapy** — dialysis or transplantation. **Age itself does not constitute a contraindication to dialysis and/or transplantation.**
2. **Absolute and urgent indications — mnemonic "AEIOU".** **A**cidosis, severe and refractory to bicarbonate. **E**lectrolytes — **refractory hyperkalaemia**. **I**ntoxications that are dialysable — lithium, salicylate, methanol, ethylene glycol, metformin. **O**verload — **refractory pulmonary oedema** unresponsive to diuretics. **U**raemic complications — **pericarditis, encephalopathy, neuropathy, intractable nausea and vomiting, and bleeding diathesis**. Any one of these mandates dialysis regardless of the eGFR number.
3. **Elective, planned initiation is symptom-driven, not number-driven.** The trigger is a cluster of **anorexia, nausea, weight loss, fatigue, declining functional status, uncontrolled fluid or blood pressure and progressive malnutrition**, typically at an eGFR of **5–10 mL/min/1.73 m^2**. Vascular access should be created well ahead — plan the **arteriovenous fistula at an eGFR of about 15–20**, as it needs weeks to mature and is harder to create in the elderly.
4. **Geriatric modifiers of the decision.** Predictors of a poor outcome on dialysis are **poor nutritional status, dementia, gait disorders, impaired ADLs and IADLs, and late nephrology referral**. Median survival on haemodialysis is **15.6 months at 80–84 years and 11.6 months at 85–89 years**; the leading causes of death are **elective withdrawal (38%)**, cardiovascular events (24%) and infection (22%). The survival gain must therefore be weighed against dialysis burden, travel, hospital time and loss of independence.
5. **Transplantation remains on the table.** Age alone does not exclude it: in the elderly, **one-year patient survival is about 85% and one-year graft survival about 75%**, with **five-year functional graft survival of 55–60% in those over 60**. Comorbidity, not the birth date, decides.
6. **Three pathways, chosen by shared decision-making.** Present **haemodialysis**, **peritoneal dialysis** (including assisted or automated forms for the more vulnerable, such as nursing-home residents) and **conservative kidney management** as genuine alternatives. Discuss prognosis honestly, undertake **advance care planning**, and where

uncertainty remains, offer a **time-limited trial of dialysis** with an agreed review point. Genuine barriers in the very old are advanced dementia with inability to cooperate, terminal malignancy, unmanageable haemodynamic instability, absence of a carer for peritoneal dialysis, and the patient's own refusal — **none of which is age**.



LATEST UPDATE (2023–PRESENT) — 1 POINT

7. **The Indian picture — access has improved, but the decision framework has changed more.** The **Pradhan Mantri National Dialysis Programme** under the National Health Mission provides **free haemodialysis at district hospitals in public–private partnership mode**, with an explicit policy push to expand **peritoneal dialysis** because it is home-based, needs no thrice-weekly transport and is haemodynamically gentler — all decisive advantages for the very old. **AB-PMJAY** covers dialysis packages, and the **Ayushman Vay Vandana Card** extends cover to **all citizens aged 70 and above irrespective of income**. Practical Indian barriers persist: distance and transport for thrice-weekly haemodialysis, caregiver availability for peritoneal dialysis, out-of-pocket cost of erythropoietin and drugs, and very low rates of transplantation in the elderly. Contemporary international practice — now reflected in Indian nephrology and geriatrics guidance — is to **start on symptoms rather than on an eGFR number**, and to present **conservative kidney management as an explicit third option**, supported where available by state palliative networks such as **Kerala's community palliative care programme**.

MEMORY ANCHORS

AEIOU for urgent dialysis · access at eGFR **15–20**, start at **5–10**, textbook threshold **<10** · uraemia appears at CrCl **25** · survival **15.6 / 11.6 months** · withdrawal **38%** · transplant **85% / 75%** at one year, **55–60%** graft survival at five years.

EXAMINER TRAPS

- Giving only a number. The examiner wants **AEIOU plus the symptom-driven elective criteria plus the geriatric modifiers**.
- Saying dialysis is contraindicated in the very old — say instead that **age is not a contraindication, but frailty and dependency predict poor outcome**.
- Omitting the **Indian delivery context** when the question explicitly asks about "our country".
- Forgetting to mention **timely access creation** — late referral is itself a predictor of poor outcome.

QUESTION 13 · NEPHROLOGY

Peritoneal dialysis versus haemodialysis in the very old

7-Mark Answer — 6 textbook points + 1 latest update = 7 points

TEXTBOOK CORE — 6 POINTS

1. **Age itself is not a contraindication to dialysis or transplantation.** Older patients can be treated effectively with **either haemodialysis (HD) or peritoneal dialysis (PD)**, and **most studies confirm that survival on PD and HD is similar** in the elderly. Replacement therapy is started when **GFR falls below 10 mL/min**.
2. **Practical problems with HD in the very old:** difficulty **creating an arteriovenous fistula**, permanent catheter or graft **infection**, gastrointestinal bleeding, **intradialytic hypotension**, headache, vomiting, cramps, angina, arrhythmias and **cardiovascular instability**.
3. **Practical problems with PD in the very old: dialysate leakage**, formation of **hernias**, backache, **difficulty in learning** the technique, **social isolation**, **family burn-out**, and worsening of **peripheral vascular disease or malnutrition**.
4. **When PD is the better choice.** PD is preferable in patients with **residual diuresis**, **severe hypotension**, **complicated or short-lived vascular access**, **intradialytic arrhythmias**, **angina**, or **cardiovascular instability**. Automated forms — **CCPD and nightly PD** — are alternatives for the more vulnerable elderly, including nursing-home residents.
5. **Nutrition and bone.** Malnutrition is frequent: provide **>1 g/kg/day protein on HD and >1.2 g/kg/day on PD**. Older dialysis patients develop **more severe bone disease** than the young because of osteopenia, unbalanced diet, reduced physical activity and lack of sun exposure.
6. **Prognosis and predictors of poor outcome.** Poor nutritional status, **dementia**, gait disorders, impaired **ADLs and IADLs**, and **late referral to nephrology**. Median survival on HD is **15.6 months at 80–84 years** and **11.6 months at 85–89 years**. Leading causes of death in the oldest dialysis patients: **elective withdrawal 38%**, cardiovascular events 24%, infection 22%.

Domain	Haemodialysis	Peritoneal dialysis
Haemodynamics	Intradialytic hypotension, arrhythmia, angina	Gentle, continuous — better in cardiac instability
Access	AV fistula often difficult; catheter infection	Peritoneal catheter; leaks, hernias
Residual renal function	Lost faster	Better preserved — favours PD
Burden	Thrice-weekly travel to centre	Home-based; needs learning ability and a carer
Typical failure mode	Vascular access failure	Peritonitis, technique failure, carer burn-out
Survival in the elderly	**Similar in most studies**	

LATEST UPDATE (2023–PRESENT) — 1 POINT

7. **Shared decision-making with conservative kidney management as a genuine third option.** Current international practice frames the choice not as "HD versus PD" but as **HD versus PD versus conservative kidney management (CKM)**, with modality decided by **frailty, cognition, functional status and what matters to the patient** rather than by age or eGFR alone — CKM may deliver comparable survival with better quality of life in the frail, dependent very old. In India, **assisted / home-based PD is being expanded under the national dialysis programme and AB-PMJAY**, because it avoids thrice-weekly travel, is haemodynamically gentler, and suits an older population living with family caregivers.

EXAM ONE-LINERS

- Survival on HD and PD in the elderly = **similar**. Choose by comorbidity and social situation, not by age.
- PD for the **cardiovascularly unstable**; HD for the **cognitively impaired without a carer**.
- Commonest cause of death in the oldest dialysis patients = **elective withdrawal (38%)**.
- Protein: **>1 g/kg HD, >1.2 g/kg PD**.
- Five-year graft survival after transplant in those >60 years = **55–60%**.

QUESTION 14 · KIDNEY DISEASE

Acute glomerulonephritis

7-Mark Note — 6 textbook points + 1 latest update = 7 points

TEXTBOOK CORE — 6 POINTS

1. **Definition — the acute nephritic syndrome.** Abrupt onset of **haematuria with dysmorphic red cells and red cell casts**, **proteinuria** (usually sub-nephrotic), **hypertension**, **oedema** and **oliguria**, with a rising creatinine. When crescents involve more than 50% of glomeruli and function is lost over days to weeks, it is termed **rapidly progressive glomerulonephritis (RPGN)**.
2. **Age-specific epidemiology — this is a disease of the old. Crescentic glomerulonephritis reaches its greatest incidence between 60 and 79 years** and presents as rapidly progressive acute renal failure. **Renal vasculitis is probably the most common primary cause of renal failure in older adults. Post-streptococcal and other post-infectious glomerulonephritis are more frequent in older than in younger patients** — in the elderly typically **staphylococcal-associated or endocarditis-related** rather than streptococcal.
3. **Classification by immunofluorescence — the framework the examiner wants. (a) Pauci-immune** — ANCA-associated vasculitis (granulomatosis with polyangiitis, microscopic polyangiitis); **commonest pattern in the elderly**. **(b) Immune-complex (granular)** — post-infectious, IgA nephropathy and Henoch–Schönlein purpura, lupus nephritis, cryoglobulinaemia (hepatitis C), membranoproliferative GN. **(c) Linear** — **anti-glomerular basement membrane disease**, which has a second incidence peak in older adults.
4. **Presentation in old age is non-specific and easily missed.** Malaise, weight loss, low-grade fever, arthralgia, anorexia, **normocytic anaemia**, cough or haemoptysis (**pulmonary–renal syndrome**), purpuric rash, mononeuritis multiplex, or scleritis. The renal failure is often **discovered incidentally on routine bloods** and is at real risk of being dismissed as "ageing", dehydration or prerenal AKI — losing treatable time in a condition where **days matter**.

5. **Investigations. Urine microscopy for dysmorphic red cells and red cell casts** is the single most useful bedside test. Then urine protein–creatinine ratio, creatinine, **ANCA (MPO and PR3)**, **anti-GBM**, ANA and anti-dsDNA, **C3 and C4**, ASO titre, blood cultures and echocardiography if endocarditis is possible, hepatitis B and C serology, cryoglobulins, and chest imaging. **Renal biopsy is the definitive investigation** — its complications are **no more frequent** in older adults, though they are more serious when they occur.
6. **Management.** Treat the underlying cause. For crescentic and vasculitic disease, **steroids and other immunosuppressive drugs such as cyclophosphamide can be used in older as in younger adults, although with special attention to their side effects**; **careful monitoring of therapy can minimise adverse effects in this population**. Add **plasma exchange** for anti-GBM disease and for severe pulmonary haemorrhage. Supportive care: blood pressure control, salt and fluid restriction, loop diuretics, correction of hyperkalaemia, and dialysis where required. **Post-infectious glomerulonephritis needs antibiotics and supportive care only**, not immunosuppression.

LATEST UPDATE (2023–PRESENT) — 1 POINT

7. **KDIGO ANCA-Associated Vasculitis guidance — rituximab and steroid sparing, both particularly valuable in the elderly.** For new-onset organ-threatening disease, induction is with **glucocorticoids plus either rituximab or cyclophosphamide**, with **rituximab preferred in older adults** and in anyone at risk of cyclophosphamide toxicity — infection, marrow suppression, malignancy and infertility. **Rituximab is also preferred for maintenance of remission**. Two further changes matter for geriatric practice: a **reduced-dose glucocorticoid taper (PEXIVAS-style)** achieves the same remission rates with significantly fewer serious infections; and the oral **C5a receptor antagonist avacopan** permits substantial steroid sparing — directly relevant in an older patient at risk of steroid-induced diabetes, osteoporosis, infection, delirium and myopathy. Prophylaxis against *Pneumocystis jirovecii* with co-trimoxazole is standard during induction.

EXAM ONE-LINERS

- Crescentic GN peaks at **60–79 years** — this is a geriatric disease, not a paediatric one.
- **Renal vasculitis is probably the commonest primary cause of renal failure in older adults.**
- Red cell casts = glomerular. Their presence changes the whole diagnostic pathway.
- Post-infectious GN in the elderly is usually **staphylococcal or endocarditis-related**, not streptococcal.
- **Low C3** = post-infectious, lupus, MPGN, cryoglobulinaemia. **Normal C3** = ANCA vasculitis, anti-GBM, IgA.
- Immunosuppression works just as well in the old — the difference is in **monitoring, not withholding**.

EXAMINER TRAP

Writing the classical **paediatric post-streptococcal** answer. In a geriatrics examination the expected emphasis is **ANCA-associated crescentic glomerulonephritis presenting as RPGN**, its non-specific systemic prodrome, and the fact that **it is treatable if biopsied early**. Lead with that.

QUESTION 15 · NEPHROLOGY

Nephrotic syndrome and membranous nephropathy in the elderly

15-Mark Answer — 10 textbook points + 2 latest updates = 12 points

TEXTBOOK CORE — 10 POINTS

1. **Definition.** Nephrotic syndrome is **proteinuria >3.5 g/24 h** with **hypoalbuminaemia (<3 g/dL)**, **oedema** and **hyperlipidaemia**. It accounts for about **50% of all renal biopsy indications** in older adults.
2. **Commonest causes in old age. Membranous nephropathy**, **minimal change disease**, **diabetic nephropathy** and **amyloidosis**. Among older patients biopsied for glomerulonephritis, 59% had primary and 20% secondary glomerulonephritis.
3. **Age-related shift in histology. Membranous nephropathy, membranoproliferative GN, minimal change disease, crescentic GN** and post-streptococcal GN are all **more frequent** in older than younger patients. Only **FSGS and IgA / non-IgA mesangioproliferative nephritis are less frequent**.
4. **Membranous nephropathy is often secondary in the elderly.** In about **20%** it is related to an **underlying malignancy** — mainly **lung or colon adenocarcinoma** — or to **drug ingestion**. A malignancy screen is mandatory before labelling it primary.
5. **Minimal change disease behaves atypically.** Senile structural renal changes make histology difficult; presentation is usually atypical with **hypertension, microhaematuria and/or renal failure**, and it may be drug-related (**NSAIDs**) or associated with **lymphoma**.
6. **Crescentic GN must not be missed.** Its incidence peaks between **60–79 years** and it presents as **rapidly progressive acute renal failure**. Renal vasculitis (granulomatosis with polyangiitis, microscopic polyangiitis) is probably the commonest primary cause of renal failure in older adults.
7. **Other secondary glomerulopathies frequent in the elderly:** diabetic nephropathy, **nephroangiosclerosis** from essential hypertension, glomerular vasculitis, and diseases with **abnormal plasma-cell proteins** — light chain, fibrillary and immunotactoid nephropathy.
8. **Investigations.** Urine protein-creatinine ratio, serum albumin, lipid profile, creatinine (mg/dL), **anti-PLA2R antibody**, blood glucose, **serum protein electrophoresis and free light chains**, ANA/ANCA, hepatitis B and C serology, and an age-appropriate malignancy screen.
9. **Supportive management.** Salt restriction, loop diuretics, **RAS blockade** for antiproteinuric effect, statin, and anticoagulation when albumin is very low — **membranous nephropathy carries the highest thromboembolic risk of the primary glomerular diseases**. Watch closely for AKI and hyperkalaemia.
10. **Immunosuppression. Steroids and cyclophosphamide can be used in older as in younger adults**, but with special attention to their side effects — infection, bone marrow suppression, hyperglycaemia, osteoporosis and delirium.

LATEST UPDATES (2023–PRESENT) — 2 POINTS

11. **KDIGO Glomerular Diseases guideline — biopsy-free diagnosis and rituximab first-line.** In nephrotic syndrome with a **positive anti-PLA2R antibody and preserved kidney function**, kidney biopsy is **not required** to diagnose membranous nephropathy. **Rituximab is first-line** for moderate- and high-risk disease (1 g × 2 doses two weeks apart, or 375 mg/m^2 weekly), while **cyclophosphamide + steroid remains preferred in the very-high-risk category**.
12. **Antibody-guided therapy and the elderly caveat.** Serial **anti-PLA2R titres at 3–6 months predict remission**; immunosuppression can be stopped once the antibody is undetectable at 6 months. Recent 2025 series support **low-dose / personalised rituximab** in frail older patients. Important caveat: in the elderly, membranous nephropathy may **co-exist with renal amyloidosis or diabetic nephropathy**, so anti-PLA2R positivity alone should not stop you from biopsying an atypical case.

CURRENT PRACTICE UPDATE

Aspect	Textbook	Current Recommendation
Diagnosis of MN	Renal biopsy in all cases	Biopsy may be omitted if anti-PLA2R positive with normal kidney function
First-line immunosuppression	Steroids ± cyclophosphamide	Rituximab first-line in moderate/high risk; cyclophosphamide reserved for very high risk
Treatment decision	Uniform, after 6 months of supportive care	Risk-stratified (proteinuria, eGFR trend, anti-PLA2R titre, epitope spreading)

Exam approach: Write the textbook histology and the malignancy association first, then add KDIGO risk stratification and rituximab as the "current practice" paragraph — this earns the highest marks.

EXAM ONE-LINERS

- Commonest cause of nephrotic syndrome in an older adult = **membranous nephropathy**.
- **1 in 5** elderly membranous nephropathy = **malignancy-associated** (lung, colon).
- Target antigen in ~70% of primary MN = **PLA2R1**; antibody is **IgG4** subclass.
- Highest thrombosis risk among primary glomerular diseases = **membranous nephropathy**.
- Nephrotic syndrome + rapidly rising creatinine in a 70-year-old = think **crescentic GN / vasculitis** — biopsy urgently.

QUESTION 16 · KIDNEY DISEASE

Interstitial nephritis

7-Mark Note — 6 textbook points + 1 latest update = 7 points

TEXTBOOK CORE — 6 POINTS

1. **Definition.** Inflammation of the **renal tubulointerstitium with relative sparing of glomeruli and vessels**. It presents either as **acute interstitial nephritis (AIN)** causing acute kidney injury, or as **chronic interstitial nephritis** causing slowly progressive CKD with tubular dysfunction.
2. **Mechanism and the classical drug causes.** Interstitial nephritis **results mainly from a delayed hypersensitivity response to drugs** — it is idiosyncratic, not dose-dependent. **Acute interstitial nephritis may develop following the use of nearly all NSAIDs or diuretics**, and although the number of reported cases is

small, **most of these patients are elderly**. Other established causes: beta-lactams, sulphonamides and co-trimoxazole, rifampicin, ciprofloxacin, allopurinol, mesalamine and phenytoin.

3. **Proton-pump inhibitors — the classic geriatric cause.** PPIs such as **omeprazole, esomeprazole and pantoprazole are increasingly recognised as a cause of acute interstitial nephritis**, and **the diagnosis requires a high index of suspicion**. It is partly for this reason that **PPIs have become a target of deprescribing strategies in older adults**. The latency is long — **weeks to months** — which is exactly why the connection is missed.
4. **Clinical features — usually silent, rarely classical.** The textbook triad of **fever, rash and eosinophilia is present in fewer than 10%** and is **even less common in the elderly**. Expect instead **non-oliguric AKI** with a bland or mildly abnormal urine: **sterile pyuria, white cell casts, mild sub-nephrotic proteinuria**, and **eosinophiluria** (poor sensitivity and specificity — do not rely on it). Tubular dysfunction may dominate: **Fanconi syndrome, renal tubular acidosis, hypokalaemia and hypomagnesaemia**.
5. **Chronic interstitial nephritis in older adults.** Patients taking **NSAIDs for months or years** may develop **papillary necrosis, chronic interstitial nephritis or even end-stage renal disease**. Other causes: **lithium**, analgesic nephropathy, chronic obstruction, reflux, sarcoidosis, Sjögren's syndrome and myeloma cast nephropathy. In India also consider **aristolochic acid in traditional herbal preparations** and **CKD of unknown aetiology / heat-stress nephropathy** in agricultural workers.
6. **Diagnosis and management. Renal biopsy confirms the diagnosis and demonstrates eosinophilic infiltration**; the usual pattern is **interstitial oedema with a lymphocyte, monocyte and eosinophil infiltrate and tubulitis**. The **cornerstone of treatment is prompt withdrawal of the offending drug**. **Usually the associated renal abnormalities improve after the drug is discontinued, with or without steroid therapy**, but possible complications include **chronic kidney disease or even end-stage renal disease**. **Renal biopsy followed by prompt steroid therapy results in improvement of renal function** — typically **prednisolone 0.5–1 mg/kg/day for 2–4 weeks with a taper over 4–6 weeks**. Add supportive care, avoid all other nephrotoxins, and correct the electrolyte disturbances.

Drug class	Typical latency	Geriatric relevance
NSAIDs	Weeks to months	Often self-medicated for osteoarthritis; may cause nephrotic-range proteinuria with AIN and papillary necrosis
Proton-pump inhibitors	Weeks to months	Frequently continued for years without indication; prime deprescribing target
Beta-lactams / sulphonamides	Days to 2 weeks	Most likely to show the classical triad
Diuretics (thiazide, furosemide)	Variable	Textbook-named cause; confounded by concurrent volume depletion
Allopurinol, rifampicin, mesalamine	Weeks	Allopurinol AIN may accompany DRESS with hepatitis
Immune checkpoint inhibitors	Weeks to months	Increasingly common as older adults receive immunotherapy

LATEST UPDATE (2023–PRESENT) — 1 POINT

7. **Immune checkpoint inhibitor–associated AIN is now a leading cause of drug-induced interstitial nephritis, and older adults receive an increasing share of immunotherapy.** It differs from classical drug AIN: the latency is **longer (weeks to months after initiation)**, **eosinophilia is usually absent**, and a concurrent PPI or NSAID often acts as a co-trigger. Management is to **hold the checkpoint inhibitor and give corticosteroids**, with successful rechallenge possible in a substantial proportion after recovery. The wider practice point, reinforced across recent series, is that **delay of more than about two weeks before starting steroids predicts incomplete renal recovery** — so in an older patient with unexplained non-oliguric AKI and a bland sediment, review not only antibiotics and NSAIDs but also the **PPI and any immunotherapy**, and consider **early biopsy rather than watchful waiting**.

EXAM ONE-LINERS

- Fever + rash + eosinophilia occurs in **<10%**, and less often still in the elderly — its absence proves nothing.
- **Sterile pyuria with white cell casts and non-oliguric AKI** = interstitial nephritis until proven otherwise.
- **Stopping the drug is the treatment.** Steroids are the adjunct, and they work best **early**.
- Unexplained AKI in an older person: think **NSAID, PPI, antibiotic** — in that order of frequency.
- Long-term NSAIDs → **papillary necrosis, chronic interstitial nephritis, ESRD**.

QUESTION 17 · NEPHROLOGY

Diabetic nephropathy and its staging

15-Mark Answer — 10 textbook points + 2 latest updates = 12 points

TEXTBOOK CORE — 10 POINTS

1. **Definition.** Diabetic nephropathy is **persistent albuminuria with progressive decline in GFR** in a person with diabetes, usually accompanied by **retinopathy and hypertension**, in the absence of another renal disease. It is one of the **frequent secondary glomerulopathies in the elderly**.
2. **Mogensen staging (classical, five stages). Stage I** — hyperfiltration and renal hypertrophy. **Stage II** — silent stage, GBM thickening and mesangial expansion with normoalbuminuria. **Stage III** — incipient nephropathy, **microalbuminuria 30–300 mg/day**. **Stage IV** — overt nephropathy, **macroalbuminuria >300 mg/day**, hypertension, falling GFR. **Stage V** — end-stage renal disease.
3. **Current KDIGO "CGA" staging** is now used in practice: **C**ause + **G**1–G5 by eGFR (≥90, 60–89, 45–59, 30–44, 15–29, <15) + **A**1–A3 by albuminuria (UACR <30, 30–300, >300 mg/g). Risk of progression is graded green–yellow–orange–red on this grid.
4. **Histopathology.** GBM thickening, **mesangial expansion**, **Kimmelstiel–Wilson nodular glomerulosclerosis**, hyaline arteriolosclerosis of both afferent and efferent arterioles. RPS classification: **Class I–IV** with separate interstitial and vascular scores.
5. **Risk factors.** Duration of diabetes, poor glycaemic control, **hypertension**, smoking, dyslipidaemia, obesity, genetic susceptibility, and pre-existing age-related nephrosclerosis.
6. **Geriatric-specific caveats.** A **normoalbuminuric CKD phenotype** is common; **sarcopenia lowers serum creatinine** so eGFR **overestimates** true renal function; ageing itself brings glomerulosclerosis and aglomerular circulation, so a fall in eGFR is often multifactorial.

7. **Screening.** Annual **UACR plus eGFR from the time of diagnosis** in type 2 diabetes. Confirm an abnormal UACR on **2 of 3 samples over 3–6 months**; exclude fever, infection, exercise, heart failure and menstruation as transient causes.
8. **When to suspect non-diabetic renal disease and biopsy: absence of retinopathy**, active urinary sediment (RBC casts), short duration of diabetes, abrupt onset of heavy proteinuria, rapid unexplained fall in GFR, or features of a systemic disease such as myeloma or vasculitis.
9. **Management pillars.** Individualised glycaemic control (see targets below), **blood pressure control**, **RAS blockade with a single agent** — ACE inhibitor or ARB, never both — statin therapy, smoking cessation, protein moderation and avoidance of nephrotoxins (NSAIDs, aminoglycosides, contrast). Accept a creatinine rise of **up to 30%** after starting a RAS blocker; monitor potassium.
10. **Complications and prognosis.** Progression to ESRD, **very high cardiovascular mortality**, hyperkalaemia, anaemia of CKD, CKD–mineral and bone disorder, and increased drug toxicity. In the very old, the **competing risk of death exceeds the risk of reaching dialysis** — this must shape how aggressively you treat.

Patient group	HbA1c target	Blood pressure	Lipids
Functionally independent / non-frail	7.0–7.5%	<140/90 mmHg	Statin unless contraindicated; intensity by CVD risk
Functionally dependent	7.0–8.0%	<140/90 mmHg	
Frail / highly comorbid / poor health	<8.0–8.5%	<150/90 mmHg	

Consolidated from EDWPOP, IDF and ADA targets as tabulated in Pathy's. Avoid HbA1c <6% — a J/U-shaped relationship exists, with optimal HbA1c around 7.5–8%.

LATEST UPDATES (2023–PRESENT) — 2 POINTS

11. **FLOW trial (NEJM 2024) — the fourth pillar.** In 3,533 adults with type 2 diabetes and CKD (**mean age 66.6 years**), **semaglutide 1 mg weekly reduced major kidney disease events by 24%**, MACE by 18% and **all-cause death by 20%**, and slowed eGFR decline by **1.16 mL/min/1.73 m^2/year**. Benefit was consistent across all strata of CKD severity. Current therapy of diabetic kidney disease is therefore **four-pillar: RAS blocker + SGLT2 inhibitor + finerenone + GLP-1 receptor agonist**.
12. **ADA Standards of Care 2026 (Section 13, endorsed by the American Geriatrics Society). A tighter blood pressure goal for those at high cardiovascular or kidney risk and a more relaxed goal for most other older adults.** Also: in kidney failure, recent blood loss or erythropoietin therapy, **HbA1c is unreliable** — use blood glucose monitoring and/or **CGM** for goal setting instead; CGM is now recommended for older adults with type 2 diabetes on insulin.

CURRENT PRACTICE UPDATE

Aspect	Textbook	Current Recommendation
Staging	Mogensen stages I–V	KDIGO CGA grid (G1–G5 + A1–A3); Mogensen retained for teaching
Renoprotection	RAS blockade + glycaemic and BP control	Four pillars: RASi + SGLT2i + finerenone + GLP-1 RA
Monitoring in advanced CKD	HbA1c	CGM / glucose profiles, as HbA1c is falsely low

Exam approach: Give Mogensen stages as the "staging" answer — that is what the question asks — then add the KDIGO grid in one line to show currency.

MEMORY ANCHORS

Mogensen mnemonic — "HS-MOE": **H**yperfiltration → **S**ilent → **M**icroalbuminuria → **O**vert → **E**SRD.

Numerical anchors: UACR 30 and 300 mg/g · creatinine rise ≤30% acceptable after RASi · SGLT2i can be started down to eGFR 20 · HbA1c 7–8% in most older adults · FLOW = 24 / 18 / 20 (kidney / MACE / death).

PART IV

Diabetes Mellitus

Q18 · Q19 · Q20 · Q21 · Q22 · Q23 · Q24

QUESTION 18 · DIABETES — CLINICAL SCENARIO

A 65-year-old newly diagnosed diabetic with HbA1c 10%, hypertension, dyslipidaemia, osteoarthritis and BMI 35 refuses to adhere to diet and exercise. Which antidiabetic to start, and what to add

7-Mark Note — 6 textbook points + 1 latest update = 7 points

TEXTBOOK CORE — 6 POINTS

1. **Frame the problem before naming a drug.** This man has **obesity-driven type 2 diabetes with a very high HbA1c (10%), hypertension, dyslipidaemia and osteoarthritis** — a cluster that is essentially the **metabolic syndrome with a mechanical complication**. Two consequences follow. First, **at 65 with a long life expectancy he is not in the "frail, relax the target" category**; aim for an HbA1c around **7.0–7.5%**. Second, his refusal of diet and exercise is not a reason to abandon lifestyle counselling but a reason to **choose drugs that deliver weight loss and cardiovascular and renal protection independently of adherence** — the pharmacological choice must do the work that lifestyle is not doing.
2. **First-line: metformin, and start it at diagnosis. Metformin is first-line therapy in overweight diabetic patients.** It is **weight-neutral to weight-reducing, does not cause hypoglycaemia, and is inexpensive** — decisive in Indian practice. Start **500 mg once daily with food and titrate slowly** to limit gastrointestinal intolerance; use the extended-release preparation if needed. Check **eGFR before starting and periodically**: **do not use metformin in renal insufficiency or heart failure**, reduce the dose below an eGFR of 45 and stop below 30, and monitor **vitamin B12** on long-term therapy.
3. **An HbA1c of 10% will not be controlled by metformin alone — start combination therapy immediately.** Metformin lowers HbA1c by about **1–1.5%**, which leaves him well short of target. Current practice is that when HbA1c is **more than 1.5–2% above target, dual therapy should be initiated from the outset** rather than sequentially. If he were **markedly symptomatic with polyuria, polydipsia and weight loss, or had glucose above 300 mg/dL or ketosis**, the correct answer would be **initial basal insulin** to relieve glucotoxicity, with subsequent de-escalation to oral agents once glucose falls — but the question describes weight gain, not catabolism, so combination oral and injectable non-insulin therapy is appropriate.
4. **Second agent: a GLP-1 receptor agonist — the best fit for this specific patient.** Its advantages align exactly with his problem list: **the largest HbA1c reduction of the non-insulin classes (about 1.0–1.5%)**, **substantial weight loss (3–6 kg, more with semaglutide or tirzepatide)**, **no intrinsic hypoglycaemia** because insulin release is glucose-dependent, **proven benefit in established atherosclerotic cardiovascular disease**, and —

critically here — **weight loss is the single most effective disease-modifying intervention for knee osteoarthritis**, so one drug addresses the diabetes, the obesity and the joint. Counsel about **nausea, vomiting and anorexia**, titrate slowly, and monitor for excessive weight loss and sarcopenia. Cost is the practical barrier in India.

5. **Third agent, or an alternative second agent: an SGLT2 inhibitor.** Add **empagliflozin or dapagliflozin** for the **hypertension** (a modest natriuretic blood-pressure reduction), for **cardiovascular and renal protection irrespective of HbA1c**, and for **2–3 kg of weight loss without hypoglycaemia**. Generic empagliflozin and dapagliflozin are **widely available and inexpensive in India**, which makes this the pragmatic partner for metformin where a GLP-1 receptor agonist is unaffordable. Counsel on **genital mycotic infection, perineal hygiene, volume depletion, and sick-day rules**, and warn about **euglycaemic ketoacidosis**.
6. **What to avoid, and the non-drug obligations you must still discharge. Avoid sulphonylureas** — they cause **hypoglycaemia and weight gain**, both wrong for this man; **avoid thiazolidinediones — do not use in heart failure**, and they cause **weight gain, fluid retention and fracture**; use **DPP-4 inhibitors only as a weight-neutral, well-tolerated fallback**, remembering that they showed **no cardiovascular benefit**. Alongside the diabetes: treat the **hypertension — the antihypertensive regimen of a diabetic should include an ACE inhibitor or ARB** — prescribe a **statin**, since **therapy of these risk factors may be of greater benefit than tight glucose control in older diabetics**, and manage the osteoarthritis with **paracetamol, topical NSAIDs, physiotherapy and quadriceps strengthening rather than systemic NSAIDs**, which threaten his kidneys and blood pressure. Finally, **his refusal is a communication problem, not a closed door**: use **motivational interviewing**, negotiate one small, specific, achievable change, involve the family, and revisit it at every visit.

Agent	HbA1c effect	Weight	Why it fits — or does not	Watch for
Metformin	↓1.0–1.5%	Neutral / ↓	**First-line in the overweight;** cheap, no hypoglycaemia	GI upset, B12 deficiency; avoid if eGFR <30 or heart failure
GLP-1 RA	↓1.0–1.5%	↓↓ 3–6 kg	**Best fit — diabetes + obesity + knee OA + ASCVD risk**	Nausea, anorexia, sarcopenia, gallstones, cost
SGLT2 inhibitor	↓0.5–0.8%	↓ 2–3 kg	BP lowering, cardiorenal protection, **cheap generics in India**	Genital infection, volume depletion, euglycaemic DKA
DPP-4 inhibitor	↓0.5–0.8%	Neutral	Fallback only — well tolerated but **no CV benefit**	Rare pancreatitis; do not combine with GLP-1 RA
Sulphonylurea	↓1.0–1.5%	↑	**Avoid** — hypoglycaemia and weight gain	Glibenclamide is a Beers-criteria drug
Pioglitazone	↓0.8–1.2%	↑↑	**Avoid** — oedema, heart failure, fracture	Bladder cancer signal; worsens immobility
Basal insulin	Unlimited	↑	Only if catabolic, glucose >300 mg/dL or ketosis	Hypoglycaemia; de-escalate once glucotoxicity resolves

65 years · HbA1c 10% · BMI 35 · HTN · dyslipidaemia · knee OA · refuses lifestyle change

↓

Symptomatic, catabolic, glucose >300 mg/dL or ketosis? → **Yes = start basal insulin first**, de-escalate later

↓ No

Start dual therapy from the outset: Metformin + **GLP-1 receptor agonist** (weight, ASCVD, knee OA)

↓ if cost prohibits GLP-1 RA

Metformin + **SGLT2 inhibitor** (BP, cardiorenal, cheap generic in India)

↓ if HbA1c still above target at 3 months

Triple therapy: **Metformin + GLP-1 RA + SGLT2 inhibitor** · then basal insulin if needed

↓ In parallel, always

ACE inhibitor or ARB · statin · paracetamol and topical NSAID with quadriceps strengthening for the knee · motivational interviewing at every visit

LATEST UPDATE (2023–PRESENT) — 1 POINT

7. **ADA Standards of Care 2026 — treat the comorbidity, not just the glucose, and treat the obesity as a disease.** Glucose-lowering therapies are now recommended explicitly **for heart, kidney and liver protection independent of HbA1c**, and there is new guidance on **individualised dosing of obesity medications in people with diabetes**. For this patient with a BMI of 35, that reframes the question: obesity is a treatment target in its own right, and **tirzepatide**, a dual GIP/GLP-1 receptor agonist, delivers the greatest combined HbA1c and weight reduction of any non-insulin agent. Two further points make this directly examinable: the **SELECT trial** showed cardiovascular benefit from semaglutide in **overweight and obese patients without diabetes**, confirming that the benefit is not purely glycaemic; and ADA Section 12 highlights a **GLP-1-associated reduction in amputation risk**. The geriatric caveat to state alongside this: these agents cause **loss of lean mass as well as fat**, so in anyone over 65 you must **ensure adequate protein intake and resistance exercise, and monitor for sarcopenia** — which, incidentally, is the argument that finally persuades many patients to accept the exercise they had refused.

EXAMINER TRAPS

- Answering "metformin" and stopping there. At an **HbA1c of 10%** the expected answer is **dual therapy from the outset**.
- Reaching for a **sulphonylurea** because it is cheap and lowers HbA1c briskly — it is exactly wrong in an obese patient at risk of hypoglycaemia.
- Prescribing **pioglitazone** in a patient who is obese and immobile.
- Treating the refusal as the end of the conversation. State that you would use **motivational interviewing and negotiate one small change** — examiners reward this explicitly.
- Forgetting the **ACE inhibitor or ARB and the statin**. The textbook is explicit that treating these risk factors may benefit an older diabetic more than tight glucose control.
- Prescribing **systemic NSAIDs** for his knee — hypertension, renal risk and gastrointestinal bleeding.

QUESTION 19 · DIABETES MELLITUS

SGLT2 inhibitors and GLP-1 receptor agonists in the older adult

15-Mark Answer — 10 textbook points + 2 latest updates = 12 points

TEXTBOOK CORE — 10 POINTS

1. **SGLT2 inhibitors — mechanism.** They block the **sodium–glucose cotransporter 2 in the proximal tubule**, producing **glycosuria and natriuresis**. The natriuresis restores **tubuloglomerular feedback**, causing afferent arteriolar constriction and a fall in **intraglomerular pressure** — this, not the glucose lowering, explains the renoprotection. Drugs: **empagliflozin, dapagliflozin, canagliflozin**.
2. **GLP-1 receptor agonists — mechanism.** They produce **glucose-dependent insulin secretion**, **suppress glucagon**, **delay gastric emptying** and act centrally to **reduce appetite**. Drugs: **liraglutide, semaglutide, dulaglutide, exenatide**; **tirzepatide** is a dual GIP/GLP-1 receptor agonist.
3. **The key geriatric advantage — negligible intrinsic hypoglycaemia risk.** Because the insulin effect of GLP-1 receptor agonists is **glucose-dependent** and SGLT2 inhibitors act independently of insulin, neither causes hypoglycaemia on its own. **Risk appears only when they are combined with a sulphonylurea or insulin** — so reduce the dose of those agents when adding either class. Both also cause **weight loss**.
4. **Cardiovascular outcome trial data.** Overall **cardiovascular safety was demonstrated across all classes** using the **three-point MACE** endpoint (cardiovascular death, non-fatal myocardial infarction, non-fatal stroke). **DPP-4 inhibitors showed no cardiovascular benefit**, whereas **empagliflozin and canagliflozin significantly reduced cardiovascular and renal events**, with **empagliflozin also reducing hospitalisation for heart failure and worsening nephropathy**.
5. **GLP-1 receptor agonists in older participants.** Among the GLP-1 receptor agonist trials, **exenatide showed cardiovascular benefit in participants aged 65 and over** — but with a **43% treatment discontinuation rate**, which is itself an important geriatric lesson about tolerability and adherence in this age group.
6. **Indications now extend well beyond glycaemia. SGLT2 inhibitors** are indicated in **heart failure with both reduced and preserved ejection fraction** and in **chronic kidney disease, with or without diabetes**. **GLP-1 receptor agonists** are indicated in **established atherosclerotic cardiovascular disease** and in **obesity**. Prescribe them for the organ, not only for the HbA1c.
7. **Adverse effects of SGLT2 inhibitors. Genital mycotic infection** and urinary tract infection, **volume depletion with postural hypotension**, **euglycaemic diabetic ketoacidosis**, an **initial reversible "eGFR dip"** of up to 30% which is haemodynamic and expected, rare **Fournier's gangrene**, and the **amputation and fracture signal seen with canagliflozin**.
8. **Adverse effects of GLP-1 receptor agonists. Nausea, vomiting and diarrhoea** (dose-limiting, and the reason for the high discontinuation rate), **anorexia with unintended weight loss**, gallstones and cholecystitis, pancreatitis, and injection-site reactions. **Contraindicated with a personal or family history of medullary thyroid carcinoma or MEN2**.
9. **Geriatric prescribing cautions — the marks are here. Avoid or use SGLT2 inhibitors very cautiously** in recurrent urinary tract infection, urinary incontinence, poor perineal hygiene, recurrent genital candidiasis, or in a patient on a loop diuretic with orthostatic hypotension and falls. **Avoid GLP-1 receptor agonists** in frailty with unintentional weight loss, **sarcopenia**, gastroparesis, poor oral intake or significant anorexia — the weight loss that is a benefit in the obese 55-year-old is a **hazard in the sarcopenic 85-year-old**. Screen for sarcopenia and monitor weight before and during therapy.

10. **Practical use.** Start an SGLT2 inhibitor down to an **eGFR of 20 mL/min/1.73 m²** and continue until dialysis. Apply **sick-day rules** — suspend during acute illness, dehydration, fasting or before surgery. Reduce sulphonylurea or insulin doses when adding either agent. In India, **generic empagliflozin and dapagliflozin are widely available and inexpensive**, whereas **GLP-1 receptor agonists remain costly and are a major barrier to access** — a point worth stating in an Indian examination.

Domain	SGLT2 inhibitor	GLP-1 receptor agonist
Route	Oral, once daily	Injection (daily or weekly); oral semaglutide available
HbA1c reduction	~0.5–0.8%	~1.0–1.5%
Weight	↓ 2–3 kg	↓ 3–6 kg (more with semaglutide/tirzepatide)
Heart failure	**Strong benefit (HFrEF and HFpEF)**	Neutral
Kidney	**Strong benefit**	**Benefit (FLOW)**
Atherosclerotic CVD	Benefit	**Strong benefit**
Main geriatric hazard	Volume depletion, genital/urinary infection, euglycaemic DKA	Anorexia, sarcopenia, GI intolerance, discontinuation
Best older candidate	Heart failure or CKD, well hydrated, continent	Obese, ASCVD, preserved appetite and muscle mass

LATEST UPDATES (2023–PRESENT) — 2 POINTS

11. **FLOW trial (NEJM 2024) — GLP-1 receptor agonists become a kidney drug.** In 3,533 adults with type 2 diabetes and CKD, **mean age 66.6 years** and median follow-up 3.4 years, **semaglutide 1 mg weekly reduced major kidney disease events by 24%** (HR 0.76), **major cardiovascular events by 18%** and **all-cause death by 20%**, and slowed the annual eGFR decline by **1.16 mL/min/1.73 m²**. The trial was **stopped early for efficacy**, and benefit held **across every stratum of baseline eGFR and albuminuria, including advanced CKD**. This established the **four-pillar** approach: RAS blocker + SGLT2 inhibitor + finerenone + GLP-1 receptor agonist.
12. **ADA Standards of Care 2026.** Glucose-lowering therapies are now recommended explicitly **for heart, kidney and liver protection independent of HbA1c**; there is new guidance on **individualised dosing of obesity medications** in people with diabetes; Section 12 highlights **GLP-1-associated reduction in amputation risk** in diabetic foot disease; and Section 13 adds guidance on **protein intake and on assessing geriatric syndromes** in older adults — relevant because both classes cause weight loss in a population already at risk of sarcopenia.

CURRENT PRACTICE UPDATE

Aspect	Textbook	Current Recommendation
Choice of agent	Driven by HbA1c reduction	Driven by comorbidity — heart failure, CKD, ASCVD, obesity
GLP-1 RA and the kidney	Renal benefit uncertain	Proven renoprotection (FLOW)
SGLT2i eGFR floor	Restricted at moderate CKD	Initiate down to eGFR 20; continue until dialysis

Exam approach: The examiner wants to see that you weigh proven organ protection against **sarcopenia, volume depletion, continence and cost** — that balance is the geriatric answer.

MEMORY ANCHORS

"Flozins for the Failing heart and Filtering kidney; -glutides for Fat and arteries." · SGLT2i harms = **4 Fs**: Fungal (genital), Fluid depletion, Fournier's, Fractures (canagliflozin) · GLP-1 RA harms = **4 As**: Anorexia, Appetite loss/sarcopenia, Abdominal upset, Acute pancreatitis · FLOW numbers = **24 / 18 / 20**.

QUESTION 20 · DIABETES

Alpha-glucosidase inhibitors

6-Mark Note — 5 textbook points + 1 latest update = 6 points

TEXTBOOK CORE — 5 POINTS

1. **Mechanism.** Alpha-glucosidase inhibitors are **competitive, reversible inhibitors of the α-glucosidase enzymes of the intestinal brush border** — glucoamylase, sucrase, maltase and isomaltase — and **acarbose additionally inhibits pancreatic α-amylase**. They **delay the breakdown of complex carbohydrates and disaccharides into absorbable monosaccharides**, so glucose absorption is **slowed and shifted distally along the small intestine**. The result is a **blunted postprandial glucose peak**. They do **not** stimulate insulin secretion and do **not** cause hypoglycaemia when used alone.
2. **Drugs and dosing. Acarbose 25–100 mg three times daily**, **miglitol 25–100 mg three times daily**, and **voglibose 0.2–0.3 mg three times daily** — voglibose being widely used in India. Each must be taken **with the first mouthful of each main meal**; a dose taken without food has no effect. **Start low (acarbose 25 mg once daily) and titrate over weeks** to limit flatulence, which is the main reason for discontinuation.
3. **Indications and place in therapy.** Type 2 diabetes with predominantly **postprandial hyperglycaemia**, as monotherapy or as add-on to metformin, sulphonylureas or insulin. The expected HbA1c reduction is modest — about **0.5–0.8%** — so they are **second- or third-line agents**. They are **particularly relevant in India**, where high-carbohydrate diets of rice, wheat and millets produce a large postprandial excursion, and where the drugs are cheap. They are also **weight-neutral or mildly weight-reducing**, and they were shown to **reduce progression from impaired glucose tolerance to diabetes**.
4. **Advantages in the older patient. No intrinsic hypoglycaemia** — the single most valuable property in geriatric practice; **no weight gain**; **no systemic absorption** to speak of (acarbose is minimally absorbed), so few systemic drug interactions; and a **lowering of postprandial glucose**, which is the component that contributes relatively more to total hyperglycaemia in older people than basal glucose does.

5. **Adverse effects, precautions and one critical rule. Adverse effects** are almost entirely gastrointestinal and arise from colonic bacterial fermentation of unabsorbed carbohydrate: **flatulence, abdominal distension, borborygmi, cramps and diarrhoea** — poorly tolerated and often socially unacceptable in older adults. **Raised transaminases** occur with higher acarbose doses, so **monitor liver function**. **Contraindications: inflammatory bowel disease, colonic ulceration, partial intestinal obstruction or predisposition to it, chronic intestinal disease with malabsorption, large hernias, cirrhosis, and severe renal impairment (avoid acarbose if eGFR is below 25–30)**. **The critical rule:** if hypoglycaemia occurs because the patient is *also* on a sulphonylurea or insulin, it **must be treated with oral glucose (dextrose) or intravenous dextrose — NOT with sucrose, table sugar, sweets or fruit juice**, because the drug blocks the very enzyme that would split sucrose into absorbable glucose.

LATEST UPDATE (2023–PRESENT) — 1 POINT

6. **Alpha-glucosidase inhibitors have been displaced from international algorithms but retain a defined niche in Indian practice.** Current international guidance — **ADA Standards of Care 2026** and the ADA/EASD consensus — organises second-line therapy around **cardiovascular, renal and weight benefit**, and on those criteria the alpha-glucosidase inhibitors have **no demonstrated cardiovascular or renal protection** and so rank below SGLT2 inhibitors and GLP-1 receptor agonists; the **ACE (Acarbose Cardiovascular Evaluation) trial** found that acarbose **did not reduce major adverse cardiovascular events** in patients with impaired glucose tolerance and coronary disease, although it **did reduce progression to diabetes**. However, **RSSDI and Indian national guidance continue to recognise a role** for them where postprandial hyperglycaemia predominates on a high-carbohydrate diet, where cost is decisive, and — importantly for geriatrics — where **hypoglycaemia must be avoided at all costs**, such as in the frail patient living alone. The examinable summary: **a safe, cheap, hypoglycaemia-free but weakly effective and poorly tolerated agent, now a third-line option internationally and a legitimate second-line adjunct in the Indian setting**.

MEMORY ANCHORS

Skeleton (drug question format): Mechanism → drugs and dose → indications → benefits → adverse effects → precautions. Six headings, one or two lines each.

Numbers: HbA1c reduction **0.5–0.8%** · acarbose and miglitol **25–100 mg TDS** · voglibose **0.2–0.3 mg TDS** · take with the **first mouthful** · avoid acarbose if eGFR **<25–30**.

EXAMINER TRAP — THE QUESTION THAT IS ALWAYS ASKED

"How would you treat hypoglycaemia in a patient taking acarbose?" The answer is **oral glucose or dextrose, or intravenous dextrose — never sucrose, table sugar, sweets, honey or fruit juice**, because the drug prevents disaccharide hydrolysis so sucrose cannot be absorbed as glucose. Volunteering this before you are asked marks the answer out.

QUESTION 21 · DIABETES MELLITUS

Newer insulins — onset, duration and advantages

7-Mark Answer — 6 textbook points + 1 latest update = 7 points

TEXTBOOK CORE — 6 POINTS

1. **Classification by pharmacokinetics. Rapid-acting analogues** (lispro, aspart, glulisine) — onset 10–20 min, peak 1–2 h, duration 3–5 h. **Ultra-rapid** (faster aspart, lispro-aabc) — onset ~5 min. **Short-acting regular human insulin** — onset 30–60 min, peak 2–4 h, duration 6–8 h. **Intermediate NPH** — onset 1–2 h, peak 4–8 h,

duration 12–18 h. **Long-acting analogues** — glargine U100 and detemir, ~24 h and 12–20 h. **Ultra-long-acting** — **glargine U300 and degludec**, flat and peakless, lasting **24–42 h**.

2. **The core textbook point on basal insulins.** The availability of **various basal insulins with a shelf life of 24 hours or longer — including glargine, degludec, glargine U300 and detemir — has provided much-needed options to lower baseline glucose with a single daily injection**, in addition to non-insulin agents. This simplification is the main gain for older patients.
3. **Insulin carries a higher hypoglycaemia risk than most oral agents in older adults.** The stated principle is to **decrease that risk by carefully choosing an insulin preparation that matches the timing of hyperglycaemia, and by matching the complexity of the regimen to the patient's coping ability** — not to the ideal physiological profile.
4. **The pattern of hyperglycaemia differs with age.** In older people, the **relative contribution of basal hyperglycaemia is lower and that of postprandial hyperglycaemia higher** than in younger patients. This argues for rapid-acting analogues at meals when a full regimen is feasible — but a **basal-only regimen remains simpler and safer** for most frail patients.
5. **Advantages of analogues over human insulin in the elderly. Fewer nocturnal and severe hypoglycaemic episodes** — degludec and glargine U300 have the flattest, most reproducible profiles and the lowest nocturnal hypoglycaemia; **no need for exact 30-minute pre-meal timing** with rapid analogues, which suits erratic eating and carer-dependent meals; and **true once-daily dosing**. For those unable or unwilling to manage a basal-bolus regimen, consider a **twice-daily premixed analogue** (analogues preferentially, to reduce hypoglycaemia).
6. **Delivery devices.** There appear to be **no benefits on glycaemic control from using a pen, pump or vial-and-syringe in older populations**. However, **visual impairment and low dexterity due to arthritis may make vial and syringe impractical in the frail older adult**. **Pre-filled insulin pens are safe and effective for older adults**, and in institutional care various patient, staff and cost factors make pen injectors the preferred mode of delivery.

Insulin	Onset	Peak	Duration	Geriatric note
Lispro / aspart / glulisine	10–20 min	1–2 h	3–5 h	Can be given *after* the meal if intake is unpredictable
Regular human	30–60 min	2–4 h	6–8 h	Needs strict pre-meal timing — poor fit for frailty
NPH	1–2 h	4–8 h	12–18 h	Distinct peak → nocturnal hypoglycaemia
Detemir	1–2 h	Minimal	12–20 h	May need twice daily
Glargine U100	2–4 h	Peakless	~24 h	Reliable once daily
Glargine U300 / degludec	2–6 h	Peakless	**24–42 h**	**Lowest nocturnal hypoglycaemia; flexible timing**

LATEST UPDATE (2023–PRESENT) — 1 POINT

7. **Once-weekly basal insulin icodec (approved 2024 in the EU, Canada and Switzerland).** In the **ONWARDS 1–5** phase 3 programme in type 2 diabetes, icodec was **non-inferior and in several trials superior to once-daily basal insulin for HbA1c reduction**, while cutting basal injections from **seven to one per week**; a pooled analysis showed **efficacy and hypoglycaemia profiles consistent across age subgroups**. It was **not recommended for type 1 diabetes** because **ONWARDS 6** showed higher rates of level 2 and level 3 hypoglycaemia versus degludec. **Relevance to geriatrics:** a once-weekly basal insulin is potentially transformative for the older adult with type 2 diabetes who depends on a family caregiver, a visiting nurse or a nursing home for injections — but note that **steady state is not reached until 3–4 injections**, a loading dose is used, and any hypoglycaemia is correspondingly **prolonged and not rapidly reversible**.

EXAM ONE-LINERS

- Flattest basal profile, lowest nocturnal hypoglycaemia = **degludec and glargine U300**.
- In the elderly, **postprandial** hyperglycaemia contributes relatively more than basal.
- No glycaemic advantage of pen over syringe — but **pens win on vision and dexterity**.
- Match the regimen to the patient's **coping ability**, not to physiology.
- Icodec: approved for T2DM, **not** for T1DM.

EXAMINER TRAP

Do not recommend a basal-bolus regimen for a frail, cognitively impaired 88-year-old simply because it is "physiological". The correct geriatric answer is **the simplest regimen that avoids symptomatic hyperglycaemia and hypoglycaemia** — often basal insulin once daily, or a twice-daily premixed analogue given by a carer.

QUESTION 22 · DIABETES MELLITUS

Hypoglycaemia — evaluation and management

15-Mark Answer — 10 textbook points + 2 latest updates = 12 points

TEXTBOOK CORE — 10 POINTS

1. **Definition and grading. Level 1 (alert value)** — glucose **<70 mg/dL**; **Level 2 (clinically significant)** — **<54 mg/dL**; **Level 3 (severe)** — any level associated with **cognitive impairment requiring external assistance**, with no glucose threshold. **Hypoglycaemia is the critical limiting factor in achieving glycaemic targets in older adults with diabetes.**
2. **Why older people are especially vulnerable.** Higher levels of **polypharmacy**, presence of **frailty**, and a **high comorbid burden**. Additional risk factors: **cognitive impairment**, **renal disease**, exposure to **sulphonylureas — particularly long-acting agents**, insulin exposure — particularly **twice-daily mixed and basal-bolus regimens** — a **low glycated haemoglobin**, and **low body mass**.
3. **Symptom classification. Autonomic** — palpitations, tremor, sweating, hunger, anxiety. **Neuroglycopaenic** — behavioural change, confusion, incoordination, seizure, coma. Autonomic symptoms are the warning system; neuroglycopaenic symptoms are the damage.
4. **The geriatric problem — blunted warning.** Hypoglycaemic symptoms **become less intense with advancing age, especially the autonomic ones**, and the **symptom profile is modified**. This reduction in autonomic symptoms is perceived by older people as **hypoglycaemia unawareness**. Their **symptom threshold may differ**

from the conventional cut-off of 70 mg/dL — so a "normal-sounding" glucose may still be symptomatic.

5. **Atypical presentation in old age.** With increasing age and disease progression, presentations become atypical: **falls**, **delirium**, **focal neurological deficit mimicking stroke**, angina or arrhythmia, nocturnal sweating and nightmares, or simply behavioural disturbance. These are routinely **misattributed to dementia, ageing or transient ischaemic attack**.
6. **Consequences.** Hypoglycaemia causes **hospital admission**, injurious falls and fracture, myocardial ischaemia and arrhythmia (the "dead-in-bed" mechanism), **accelerated cognitive decline** — with a bidirectional relationship between hypoglycaemia and dementia — loss of confidence, driving cessation and treatment non-adherence.
7. **Evaluation in a diabetic patient.** Document the episode and its timing; review **every drug**, especially sulphonylurea type and dose and insulin regimen; assess **renal and hepatic function**, nutritional intake and meal pattern, alcohol, recent weight loss, cognition and the patient's **ability to self-administer treatment**; look for a precipitating acute illness; and check whether the HbA1c is inappropriately low.
8. **Evaluation in a non-diabetic older adult.** Establish **Whipple's triad** — symptoms, a documented low plasma glucose, and relief on raising the glucose. Then, **during a spontaneous episode or a supervised 72-hour fast**, measure **insulin, C-peptide, proinsulin, β-hydroxybutyrate and a sulphonylurea screen**. Causes to consider: **drugs (sulphonylurea, quinolones, quinine), alcohol, sepsis, hepatic or renal failure, adrenal insufficiency, hypopituitarism, non-islet-cell tumour hypoglycaemia (IGF-2), insulinoma** and severe malnutrition.
9. **Acute treatment. Conscious and able to swallow** — the **"15/15 rule"**: 15–20 g of fast-acting carbohydrate (glucose tablets, 3–4 teaspoons of sugar, 150 mL fruit juice), recheck at 15 minutes, repeat if still <70 mg/dL, then give a complex carbohydrate snack. **Unconscious or unable to swallow** — **25% dextrose 50–100 mL IV** followed by a 10% dextrose infusion, or **glucagon 1 mg IM/SC** if no venous access. Glucagon is **ineffective in hepatic glycogen depletion** — malnutrition, alcohol, prolonged fasting.
10. **Prevention — the highest-scoring section.** Key recommendations to minimise hypoglycaemia and its admissions are to **assess hypoglycaemia risk and adjust glucose targets for frailty, functional status and life expectancy**, and to **regularly review diabetes medicines** — sulphonylureas, insulin and its regimens — including **the patient's capacity to self-administer treatment**. Practically: relax HbA1c (7.0–7.5% if non-frail, up to 8.5% if frail), **avoid HbA1c <6%**, **de-intensify** therapy, prefer agents with low hypoglycaemia risk (metformin, DPP-4 inhibitors, SGLT2 inhibitors, GLP-1 receptor agonists), avoid **glibenclamide** (long-acting, renally cleared active metabolites — a Beers criteria drug), and educate the patient and carer.

LATEST UPDATES (2023–PRESENT) — 2 POINTS

11. **ADA Standards of Care 2026, Section 13 "Older Adults" (endorsed by the American Geriatrics Society). Continuous glucose monitoring is now recommended for older adults with type 1 diabetes and, newly in 2026, for those with type 2 diabetes on insulin** — explicitly "to improve glycaemic outcomes, **reduce hypoglycaemia** and reduce treatment burden". Alongside this, **Recommendation 13.2 requires at least annual screening for geriatric syndromes (cognitive impairment, depression, incontinence, falls, persistent pain, frailty), for hypoglycaemia and for polypharmacy** in every older adult with diabetes. There is also new guidance on assessing geriatric syndromes with validated screening questions, and on protein intake.
12. **Sulphonylurea-induced hypoglycaemia is a prolonged, relapsing admission — not a treat-and-discharge event.** Current practice is to **admit and observe for at least 24–48 hours** with a continuous dextrose infusion, because recurrence after apparent recovery is the rule — especially with renal impairment. **Octreotide 50–100 µg subcutaneously** is used for refractory or recurrent sulphonylurea hypoglycaemia because it suppresses further insulin release; it is more effective than escalating dextrose alone. Deprescribing the sulphonylurea, not merely reducing it, is the discharge action.

CURRENT PRACTICE UPDATE

Aspect	Textbook	Current Recommendation
Monitoring	Self-monitored capillary glucose	CGM for older adults with T1DM and T2DM on insulin
HbA1c in CKD/anaemia	Used as the target measure	Unreliable — use CGM / glucose profiles
Screening	Opportunistic enquiry	Structured annual screening for hypoglycaemia, geriatric syndromes and polypharmacy
Goal of care	Intensification to reach target	**De-intensification** is an explicit quality-of-care goal

Exam approach: Emphasise that in geriatrics *avoiding hypoglycaemia outranks achieving a glycaemic target*. State it as a principle at the start of the management section.

MEMORY ANCHORS & 15-SECOND SKELETON

Skeleton: Three levels (70 / 54 / any-with-assistance) → why the old are vulnerable → blunted autonomic warning & unawareness → atypical presentation (falls, delirium, pseudo-stroke) → consequences → evaluate drugs and renal function → Whipple's triad if non-diabetic → 15/15 rule and IV dextrose/glucagon → prevention by relaxing targets and de-intensifying → CGM.

Numbers: 70 / 54 · 15 g and 15 minutes · 25% dextrose 50–100 mL · glucagon 1 mg · observe sulphonylurea overdose 24–48 h · HbA1c 7.0–7.5 / 8.0 / 8.5% by frailty tier.

EXAMINER TRAPS

- Quoting a single glucose threshold — the examiner wants **three levels**, with Level 3 defined by **function, not a number**.
- Discharging a sulphonylurea hypoglycaemia after one correction.
- Giving glucagon in an alcoholic or malnourished patient — **no hepatic glycogen, so it will not work**.
- Treating a low HbA1c as a success in a frail 85-year-old — it is a **red flag for hypoglycaemia**.

QUESTION 23 · DIABETES MELLITUS

Diabetic ketoacidosis and hyperosmolar hyperglycaemic state

15-Mark Answer — 10 textbook points + 2 latest updates = 12 points

TEXTBOOK CORE — 10 POINTS

1. **Definitions. DKA** = hyperglycaemia + **ketonaemia** + **metabolic acidosis** (classically glucose >250 mg/dL, pH <7.30, HCO_3 <18 mEq/L, ketones positive). **HHS** = **marked hyperglycaemia (usually >600 mg/dL)** with **raised effective osmolality** and **profound dehydration**, but **no significant ketosis or acidosis**. Both are the **metabolic comas** of diabetes.
2. **Which one occurs in whom. HHS is the characteristic emergency of the older person with type 2 diabetes**; DKA is classically type 1 but occurs in older type 2 patients under severe physiological stress. **Overlap syndromes are common in the elderly** — up to a third have mixed features.

3. **Precipitants. Infection is the commonest** — urinary tract infection and pneumonia — followed by **myocardial infarction, stroke, sepsis**, non-adherence or omission of insulin, newly diagnosed diabetes, and drugs: **corticosteroids, thiazides, atypical antipsychotics** and **SGLT2 inhibitors** (euglycaemic DKA).
4. **Why older adults do worse.** A **blunted thirst mechanism**, raised osmotic threshold for thirst, **impaired urinary concentrating ability**, reduced free water clearance and dependence on carers for fluids mean that dehydration is **deeper and better tolerated symptomatically** before presentation. **Mortality in HHS is several-fold higher than in DKA** and is driven largely by the precipitating illness.
5. **Clinical features.** Common to both — polyuria, polydipsia, weight loss, dehydration, tachycardia, hypotension. **DKA**: **Kussmaul respiration**, acetone breath, abdominal pain, vomiting, developing over hours. **HHS**: insidious over days to weeks, with **altered sensorium, focal neurological deficits mimicking stroke, seizures and coma**. **In the older adult, delirium or a fall may be the only presentation.**
6. **Investigations.** Capillary and laboratory glucose, **blood β-hydroxybutyrate** (preferred to urine ketones), venous or arterial blood gas, urea, creatinine and electrolytes, **corrected sodium** (add 1.6 mEq/L per 100 mg/dL glucose above 100), **effective osmolality = 2 × Na^+ + glucose/18**, anion gap, full blood count, CRP, blood and urine cultures, chest radiograph and **ECG** (silent myocardial infarction).
7. **Fluids are the first priority.** Start **0.9% sodium chloride**. In older adults with cardiac or renal disease, use **slower, more cautious rates with close monitoring of jugular venous pressure, chest and urine output**. HHS requires a **larger total fluid deficit to be replaced but more slowly**, aiming to lower osmolality by no more than **3–8 mOsm/kg/h** to avoid cerebral oedema.
8. **Insulin.** DKA — **fixed-rate intravenous insulin infusion at 0.1 units/kg/h**. HHS — insulin is started at a **lower rate (0.05 units/kg/h)** and **only after fluids alone have stopped lowering the glucose**, since fluid alone corrects much of the hyperglycaemia. Target a fall of **50–70 mg/dL/h**; add **5–10% dextrose** when glucose reaches ~250 mg/dL in DKA or ~300 mg/dL in HHS, continuing insulin to clear ketones.
9. **Potassium and other electrolytes.** Total body potassium is always depleted despite a normal or high initial level. **Withhold insulin if K^+ <3.3 mEq/L** until potassium is replaced; **add potassium once K^+ <5.3 mEq/L** with adequate urine output. **Bicarbonate only if pH <6.9.** Phosphate only for severe depletion (<1 mg/dL) with cardiac or respiratory muscle weakness.
10. **Complications and resolution.** Iatrogenic **hypokalaemia, hypoglycaemia, fluid overload and pulmonary oedema** (especially in the elderly), hyperchloraemic acidosis, **venous thromboembolism** — give prophylactic low-molecular-weight heparin, particularly in HHS — aspiration, AKI, and rarely cerebral oedema. **DKA has resolved** when ketones have cleared with pH ≥7.3 or HCO_3 ≥18 mEq/L; **HHS has resolved** when osmolality is normal, urine output adequate and mental status has returned to baseline.

Feature	DKA	HHS
Typical patient	Type 1; younger, or stressed type 2	**Older type 2 diabetic**
Onset	Hours	Days to weeks
Glucose	>250 mg/dL (may be euglycaemic)	Usually >600 mg/dL
Ketones (β-OHB)	≥3.0 mmol/L	<3.0 mmol/L
pH / HCO_3	<7.3 / <18 mEq/L	≥7.3 / ≥15 mEq/L
Effective osmolality	Variable	**>300–320 mOsm/kg**
Fluid deficit	~100 mL/kg	**~100–200 mL/kg (greater)**
Insulin rate	0.1 units/kg/h	0.05 units/kg/h, after fluids
Neurological signs	Uncommon	**Common — coma, seizures, focal deficit**
Mortality	Lower	**Substantially higher**

LATEST UPDATES (2023–PRESENT) — 2 POINTS

11. **ADA/EASD/JBDS/AACE/DTS 2024 Consensus Report on Hyperglycaemic Crises — new diagnostic criteria.** **DKA** now requires the "**DKA triad**": **D** — glucose **≥200 mg/dL** (lowered from 250) **or any prior history of diabetes irrespective of glucose**; **K** — **β-hydroxybutyrate ≥3.0 mmol/L** or urine ketones ≥2+; **A** — **pH <7.3 and/or HCO_3 <18 mmol/L**. The **anion gap has been removed** from both the diagnostic and the resolution criteria (it is distorted by hyperchloraemia from saline). **HHS** now requires glucose ≥600 mg/dL, **effective osmolality >300 mOsm/kg (or total >320)**, β-OHB <3.0 mmol/L and pH ≥7.3 with HCO_3 ≥15. **Direct measurement of β-hydroxybutyrate is strongly recommended** over urine ketones. Severe DKA = β-OHB >6 mmol/L, pH <7.0, HCO_3 <10, or stupor/coma — these plus all HHS need **ICU care**.
12. **SGLT2 inhibitors and euglycaemic DKA.** SGLT2 inhibitors now account for the majority of euglycaemic DKA cases. Practical consequences: **check ketones whenever an unwell patient on an SGLT2 inhibitor presents, whatever the glucose**; apply **sick-day rules** — withhold the drug during acute illness, dehydration, fasting or reduced oral intake, and **stop it 3–4 days before elective surgery**. The 2024 consensus also recommends that **CGM be offered at discharge to all patients admitted with DKA**, and gives specific guidance for frail older adults.

CURRENT PRACTICE UPDATE

Aspect	Textbook	Current Recommendation
Glucose criterion for DKA	>250 mg/dL	≥200 mg/dL, or any prior diabetes regardless of glucose
Ketone assessment	Urine nitroprusside	Blood β-hydroxybutyrate ≥3.0 mmol/L (point-of-care)
Anion gap	Part of diagnosis and resolution	Removed from both criteria
Mild–moderate uncomplicated DKA	IV insulin infusion for all	Subcutaneous rapid-acting analogue protocols acceptable outside ICU

Exam approach: Give the classical criteria, then state the 2024 DKA "D–K–A" triad in one sentence. Examiners reward the euglycaemic DKA point.

MEMORY ANCHORS & 15-SECOND SKELETON

Skeleton: Define both → who gets which → precipitants (infection commonest) → why the old do worse (thirst + concentrating defect) → clinical picture → investigations including corrected Na and osmolality → **Fluids, Insulin, Potassium (FIP)** in that order → complications → resolution criteria → 2024 criteria + SGLT2i.

Numbers to memorise: 200 / 3.0 / 7.3 / 18 (DKA) · 600 / 300 / 320 (HHS) · insulin 0.1 vs 0.05 units/kg/h · K^+ 3.3 and 5.3 · bicarbonate only if pH <6.9 · fall in glucose 50–70 mg/dL/h.

EXAMINER TRAPS

- Starting insulin before fluids, or before checking potassium — both are marked down heavily.
- Forgetting that **a normal or high initial potassium hides total body depletion**.
- Aggressive fluid resuscitation in an 85-year-old with heart failure — say "cautious, monitored" explicitly.
- Missing **euglycaemic DKA** on an SGLT2 inhibitor because the glucose "looked fine".
- Forgetting **thromboprophylaxis in HHS**.

QUESTION 24 · DIABETES MELLITUS

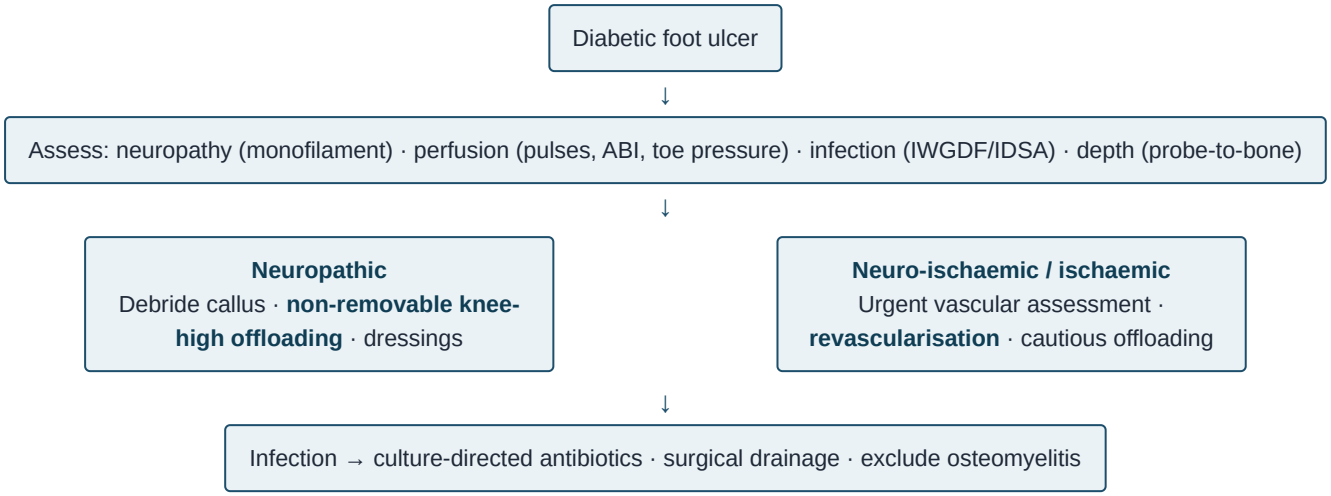
Diabetic foot syndrome

15-Mark Answer — 10 textbook points + 2 latest updates = 12 points

TEXTBOOK CORE — 10 POINTS

1. **Definition.** Diabetic foot syndrome is **ulceration, infection and/or destruction of deep tissues of the foot** in a person with diabetes, arising from the interaction of **peripheral neuropathy, peripheral arterial disease and repetitive mechanical stress**. **The majority of the elderly diabetic population is at increased risk of developing foot ulcers.**
2. **Principal antecedents. Peripheral vascular disease**, **sensorimotor and autonomic neuropathy**, **limited joint mobility — which impairs the ability of older people to inspect their own feet** — and **high foot pressures**. **Coexisting visual loss exacerbates the consequences** of this situation, and visual acuity is impaired in about 40% of older diabetics.
3. **Peripheral sensorimotor neuropathy is the dominant mechanism** — the primary cause or a contributory factor in the **vast majority of cases**. It causes numbness, **lancinating and burning pain, "pins and needles" and hyperaesthesia typically worse at night**, and produces high foot pressures leading to **gait disturbance, falls and other foot injuries**. Autonomic neuropathy causes dry, fissured skin and arteriovenous shunting.
4. **Ulcer characteristics.** Diabetic ulcers most commonly form in **weight-bearing areas — the metatarsal heads and heel**. They are usually **deep, with undermined edges, surrounded by hyperkeratosis (callus)**, and are **often painless because of the peripheral neuropathy** — which is why they present late.
5. **Distinguish from the other foot ulcers of old age. Arterial** — painful, with coldness, atrophy and hairlessness of surrounding skin. **Venous** — between the ankle and calf, shallow, irregular borders, substantial exudate, with oedematous and hyperpigmented surrounding tissue. **Decubitus (pressure)** — posterior heel or malleoli in those confined to bed or using wheelchairs.
6. **Clinical classification.** Describe the ulcer as **neuropathic** (loss of protective sensation, no PAD), **neuro-ischaemic** (both) or **ischaemic** (PAD without loss of protective sensation). Severity is graded by the **Wagner** grades, or by the **SINBAD** and **WIfI** systems; infection severity by the **IWGDF/IDSA** scheme.

7. **Assessment. 10 g Semmes–Weinstein monofilament** and 128 Hz tuning fork for loss of protective sensation; palpation of pedal pulses and **ankle–brachial index**, with **toe pressures or $TcPO_2$ when vessels are calcified and the ABI is falsely high**; **probe-to-bone test**; plain radiograph and MRI for **osteomyelitis**; deep tissue culture rather than superficial swab; plus glycaemic, renal, nutritional and **vision and gait** assessment.
8. **Management principles.** Managing foot ulceration in older people involves **addressing the underlying aetiology** — **surgical revascularisation for arterial ulcers** and **optimum management of blood sugar levels for diabetic foot ulcers** — **in addition to debridement, management of infection, and application of appropriate topical medications, wound dressings and offloading techniques**.
9. **Prognosis and burden.** The **three-year survival following lower extremity amputation is about 50%**, and in about **70% of cases amputation is precipitated by foot ulceration**. **Increasing age and a higher level of amputation** are important factors increasing both length of hospitalisation and cost. A **history of foot ulceration is independently associated with visual loss and with increased mortality** at ten years.
10. **Prevention — five key elements. Identify the at-risk foot**; **inspect and examine the at-risk foot regularly**; **educate the patient, family and healthcare providers**; **ensure routine wearing of appropriate footwear**; and **treat pre-ulcerative signs** — callus, haemorrhage under callus, blister, fissure, ingrowing nail. A **multidisciplinary foot-care team approach decreases the incidence of major amputation**.



LATEST UPDATES (2023–PRESENT) — 2 POINTS

11. **IWGDF 2023 classification guideline. SINBAD (Site, Ischaemia, Bacterial infection, Area, Depth) is the recommended first-choice system** for communication between healthcare professionals — **stating the presence or absence of each composing variable rather than only a total score** — and the **full SINBAD score is recommended for regional, national and international audit**. Use the **IWGDF/IDSA (2015) system to classify infection severity** and consider **WIfI** to stratify healing likelihood and amputation risk in peripheral arterial disease. Crucially, **no currently available classification should be used to give an individual patient an outcome prognosis**.
12. **IWGDF 2023 offloading guideline — offloading is the single most important healing intervention.** The **preferred treatment for a neuropathic plantar forefoot or midfoot ulcer is a non-removable knee-high offloading device** — either a **total contact cast** or a **removable walker rendered irremovable by the provider fitting it**. If a non-removable device is contraindicated or not tolerated, use a **removable knee-high or ankle-high device** with explicit education on adherence; if no other biomechanical relief is available, use **felted foam only in combination with appropriate footwear**. For ulcers on **digits 2–5 secondary to a flexible toe deformity, perform digital flexor tenotomy**. Be **more cautious with offloading when infection or ischaemia is present**.

CURRENT PRACTICE UPDATE

Aspect	Textbook	Current Recommendation
Classification	Wagner grading	SINBAD first choice for communication and audit; IWGDF/IDSA for infection; WIfI for PAD
Offloading	Listed among treatment measures	Non-removable knee-high device (TCC) is first choice and the key determinant of healing
Prognostication	Grades used to predict outcome	No classification should be used for individual prognosis

Exam approach: Wagner is still acceptable to quote, but naming **SINBAD** and the **total contact cast** marks the answer out as current.

INDIAN CONTEXT

- **Barefoot walking and religious barefoot practice** are major, modifiable ulcer risks — address footwear explicitly in counselling.
- Presentation is **late and more often infected**; Charcot neuro-osteoarthropathy is **under-diagnosed** and misread as cellulitis or gout.
- **NPCDCS** provides opportunistic diabetes and foot screening at HWC/CHC level; therapeutic footwear can be accessed through **ADIP–ALIMCO** for eligible older persons.
- Multidisciplinary foot clinics, where available, reduce major amputation — cite this as the service-level recommendation.

MEMORY ANCHORS & 15-SECOND SKELETON

Skeleton: Define the triad → antecedents (neuropathy, PAD, limited joint mobility, high pressure, poor vision) → why painless and late → ulcer site and appearance → differentiate arterial/venous/pressure → classify (neuropathic / neuro-ischaemic / ischaemic; SINBAD) → assess (monofilament, ABI, probe-to-bone) → manage (revascularise, debride, treat infection, **offload**, control glucose) → prognosis (50% three-year survival post-amputation) → prevent (five elements + MDT).

Numbers: 70% of amputations preceded by ulcer · **50%** three-year survival after amputation · **10 g** monofilament · **SINBAD** = Site, Ischaemia, Neuropathy/Bacterial infection, Area, Depth.

PART V

Thyroid Disease

Q25 · Q26 · Q27 · Q28 · Q29

QUESTION 25 · THYROID

Subclinical hypothyroidism in the elderly

7-Mark Note — 6 textbook points + 1 latest update = 7 points

Recurring question — asked ×2. Extra scaffolding provided below.

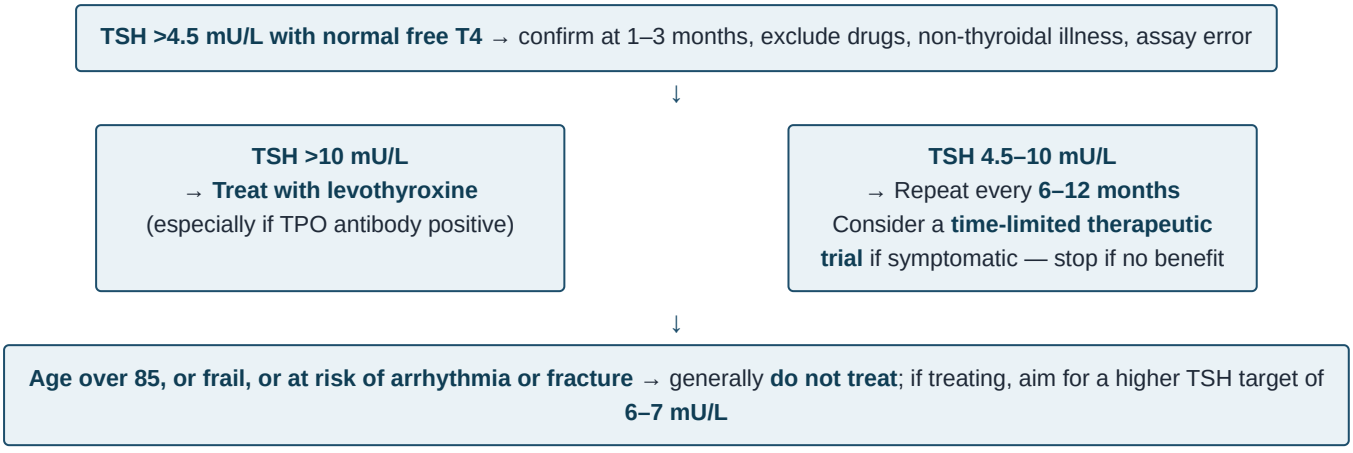
TEXTBOOK CORE — 6 POINTS

1. **Definition and prevalence.** Subclinical hypothyroidism is **an elevated serum TSH level in the face of normal or normal-low free thyroid hormone values**. Overall prevalence is **4–10% in the general population and up to 20% in women older than 60**. Subclinical disorders are **more prevalent than overt disease in older populations** — a consequence of sensitive TSH assays and more frequent testing.
2. **Confirm before you act.** A TSH measurement **over 4.5 mU/L should be confirmed within 1–3 months**, and then **repeated every 6–12 months in asymptomatic patients**. If confirmed, consider **past radioiodine administration, previous thyroid surgery, thyroid enlargement, and a family history of thyroid dysfunction**, explore **subtle clinical signs of hypothyroidism**, and check the **lipid profile**. Also exclude non-thyroidal illness recovery, drugs and assay interference.
3. **Natural history. Progression to overt hypothyroidism varies from 3 to 20% but is generally around 5% per year**; the risk is **greater in patients with goitre or thyroid antibodies, or both**. **Antithyroid antibodies are useful for improving the prognosis of progression to overt hypothyroidism, but they may not change the management of the patient**. Hence patients should be **followed and eventually treated** rather than treated immediately.
4. **The treatment threshold. If, during follow-up, TSH increases to over 10 mU/L, the patient should be treated with levothyroxine. When TSH is between 4.5 and 10 mU/L, an *ex adiuvantibus* (therapeutic trial) administration of levothyroxine may be considered on an individual basis** to help improve subtle clinical symptoms — with a clear plan to stop if no benefit accrues.
5. **The paradox of advanced age — mild thyroid failure may be protective. In the oldest old (over 85 years), thyroid hormone substitution in subclinical hypothyroidism is not likely to be beneficial and may even be harmful.** In the Leiden 85-plus study, **none of the patients with subclinical hypothyroidism had progressed to overt disease when retested at age 88**, there was **no association between TSH and cognition, depression or ADL disability**, and **increased TSH at baseline was associated with a slower decline in instrumental ADL ability and with lower all-cause and cardiovascular mortality** despite higher cholesterol. The explanation offered is that **subclinical hypothyroidism seems detrimental in young to middle-aged people, whereas it may be harmless or possibly beneficial at an advanced age**.

6. **Individualise, and use a higher TSH target in the old. The decision to treat should be individualised — not based on TSH cut-offs alone, but also considering chronic comorbidities and frailty — and restricted to high-risk patients**, to avoid the adverse effects of unnecessary replacement in vulnerable older people. **A higher target serum TSH of 6 or 7 mU/L has been suggested in people over 70–80, particularly if they are at risk of cardiac arrhythmias or fragility fractures.** Remember too that **many older patients started levothyroxine when younger for inappropriate reasons or for transient hypothyroidism and continue it without review** — in one nursing-home study **thyroid hormone therapy was successfully withdrawn from half the residents studied**.

LATEST UPDATE (2023–PRESENT) — 1 POINT

7. **The TRUST trial settled the symptomatic question, and subsequent practice has consolidated around not treating.** TRUST was a double-blind, randomised, placebo-controlled trial of **737 adults aged 65 and over (mean age 74.4) with persistent subclinical hypothyroidism**, randomised to **levothyroxine 50 µg/day — or 25 µg/day if body weight was under 50 kg or coronary heart disease was present** — titrated to TSH, versus placebo with simulated dose adjustment. Mean baseline TSH was **6.40 mU/L**; at one year it fell to **3.63 mU/L on levothyroxine versus 5.48 on placebo**, yet there was **no difference in the Hypothyroid Symptoms score or the Tiredness score**, and **no benefit on executive cognitive function, blood pressure, weight, waist circumference, Barthel index or instrumental ADL scores**. Current guidance and the consolidated position is therefore that **treatment of minor TSH abnormalities meeting the criteria for subclinical hypothyroidism in the elderly is not recommended**. Two honest caveats to state in the exam: the trial was **underpowered for cardiovascular outcomes**, and **patients with higher TSH concentrations, high symptom burden, positive TPO antibodies, and the oldest patients over 80 were under-represented or not assessed**, so no definitive conclusion can be drawn for those subgroups.



CURRENT PRACTICE UPDATE

Aspect	Textbook	Current recommendation
Symptomatic benefit of levothyroxine	Trial of therapy may be considered at TSH 4.5–10	TRUST: no symptomatic, cognitive or functional benefit in those over 65 — do not treat routinely
TSH target when treating an older patient	Normalise TSH	Accept a higher target of 6–7 mU/L over age 70–80, especially with arrhythmia or fracture risk
Existing long-term levothyroxine	Continued indefinitely	Actively review for deprescribing — successfully withdrawn in half of nursing-home residents studied

Exam approach: The single sentence that earns the mark is that **subclinical hypothyroidism appears detrimental in the middle-aged but harmless or possibly protective in the oldest old**. Say it explicitly, then give the TSH 10 threshold.

MEMORY ANCHORS & 15-SECOND SKELETON

Skeleton: Define (high TSH, normal free T4) → prevalence 4–10%, up to 20% in women over 60 → confirm at 1–3 months, repeat 6–12 monthly → progression ~5%/year, higher with antibodies or goitre → **treat if TSH >10**, individualise 4.5–10 → in the oldest old do not treat, possibly protective → higher TSH target 6–7 → review existing prescriptions → TRUST.

Numbers: TSH **4.5** to confirm · **10** to treat · **6–7** target over age 70–80 · prevalence **4–10%**, **20%** in women over 60 · progression **~5%/year** · TRUST **737** patients, mean age **74.4**, levothyroxine **50 µg** (25 µg if under 50 kg or CHD), baseline TSH **6.40** → **3.63** versus **5.48**.

EXAMINER TRAPS

- Treating on a **single TSH value** — always confirm at 1–3 months.
- Forgetting that **TSH rises physiologically with age**, so an age-specific reference range matters; a TSH of 6 in an 85-year-old may be normal for them.
- Missing the **non-thyroidal illness** explanation — a recovering acute illness transiently raises TSH.
- Quoting TRUST as proving levothyroxine is useless in *everyone* — it was underpowered for cardiovascular outcomes and under-represented those over 80 and those with high TSH or positive antibodies.
- Overlooking **iatrogenic thyrotoxicosis**: over-replacement in an older person causes atrial fibrillation, bone loss and fracture.

QUESTION 26 · THYROID — CLINICAL SCENARIO

A 70-year-old on treatment for cardiac failure has TSH 50 µIU/mL with suppressed T3 and T4, constipation, cold intolerance and considerable weight gain. Treatment, follow-up and specific instructions

7-Mark Note — 6 textbook points + 1 latest update = 7 points

TEXTBOOK CORE — 6 POINTS

1. **Establish the diagnosis and its cause before prescribing.** This is **overt primary hypothyroidism** — a markedly elevated TSH with low free T4. **The most frequent cause of hypothyroidism in old age is autoimmune thyroiditis, followed by earlier thyroid surgery or radiation therapy for previous thyrotoxicosis.** Confirm with **free T4 and antithyroid (anti-TPO) antibodies**. In this patient specifically, ask about **amiodarone** — a common drug in heart failure and a classic cause of hypothyroidism — and about lithium, and check the **lipid profile, creatine kinase (often raised), haemoglobin** (normocytic anaemia responds to replacement), sodium and an **ECG**.
2. **Recognise that his "heart failure" may itself be the hypothyroidism.** The symptoms of hypothyroidism in old age are atypical and include **memory loss, lethargy, constipation, cold intolerance, fatigue, congestive heart failure, depression and weight gain — all of them often attributed to old age or other causes rather than to decreased thyroid function**. Hypothyroidism causes **bradycardia, reduced cardiac output, diastolic hypertension, pericardial effusion and non-pitting myxoedematous oedema** that mimics cardiac failure. Reassess the cardiac diagnosis after he becomes euthyroid — his diuretic dose may need reduction.
3. **Treatment — start low and go slow, because he has cardiac disease. Therapy should be started at 0.025 to 0.05 mg (25–50 µg) of levothyroxine per day and increased by the same increment at 1- to 3-week intervals. Given established cardiac disease, start at 12.5–25 µg daily.** The rationale is that thyroid hormone increases myocardial oxygen demand and can precipitate **angina, atrial fibrillation, acute coronary syndrome or worsening failure**. **The decreased metabolic clearance rate of thyroid hormone in ageing may lead to a lower maintenance dose of T4**, and **the replacement dose in older people is usually lower than in younger people**.
4. **Titration and monitoring. Increase the dose slowly, every six to eight weeks**, guided by TSH — a shorter interval simply measures a level that has not yet equilibrated. Monitor **heart rate, symptoms of angina, weight, bowel habit and mental state** at each visit, and **the TSH level in the laboratory**. **When indicated for symptomatic cardiovascular disease, a beta blocker may be added to the levothyroxine regimen.** Aim for a TSH in the reference range but at the **higher end — around 4–6 mU/L is acceptable in a 70-year-old with heart disease**; avoid a suppressed TSH, which risks atrial fibrillation and bone loss.
5. **Follow-up schedule.** Review **clinically and with TSH every 6–8 weeks during titration**; once stable, check TSH at **6 months and then annually**. Recheck earlier if there is a change in weight, a new drug that alters absorption or metabolism, an intercurrent illness, or new symptoms. At every review, reassess the **heart failure medication**, since diuretic and rate-control requirements often fall as he becomes euthyroid, and screen for **over-replacement — palpitations, tremor, weight loss, insomnia, angina, atrial fibrillation**.
6. **Specific instructions to give this patient. (i)** Take levothyroxine **once daily on an empty stomach, 30–60 minutes before breakfast, with water** — or at bedtime, at least two hours after food, if that is easier to remember. **(ii) Separate it by at least four hours** from **calcium, iron, antacids, proton-pump inhibitors, sucralfate and soya products**, which impair absorption — an important point in an older patient on multiple drugs. **(iii) Do not stop or alter the dose yourself, and do not double up a missed dose**; take it the next day. **(iv) Treatment is lifelong. (v) Report immediately** chest pain, palpitations, breathlessness or an irregular pulse. **(vi)** Expect **gradual improvement over weeks to months, not days** — constipation and cold intolerance

settle first, weight last. **(vii)** Tell every doctor and pharmacist that you take levothyroxine, and **stay on the same brand** where possible. **(viii)** Continue a high-fibre diet, fluids and activity for the constipation while replacement takes effect.

Stage	Action	Rationale in this patient
Confirm	Free T4, anti-TPO antibodies, review amiodarone, CK, lipids, haemoglobin, ECG	Autoimmune is commonest; amiodarone is a heart-failure drug and a classic cause
Initiate	**Levothyroxine 12.5–25 µg once daily**	Cardiac disease — avoid precipitating angina, arrhythmia or worsening failure
Titrate	Increase by 12.5–25 µg every **6–8 weeks** guided by TSH	TSH takes 6 weeks to equilibrate; ageing lowers clearance so the final dose is small
Target	TSH in range, upper half (**~4–6 mU/L** acceptable)	Avoid iatrogenic thyrotoxicosis, atrial fibrillation and fracture
Maintain	TSH at 6 months, then annually	Stable requirement; recheck on any drug or weight change
Reassess	Re-evaluate the heart failure diagnosis and drug doses	Myxoedema mimics failure; diuretic need often falls

LATEST UPDATE (2023–PRESENT) — 1 POINT

7. **Current practice has moved further towards individualised, age-adjusted TSH targets and away from combination therapy.** Three points are worth stating. **(a)** An **age-specific upper TSH reference limit** is now widely accepted, since TSH rises physiologically with age; targeting the young adult range in a 70-year-old with heart disease produces **over-treatment**, and registry data link a suppressed TSH on levothyroxine to **atrial fibrillation, osteoporotic fracture and increased mortality in older adults** — so the target here is the **upper half of the reference range**. **(b) Liothyronine (T3) and combination T4/T3 therapy are not recommended** for routine use, and are specifically inadvisable in older patients with cardiac disease because of the sharp, non-physiological T3 peaks. **(c)** If he is on **amiodarone**, the guidance is that amiodarone-induced hypothyroidism **does not usually require stopping the amiodarone** — treat with levothyroxine and continue the antiarrhythmic if it is needed, monitoring thyroid function **every 6 months** for as long as he takes it and for a year afterwards.

EXAMINER TRAPS

- Starting a **full weight-based replacement dose (1.6 µg/kg)** in a 70-year-old with cardiac failure. This is the single commonest error in this scenario — the marks are in "start low, go slow".
- Rechecking TSH at **two weeks**. It has not equilibrated; wait 6–8 weeks.
- Aiming for a low-normal or suppressed TSH.
- Omitting the **drug-absorption instructions** — the question explicitly asks what you would "specifically instruct him", so calcium, iron, PPIs and the empty-stomach rule carry marks.
- Failing to consider that the **heart failure may be the hypothyroidism**, and failing to plan a reassessment.
- Not asking about **amiodarone** in a patient on heart-failure treatment.

IF ASKED TO EXTEND — MYXOEDEMA COMA

If the examiner escalates the scenario to **hypothermia, hypoventilation, hyponatraemia, bradycardia and reduced consciousness**, that is **myxoedema coma**: an intensive-care emergency in which you give **intravenous levothyroxine (a loading dose, then daily), intravenous hydrocortisone before thyroid hormone until adrenal insufficiency is excluded**, passive rewarming, ventilatory and haemodynamic support, cautious fluid and sodium correction, and treatment of the precipitant — usually **infection, cold exposure, a sedative drug, or omission of levothyroxine**. Mortality remains high.

QUESTION 27 · THYROID

Hashimoto's thyroiditis

7-Mark Note — 6 textbook points + 1 latest update = 7 points

TEXTBOOK CORE — 6 POINTS

1. **Definition and importance in old age.** Hashimoto's thyroiditis is **chronic autoimmune (lymphocytic) thyroiditis** — the classic organ-specific autoimmune disease, in which lymphocytic infiltration and antibody-mediated destruction of thyroid follicular cells produce progressive gland failure. **The most frequent cause of hypothyroidism in old age is autoimmune thyroiditis**, followed by earlier thyroid surgery or radiation therapy for previous thyrotoxicosis.
2. **Why it is commoner with ageing — the immunological basis. With ageing there is a decrease in T-suppressor lymphocytes and an increase in autoantibodies.** Since many endocrine disorders are autoimmune, **these changes amplify the possibility of an older person developing hypoendocrine disease in old age**, and there is **an increased likelihood of developing autoimmune polyglandular failure syndromes**. A caution the textbook adds: **ageing is frequently associated with an increased prevalence of thyroid autoantibodies, which may be an expression of age-associated diseases rather than a consequence of the ageing process itself** — so a positive antibody in an 80-year-old is not automatically clinically significant.
3. **Pathology.** Diffuse **lymphocytic and plasma cell infiltration with germinal centre formation**, **Hürthle (Askanazy) cell change** of follicular epithelium, follicular atrophy and **fibrosis**. Two clinical forms: the **goitrous** variant with a firm, bosselated, painless gland, and the **atrophic** variant — commoner in the elderly — with a small, impalpable gland. Antibodies are directed against **thyroid peroxidase (anti-TPO, the most sensitive)** and **thyroglobulin**; blocking TSH-receptor antibodies contribute to the atrophic form.
4. **Clinical spectrum.** May be **euthyroid with goitre or antibodies alone**, **subclinically hypothyroid**, or **overtly hypothyroid**. An early transient thyrotoxic phase — **"Hashitoxicosis"** — occurs from destructive hormone release. In older people the presentation is atypical and insidious: **memory loss, lethargy, constipation, cold intolerance, fatigue, congestive heart failure, depression and weight gain, all often attributed to old age or other causes**. **Lack of these symptoms does not rule out hypothyroidism, so a high index of suspicion is needed.** Associated laboratory findings include a **raised creatine phosphokinase** and a **normocytic normochromic anaemia which responds to thyroid hormone replacement**; there is an **increased incidence of pernicious anaemia**, though **iron deficiency remains the most common anaemia associated with hypothyroidism**.
5. **Diagnosis and prognostic value of antibodies. An elevated serum TSH level should be confirmed and supplemented with measurements of serum thyroxine (T4) and with thyroid antibodies.** Ultrasound shows a **hypoechoic, heterogeneous gland with increased vascularity** — useful but not required. In

subclinical disease, **antithyroid antibodies are useful for improving the prognosis of progression to overt hypothyroidism, but they may not change the management of the patient**: progression is **generally around 5% per year and greater in those with goitre or thyroid antibodies, or both**.

6. **Associations, complications and treatment. Associations:** other autoimmune disease — **pernicious anaemia, vitiligo, Addison's disease, type 1 diabetes, coeliac disease, rheumatoid arthritis, Sjögren's syndrome** — as part of **autoimmune polyglandular failure**. **Complications:** overt hypothyroidism and, rarely, **myxoedema coma**; compressive symptoms from a large goitre; **encephalopathy associated with autoimmune thyroiditis (Hashimoto's encephalopathy)**, a steroid-responsive cause of subacute confusion, seizures and myoclonus that is a genuinely reversible dementia mimic in the elderly; and the important association with **primary thyroid lymphoma** — **lymphoma of the thyroid and undifferentiated cancers occur with increasing frequency in the elderly**, so a rapidly enlarging gland in known Hashimoto's demands urgent biopsy. **Treatment:** levothyroxine for overt disease, **started at 25–50 µg daily and increased slowly every six to eight weeks**, at a **lower maintenance dose than in younger people**; no immunosuppression is indicated; euthyroid antibody-positive patients need **surveillance only, with annual TSH**.

LATEST UPDATE (2023–PRESENT) — 1 POINT

7. **Two current themes are worth a sentence each. (a) Immune checkpoint inhibitor–induced thyroiditis** is now a common iatrogenic cause of autoimmune thyroid destruction, and older adults receive an increasing share of immunotherapy: it typically produces a **transient thyrotoxic phase within the first weeks followed by permanent hypothyroidism**, is managed with **levothyroxine without stopping the immunotherapy**, and requires **thyroid function monitoring before each cycle** — it is the modern differential for new autoimmune hypothyroidism in an older cancer patient. **(b) Selenium supplementation**, long promoted to lower anti-TPO titres, has **not been shown to alter progression to overt hypothyroidism or improve quality of life** and is **not recommended**. The wider position — reinforced by the TRUST trial and current guidance — is that **antibody positivity in an older adult justifies surveillance, not treatment**: it refines the prognosis without changing management until TSH crosses the treatment threshold.

EXAM ONE-LINERS

- **Commonest cause of hypothyroidism in old age = autoimmune (Hashimoto's) thyroiditis.**
- Ageing → **fewer T-suppressor cells, more autoantibodies** → more autoimmune endocrine failure.
- **Anti-TPO** is the most sensitive antibody; it predicts progression but does not change management.
- Progression to overt disease **~5% per year**, higher with goitre or antibodies.
- Rapidly enlarging gland in known Hashimoto's = **primary thyroid lymphoma until proven otherwise**.
- **Hashimoto's encephalopathy** — a steroid-responsive, reversible cause of confusion in the elderly.
- Raised **CPK** plus normocytic anaemia in a tired older patient — check the TSH.

EXAMINER TRAP

Treating **antibody positivity itself**. A euthyroid older adult with positive anti-TPO needs an **annual TSH**, not levothyroxine. Equally, do not dismiss the antibodies as meaningless — they identify the patient who *will* need treatment, and they raise the index of suspicion for **other autoimmune disease and for thyroid lymphoma**.

QUESTION 28 · THYROID

Hyperthyroidism in the elderly — presentation, investigation and treatment

7-Mark Note — 6 textbook points + 1 latest update = 7 points

TEXTBOOK CORE — 6 POINTS

1. **Definition and causes.** Overt hyperthyroidism is characterised by **high levels of thyroid hormones with a low TSH**. The most common causes in old age are **Graves' disease in iodine-sufficient areas** and **nodular goitre (Plummer's disease or multinodular toxic goitre) and functioning follicular adenoma in iodine-deficient areas**. **Toxic multinodular goitre is more frequent in older persons than in the young. Transient thyrotoxicosis** occurs in **subacute thyroiditis** and during treatment with **amiodarone** (particularly in iodine-deficient areas), which may represent a major therapeutic challenge; thyrotoxicosis during levothyroxine or interferon treatment is self-limited and remits on drug withdrawal.
2. **Presentation is paucisymptomatic and atypical — the highest-yield point.** Whereas younger patients have multiple emblematic symptoms, **the older patient may have few symptoms and they are frequently atypical**. Patients **lack the hyperdynamic symptomatology — tremor, heat intolerance, ocular signs, nervousness or tachycardia — and instead have a more sedated, apathetic presentation**. However, **the frequency of atrial fibrillation is higher**. **Weight loss and cardiac symptoms frequently predominate, and goitre is frequently absent, making the diagnosis less obvious. Approximately one-third have no goitre. Depression and mania can be manifestations of hyperthyroidism in older adults.**
3. **Apathetic thyrotoxicosis and the masked presentation. Apathetic thyrotoxicosis is due in part to the decreased post-receptor responsiveness of β-adrenergic receptors that occurs with ageing.** Its classical features are **depression, weight loss, atrial fibrillation, heart failure, blepharoptosis and proximal myopathy**, and it occurs in **about 7% of older people with hyperthyroidism**. **Severe non-thyroidal disease may disguise thyrotoxicosis; congestive heart failure, stroke and infection are common disorders associated with masked hyperthyroidism.** Suspect it in **unexplained heart failure or tachyarrhythmia, recent-onset psychiatric disorder, or profound myopathy**. The triad of **weight loss, anorexia and constipation — which may raise the possibility of neoplastic disease — occurs in 15% of older thyrotoxic patients**.
4. **Investigation. Ultrasensitive TSH assays can differentiate hyperthyroidism from normal, and in the absence of acute non-thyroidal disease this test alone may confirm the clinical diagnosis. In the presence of acute illness, concomitant determination of TSH and free T4 is more appropriate.** Then free T3 (for T3 toxicosis), **TRAb** for Graves' disease, and a **radioisotope uptake scan** to separate Graves' (diffuse uptake), toxic nodular goitre (patchy or focal) and thyroiditis or exogenous hormone (low uptake). Also ECG for atrial fibrillation, liver function and full blood count before antithyroid drugs, and bone densitometry where relevant. **A T3 suppression test should not be done in older adults because of the risk of angina or myocardial infarction.**
5. **Treatment — radioiodine is the treatment of choice. Treatment of older patients with hyperthyroidism includes antithyroid drugs and radioactive iodine. ^{131}I is the treatment of choice, especially when concomitant cardiovascular diseases are present**, while **surgery may be considered if obstructive symptoms caused by large goitres are present, even though it is rarely indicated owing to the high operative risk in older patients with comorbidity** — though **radioiodine may also be successfully used in large compressive goitres**. **Antithyroid drugs (methimazole/carbimazole or propylthiouracil) may be the treatment of choice for controlling hyperfunction before definitive treatment with ^{131}I.** A crucial closing line: **treatment of an underactive thyroid after definitive treatment with ^{131}I may be simpler than a recurrence of hyperthyroidism in an older patient** — that is, accept hypothyroidism as a planned outcome.

6. **Adjunctive treatment and the special situations. Beta-adrenergic blockers (propranolol, metoprolol) may be used with prudence, and when needed the dosage should be the lowest possible; treatment with beta blockers may prevent symptoms of thyrotoxicosis following ^{131}I treatment. In general, atrial fibrillation reverts to sinus rhythm within a few months after the restoration of euthyroidism** — so do not rush to cardiovert, but do anticoagulate according to stroke risk. For **severe thyrotoxicosis or thyroid storm**: antithyroid drugs, **preferably propylthiouracil because it blocks peripheral conversion of T4 to T3**, plus **iodides to block thyroid hormone secretion** (given after the thionamide) and beta blockers. Where beta blockers are contraindicated or the patient is allergic to antithyroid drugs, **calcium ipodate 3 g every 3 days** inhibits peripheral T4-to-T3 conversion, and **dexamethasone 2 mg every 6 hours** does the same and may be added to any of these regimens.

Feature	Young patient	Older patient
Adrenergic symptoms	Tremor, heat intolerance, nervousness, tachycardia	**Often absent — apathetic, sedated presentation**
Goitre	Usually present	**Absent in about one-third**
Eye signs	Common in Graves'	Uncommon; **blepharoptosis** rather than lid retraction
Atrial fibrillation	Less common	**Markedly more frequent**
Dominant complaint	Weight loss with increased appetite	**Weight loss, anorexia, constipation — mimics malignancy (15%)**
Psychiatric	Anxiety, agitation	**Depression, mania, apathy**
Muscle	Mild weakness	**Profound proximal myopathy**
First-line definitive treatment	Antithyroid drugs, then choice	**Radioactive iodine (^{131}I)**

LATEST UPDATE (2023–PRESENT) — 1 POINT

7. **Long-term low-dose antithyroid drug therapy has become an accepted alternative to definitive treatment, and a large network meta-analysis has now compared the three modalities head to head.** Revised American Thyroid Association guidance reflects **less of a push towards definitive therapy**: whereas antithyroid drug duration was previously limited on safety grounds, **it is now accepted to remain on antithyroid drugs long term**, with methimazole at **2.5–10 mg/day continued for many years shown to be safe and effective**, and prolonged low-dose therapy producing **better outcomes and fewer side effects than radioiodine** in relapsed Graves' disease. A **2025 systematic review and network meta-analysis in *Thyroid* examined the long-term mortality risks of antithyroid drugs, radioiodine and surgery**. Practical relevance for geriatrics: in a frail older patient with toxic multinodular goitre, radioiodine remains preferable because the disease does not remit; but in Graves' disease, **continuing low-dose carbimazole with six-monthly thyroid function tests is a legitimate, low-burden option** that avoids radiation precautions, the risk of transient post-radioiodine thyrotoxic exacerbation, and the near-certain need for lifelong levothyroxine. Always warn about **agranulocytosis** — stop the drug and check a full blood count urgently for fever or sore throat — and about hepatotoxicity.

MEMORY ANCHORS & 15-SECOND SKELETON

Skeleton: High T4/T3 with low TSH → Graves' or toxic nodular goitre (commoner in the old) → **apathetic, paucisymptomatic presentation, AF, weight loss, no goitre in one-third** → TSH first, then free T4/T3, TRAb, uptake scan; no T3 suppression test → **^{131}I is the treatment of choice**, antithyroid drugs to prepare, surgery rarely → beta blocker at lowest dose, AF usually reverts, accept hypothyroidism.

Numbers: apathetic thyrotoxicosis **7%** · no goitre in **one-third** · weight loss/anorexia/constipation triad **15%** · calcium ipodate **3 g every 3 days** · dexamethasone **2 mg every 6 hours** · long-term methimazole **2.5–10 mg/day**.

EXAMINER TRAPS

- Describing the **young patient's presentation**. The examiner is testing whether you know that in old age it is **apathetic, cardiac and paucisymptomatic**.
- Excluding hyperthyroidism because there is **no goitre and no eye signs**.
- Ordering a **T3 suppression test** — contraindicated in older adults.
- Missing **amiodarone-induced thyrotoxicosis**, which is a genuine therapeutic challenge and needs distinction between type 1 (iodine-induced excess synthesis — thionamide) and type 2 (destructive thyroiditis — glucocorticoid).
- Interpreting a low TSH in an acutely ill older patient as thyrotoxicosis — **non-thyroidal illness, fasting and glucocorticoids must be excluded first**.

QUESTION 29 · ENDOCRINOLOGY

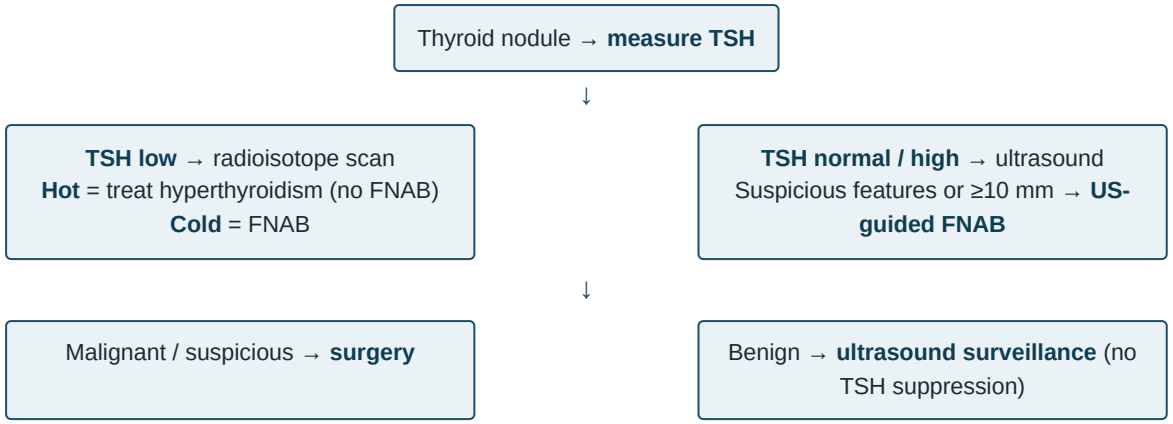
Solitary thyroid nodule and thyroid malignancy

7-Mark Answer — 6 textbook points + 1 latest update = 7 points

TEXTBOOK CORE — 6 POINTS

1. **Prevalence and baseline risk.** Thyroid nodules **occur with increasing frequency in older people** and about **95% are benign**. Palpable nodules are found in **3–7%** of the population, but prevalence rises to about **50% on ultrasound or at autopsy**. Crucially, **the risk of malignancy is the same for a solitary nodule as for a multinodular goitre** — so a multinodular gland does not reassure.
2. **Red flags requiring urgent referral.** A nodule that is **growing rapidly over a few weeks**, or is associated with **stridor, hoarseness or cervical lymphadenopathy**. Otherwise, routine evaluation is appropriate.
3. **Start with TSH, and let it direct the pathway.** If **TSH is low or suppressed**, perform a **radioisotope scan**: a **"hot" (autonomous) nodule** is treated as hyperthyroidism and **does not require FNAB**, whereas a **"cold" nodule requires FNAB**. If **TSH is normal**, proceed directly to **ultrasound and FNAB**. If TSH is raised with positive antithyroid antibodies and low free T4, treat with levothyroxine.
4. **FNAB is the cornerstone. Fine-needle aspiration biopsy is the most accurate method of evaluating a thyroid nodule** and determines who is referred for surgery; its **accuracy is improved by high-resolution ultrasound guidance**. FNAB is recommended for nodules **≥10 mm** with any of: **irregular margins, chaotic intranodular vascular spots, a shape more tall than wide, or microcalcifications**. For nodules **<10 mm**, FNAB only if clinical information or ultrasound features are suspicious.
5. **Limitations and risk stratification.** A significant limitation of FNAB is an **inadequate or indeterminate result in 10–25%** of cases, after which surgery is usually recommended for diagnosis — though the majority prove benign. **No ultrasound finding is diagnostic on its own.** EU-TIRADS malignancy risk: **category 2 benign ≈ 0%, 3 low risk 2–4%, 4 intermediate 6–17%, 5 high risk 26–87%**.

6. **Malignancy in old age, and what not to do.** Thyroid cancer in old age is **generally well differentiated, but its course is frequently less predictable than in younger patients**; **lymphoma of the thyroid and undifferentiated (anaplastic) cancers occur with increasing frequency in the elderly**. Malignant or suspicious nodules are treated **surgically**; radioiodine is first choice for thyroid autonomy and hyperthyroidism; surgery is advised for large non-toxic goitres with compressive symptoms. **Routine TSH-suppressive levothyroxine for benign nodules is NOT recommended** — the potential harm outweighs benefit, and in multinodular goitre it may produce **exogenous hyperthyroidism with cardiac and bone adverse effects**.



LATEST UPDATE (2023–PRESENT) — 1 POINT

7. **Natural-history data have shifted practice towards active surveillance rather than intervention in the older adult.** In a prospective multicentre study of 992 patients with cytologically and sonographically benign nodules, **only 11.1% grew over five years, thyroid cancer was diagnosed in just 0.3%, and age ≥60 was associated with a LOWER risk of nodule growth than in younger participants**. Current practice is therefore **ultrasound at 12 months, then at progressively longer intervals of 2–5 years**, with **repeat FNA only for substantial growth (>50% change in volume or ≥20% increase in at least two dimensions), new suspicious ultrasound features, or new symptoms**. In parallel, **active surveillance of low-risk papillary microcarcinoma** is now an accepted alternative to immediate surgery in older adults, where the competing risk of death and the operative risk often outweigh the risk from the tumour.

EXAM ONE-LINERS

- **~95% benign**; malignancy risk is **the same** for solitary nodule and multinodular goitre.
- **First test = TSH**, not ultrasound and not FNAB.
- **Hot nodule = no FNAB.** Cold nodule = FNAB.
- FNAB = cornerstone; indeterminate in **10–25%**.
- Anaplastic carcinoma and **primary thyroid lymphoma** are diseases of the elderly — a rapidly enlarging, hard goitre with compressive symptoms.
- **Never** give suppressive levothyroxine for a benign nodule in an older person.

EXAMINER TRAP

A calcified large goitre in an older woman can be **misdiagnosed as cancer metastatic to lymph nodes** — FNAB is recommended to determine the nature of calcified lesions. Also remember that **hyperkyphosis and postural change from osteoporosis complicate the physical examination**: if you can palpate the thyroid in an older woman, it is probably enlarged.

PART VI

Endocrine and Metabolic Bone Disease

Q30 · Q31

QUESTION 30 · ENDOCRINOLOGY

Testosterone replacement and andropause

7-Mark Answer — 6 textbook points + 1 latest update = 7 points

TEXTBOOK CORE — 6 POINTS

1. **"Andropause" does not exist.** Circulating testosterone declines with age, but **unlike menopause, men do not suffer a rapid, inevitable loss of gonadal function**. After age 30 free testosterone falls at about **1% per year**; in the European Male Ageing Study, between ages 40 and 79 **total testosterone fell 0.4% per year and free testosterone 1.3% per year**. The steeper fall in free testosterone is due to the **age-related rise in SHBG**. The preferred term is **late-onset hypogonadism**, whose prevalence is only about **2–3%**.
2. **Definition and classification.** Hypogonadism is a **combination of subnormal serum testosterone AND one or more symptoms attributable to it** — a biochemical value alone is not a diagnosis. It may be **primary** (testicular) or **secondary** (pituitary or hypothalamic). It is common, **underdiagnosed and often untreated**: only 5–35% of hypogonadal men receive treatment.
3. **Mechanism of the age-related fall.** Older men show a **decrease in episodic and stimulated gonadotropin secretion**, together with a **reduced ability of the testicular Leydig cell to respond to luteinising hormone** with increased testosterone production. Comorbidity contributes strongly — **obesity, type 2 diabetes and chronic illness** all lower testosterone.
4. **Diagnosis — look for a correctable cause first. When faced with a symptomatic decrease in circulating testosterone, one should look first for a correctable cause before attributing the decrease to age alone.** Take **two morning fasting total testosterone measurements**; measure **free testosterone** when SHBG is altered (obesity, ageing, thyroid disease, liver disease). Then measure LH and FSH to separate primary from secondary, and image the pituitary if secondary.
5. **Benefits of replacement.** Testosterone replacement at relatively high doses in older hypogonadal men **increases muscle mass, strength, functional status and bone mineral density**, and improves **erythropoiesis**. It **increases libido in both sexes**, and in men increases **erectile strength, volume of ejaculation and visuospatial cognition**. In the **"T-Trials"** (790 symptomatic men over 65, testosterone gel versus placebo for one year), raising testosterone from low to moderate levels gave a **moderate benefit in sexual function and some benefit in mood and depressive symptoms, but NO benefit in vitality or walking distance**.
6. **Risks, contraindications and monitoring.** The **major side effect is an excessive increase in haematocrit**; also **gynaecomastia, water retention and at least temporary worsening of sleep apnoea**. The **major contraindication is prostate carcinoma**; effects on benign prostatic hypertrophy and prostate cancer are

unclear, and **long-term safety data are lacking**. **Control of prostate size, PSA and haematocrit is mandatory.** Do **not** replace testosterone in **asymptomatic older men with an age-related decline alone**; offer it on an **individualised basis** after explicit discussion. **Target a lower concentration in older men — 300–400 ng/dL rather than 500–600 ng/dL.** If symptoms and signs do not improve, **discontinue** and investigate other causes.

LATEST UPDATE (2023–PRESENT) — 1 POINT

7. **TRAVERSE trial (NEJM 2023) — the definitive cardiovascular safety study.** In **5,246 men aged 45–80** with hypogonadism and pre-existing or high risk of cardiovascular disease, randomised to transdermal testosterone gel or placebo for a mean of 27 months, **testosterone was non-inferior to placebo for the primary MACE endpoint** (cardiovascular death, non-fatal myocardial infarction, non-fatal stroke), and there was **no increase in prostate cancer or prostate-related events**. However, the testosterone arm had **more atrial fibrillation and other non-fatal arrhythmias requiring intervention, more acute kidney injury, more pulmonary embolism, and — unexpectedly — MORE fractures** despite the known gain in bone mineral density. **Interpretation for the examination:** TRAVERSE resolves the cardiovascular controversy that prompted the FDA's 2015 caution, but it is **not a licence to treat age-related testosterone decline** — the indication remains **symptomatic, biochemically confirmed hypogonadism**, and **fracture protection cannot be claimed**.

CURRENT PRACTICE UPDATE

Aspect	Textbook	Current Recommendation
Cardiovascular risk	Possible increase in events in men ≥65, especially in the first year (FDA caution)	TRAVERSE: non-inferior to placebo for MACE; new signals for atrial fibrillation, VTE and AKI
Bone	Increases bone mineral density; fracture data unavailable	TRAVERSE: fracture rate **higher** on testosterone — do not treat to prevent fracture
Prostate	Effects unclear; PSA monitoring mandatory	No increase in prostate cancer or prostate events; PSA monitoring still advised

Exam approach: State the textbook FDA caution, then TRAVERSE, then the conclusion — **cardiovascular reassurance without expansion of the indication**.

EXAM ONE-LINERS

- **"Andropause does not exist"** — open the answer with this sentence.
- Free T falls faster than total T because **SHBG rises with age**.
- Diagnosis needs **low testosterone PLUS symptoms** — never a number alone.
- Commonest adverse effect = **rising haematocrit**; absolute contraindication = **prostate carcinoma**.
- Target **300–400 ng/dL** in older men, not the young adult range.
- T-Trials: **sex and mood yes; vitality and walking distance no.**

QUESTION 31 · METABOLIC BONE DISEASE

Osteomalacia and vitamin D deficiency

15-Mark Answer — 10 textbook points + 2 latest updates = 12 points

TEXTBOOK CORE — 10 POINTS

1. **Definition and distinction.** Osteomalacia is **defective mineralisation of newly formed osteoid in the mature skeleton**, most often due to vitamin D deficiency; the childhood equivalent is **rickets**. Contrast with **osteoporosis**, in which mineralisation is normal but bone **quantity** is reduced. The two commonly **coexist** in older adults.
2. **Vitamin D falls with ageing.** Longitudinal data show vitamin D **declines with age**, and deficiency is **associated with increased mortality**. **Old skin exposed to ultraviolet light is less effective than young skin at manufacturing cholecalciferol**, so sun exposure alone corrects deficiency less reliably in the elderly.
3. **Causes in older adults. Reduced sun exposure** — housebound, institutionalised, fully covered clothing, urban living, skin pigmentation (all highly relevant in India); **poor dietary intake**; **malabsorption** — coeliac disease, post-gastrectomy, bariatric surgery, pancreatic insufficiency; **chronic kidney disease** with reduced 1α-hydroxylation; liver disease; and **drugs** — anticonvulsants, rifampicin, glucocorticoids. Non-vitamin-D causes include **hypophosphataemia, renal tubular acidosis** and **tumour-induced osteomalacia**.
4. **Clinical features. Diffuse bone pain and bony tenderness** — pelvis, ribs, spine; **proximal myopathy** with a **waddling gait and difficulty rising from a chair**; **falls**; and **fragility fractures**. In older people this is routinely misdiagnosed as "just ageing", fibromyalgia, polymyalgia rheumatica or metastatic bone disease.
5. **The key biochemical clue. Vitamin D deficiency should be suspected in any older person with a borderline low calcium level and an elevated alkaline phosphatase.** The full biochemical picture is: **calcium low or low-normal (<8.5 mg/dL), phosphate low, alkaline phosphatase raised, PTH raised (secondary hyperparathyroidism), and 25(OH)D low**.
6. **Thresholds. Deficiency <20 ng/mL, insufficiency 20–30 ng/mL, sufficiency ≥30 ng/mL.** The textbook position is that **ideally vitamin D levels should be 30 ng/mL or greater**, matching the International Osteoporosis Foundation target, whereas the Institute of Medicine suggests **20 ng/mL**; the threshold concentration associated with clinically relevant disease events is **near 20 ng/mL**.
7. **Radiology.** Generalised osteopenia with blurred, indistinct trabeculae; **Looser's zones (pseudofractures)** — radiolucent bands perpendicular to the cortex at the **medial femoral neck, pubic rami, scapula and ribs**; "codfish" biconcave vertebrae; and, in severe disease, a triradiate pelvis. **Bone biopsy with tetracycline double labelling is the gold standard** but is rarely needed in practice.
8. **Why vitamin D matters beyond bone. In bone, vitamin D stimulates bone turnover while protecting osteoblasts from apoptosis; in muscle it maintains the function of type II fibres, preserving strength and preventing falls.** In the Cardiovascular Health Study, vitamin D deficiency was associated with **poorer physical performance, lower muscle strength and prevalent mobility and ADL disability**. Both osteoporosis and sarcopenia have been linked to the development of **frailty**.
9. **Treatment.** Replete with **cholecalciferol**: a loading phase — the common Indian regimen is **60,000 IU weekly for 6–8 weeks** — followed by maintenance of **800–2000 IU daily**, together with **elemental calcium 1000–1200 mg/day** and adequate dietary protein. Recheck **calcium, phosphate, alkaline phosphatase and 25(OH)D at about 3 months**; alkaline phosphatase falls last, as healing completes. **Always correct vitamin D deficiency BEFORE starting a bisphosphonate or denosumab**, or severe hypocalcaemia may follow.
10. **Evidence for benefit and dosing.** There is **clear evidence that vitamin D 800 IU together with calcium decreases the rate of hip fracture in older people**, and this is associated with a **decline in mortality**. **Vitamin D replacement in people who have clear vitamin D deficiency improves muscle strength and decreases**

falls. Older people should get **regular skin exposure of 15–30 minutes per day without sunblock, or take 800–1000 IU of vitamin D per day** — this is especially true for **residents of long-term care facilities**, who require at least **800 IU per day**, double the usual recommended daily requirement.

Parameter	Osteomalacia	Osteoporosis	Primary hyperparathyroidism
Serum calcium	Low or low-normal	Normal	**High**
Serum phosphate	Low	Normal	Low
Alkaline phosphatase	**Raised**	Normal	Raised
PTH	Raised (secondary)	Normal	**Raised (primary)**
25(OH)D	**Low**	Variable	Normal or low
Pain	Diffuse bone pain, tenderness	Painless until fracture	Bones, stones, groans
Muscle	**Proximal myopathy**	Normal	Weakness, fatigue
Radiology	**Looser's zones**	Low BMD, wedge fractures	Subperiosteal resorption, brown tumours

LATEST UPDATES (2023–PRESENT) — 2 POINTS

11. **Endocrine Society 2024 guideline "Vitamin D for the Prevention of Disease" — a major change in the testing paradigm.** The panel suggests **empiric daily vitamin D supplementation for everyone aged 75 years and older**, because of its **potential to lower mortality**, and suggests **AGAINST empiric supplementation above the dietary reference intake in healthy adults younger than 75**. It also suggests **against routine 25(OH)D testing in all the populations considered**, because no clinical trial evidence supports screening and no optimal target level could be defined — a clear departure from the 2011 guideline's 30 ng/mL target. For non-pregnant people over 50 in whom vitamin D is indicated, **daily low-dose administration is preferred to intermittent high doses**. **Critical distinction for the exam:** this guideline addresses **disease prevention in generally healthy people** — it does **not** apply to the diagnosis or treatment of **established osteomalacia, CKD, malabsorption or hypocalcaemia**, where measuring 25(OH)D remains essential.
12. **VITAL trial and the Indian public-health position.** In **VITAL** (25,871 participants, cholecalciferol 2000 IU/day), **supplementation did not lower the incidence of invasive cancer or cardiovascular events compared with placebo** — but **only 12.7% of participants had a 25(OH)D below 20 ng/mL**, so the results **cannot be applied to genuinely deficient people**. This is the key to reconciling "vitamin D does nothing" trial headlines with the real benefit seen in deficiency. In India, where vitamin D deficiency is **highly prevalent across all age groups**, the pragmatic approach endorsed by **ICMR-NIN** is **food fortification plus routine supplementation of at-risk groups — the housebound, institutionalised and older adults — rather than population-wide testing**, which is expensive and does not change management.

CURRENT PRACTICE UPDATE

Aspect	Textbook	Current Recommendation
Testing 25(OH)D	Measure; target ≥30 ng/mL	Endocrine Society 2024 advises against routine testing in healthy people; no target level defined. Testing retained for suspected osteomalacia, CKD, malabsorption, hypocalcaemia
Who to supplement	All older adults, 800–1000 IU/day	Empiric supplementation for **all aged ≥75**; against routine supplementation above DRI in healthy adults <75
Dosing schedule	Daily or intermittent high dose	**Daily low dose preferred** over intermittent high dose in those >50

Exam approach: Present the textbook thresholds for **diagnosis of osteomalacia**, then state the 2024 shift for **prevention in the healthy**. Making that distinction explicitly is what earns the top mark here.

MEMORY ANCHORS & 15-SECOND SKELETON

Skeleton: Define (defective mineralisation of osteoid) → contrast with osteoporosis → causes (sun, diet, malabsorption, CKD, drugs) → bone pain + proximal myopathy + falls → **low Ca, low PO_4, high ALP, high PTH, low 25(OH)D** → Looser's zones → treat with cholecalciferol loading then maintenance + calcium → recheck at 3 months → prevention 800 IU/day → Endocrine Society 2024 and VITAL.

Numbers: 20 / 30 ng/mL · 800 IU (fracture reduction) · 60,000 IU weekly × 6–8 weeks (Indian loading) · calcium 1000–1200 mg/day · sun 15–30 min/day · age **75** (Endocrine Society empiric supplementation cut-off).

EXAMINER TRAPS

- Calling a raised alkaline phosphatase "hepatic" without checking calcium, phosphate and 25(OH)D — this is **the** missed diagnosis of osteomalacia.
- Starting a bisphosphonate or denosumab before repleting vitamin D → **severe hypocalcaemia**.
- Quoting VITAL as proof that vitamin D "does not work" without noting that **only 12.7% of participants were deficient**.
- Confusing the **prevention** guideline (no testing) with the **diagnosis** of osteomalacia (test).

PART VII

Crystal Arthropathy

Q32 · Q33 · Q34

QUESTION 32 · CRYSTAL ARTHROPATHY — CLINICAL SCENARIO

A 65-year-old man presents with acute onset of severe pain in the right big toe for two days following a binge of alcohol. Most probable diagnosis and management

7-Mark Note — 6 textbook points + 1 latest update = 7 points

TEXTBOOK CORE — 6 POINTS

1. **The diagnosis is acute gout — podagra of the first metatarsophalangeal joint. Gout is characterised by intra-articular monosodium urate crystals** and **generally presents as acute, affecting the first metatarsophalangeal joint, mid foot or ankle**, although the knee, elbow or wrist can also be involved. The features that make it near-certain here are the **abrupt onset reaching maximum severity within 6–24 hours**, the **site**, the **male sex and age**, and the **alcohol binge**. Beer and spirits raise urate by **increasing purine load and by lactate-mediated inhibition of renal urate excretion**; dehydration after a binge concentrates urate further.
2. **Examine and exclude the one diagnosis that would kill him.** The joint will be **exquisitely tender, hot, red and swollen with overlying desquamation**, and even bedclothes may be intolerable. Look for **tophi — subcutaneous urate deposits on extensor surfaces**, which **can develop in later phases and are sometimes confused with rheumatoid arthritis and its nodules**. **The critical differential is septic arthritis**, which can be indistinguishable and may coexist; also consider **pseudogout, cellulitis, trauma or fracture, reactive arthritis and acute osteoarthritic flare**. Fever, rigors, a portal of entry, immunosuppression, diabetes or a prosthetic joint mandate urgent aspiration.
3. **Investigations. The definitive diagnosis of gout is established by observing the presence of needle-shaped crystals in the involved joint** — **negatively birefringent under polarised light, and intracellular within neutrophils during an attack**. Send the same aspirate for **Gram stain, culture and cell count**. **Some patients will have elevated uric acid levels** — note the wording: **serum urate is often normal or low during an acute attack and does not confirm or exclude the diagnosis**; repeat it two weeks after the flare settles. **Radiographs may reveal well-defined gouty erosions in or around joints** — punched-out, with overhanging sclerotic margins and preserved joint space — but these are features of chronic disease. Also check **renal function, glucose, lipids, full blood count and liver function**, both to find comorbidity and to choose the safest drug.
4. **Treat the acute attack — the choice is dictated by comorbidity, not by efficacy. The goal of treatment is to terminate the acute attack and then to prevent future attacks by considering underlying causes. The acute phase should be managed using short-term NSAIDs, colchicine, corticotrophin or corticosteroids, and treatment choices should be based on patient comorbidities such as renal function and gastrointestinal disease.** All three first-line options are of **similar efficacy**, so choose on safety: **NSAIDs** —

effective but hazardous in the older adult with renal impairment, heart failure, hypertension, anticoagulation or peptic ulcer; **colchicine — low-dose regimens (1.2 mg then 0.6 mg one hour later, then 0.5–0.6 mg once or twice daily)** are as effective as and far better tolerated than the old high-dose regimen, but **reduce the dose in renal impairment and avoid with clarithromycin, ciclosporin, verapamil, diltiazem and statins** because of myopathy and marrow suppression; **corticosteroids — intra-articular injection is ideal for a single joint**, or oral prednisolone 30–35 mg daily for 5 days, and this is often the **safest choice in an older patient with chronic kidney disease**. Add rest, elevation and ice.

5. **Do not start urate-lowering therapy during the attack — but do plan it. Treatment to decrease uric acid levels should not be initiated during the acute phase, because drugs such as allopurinol or probenecid may exacerbate the acute attack** by mobilising crystals. **If the patient is already established on allopurinol, continue it** — never stop it during a flare. Plan to start urate-lowering therapy **2–4 weeks after the flare has fully settled**, under anti-inflammatory cover.
6. **Address the cause and the man, not just the toe.** Review **alcohol** — advise reduction and screen for alcohol-use disorder, since a "binge" in a 65-year-old may signal a wider problem. Review **drugs that raise urate**: **thiazide and loop diuretics, low-dose aspirin, ciclosporin, tacrolimus, pyrazinamide and ethambutol** — substituting a diuretic is often curative. Advise reduction of **red meat, organ meat, shellfish and fructose-sweetened drinks**, **adequate hydration**, and **weight loss**. Screen for the associated conditions that make gout a cardiovascular marker: **hypertension, chronic kidney disease, obesity, diabetes, dyslipidaemia and ischaemic heart disease**.

Feature	Acute gout	Septic arthritis	Pseudogout
Typical joint	**First MTP, midfoot, ankle**	Knee, hip; any joint	**Knee, wrist**
Onset	Peak in 6–24 hours	Hours to days, progressive	Subacute, over days
Systemic upset	Mild fever possible	**Fever, rigors, unwell**	Mild
Crystals	**Needle-shaped, negatively birefringent**	None	**Rhomboid, positively birefringent**
Synovial white cells	2,000–50,000/mm^3	**>50,000/mm^3, >90% neutrophils**	2,000–50,000/mm^3
Gram stain / culture	Negative	**Positive**	Negative
Radiograph	Punched-out erosions with overhanging edge	Early normal, later destruction	**Chondrocalcinosis**

LATEST UPDATE (2023–PRESENT) — 1 POINT

7. **Current guidance has reversed the old rule that urate-lowering therapy must never be started during or soon after a flare.** The **ACR gout guideline** and subsequent practice hold that **starting urate-lowering therapy during a flare does not increase or prolong symptoms as long as anti-inflammatory treatment is given concurrently**, and the ACR explicitly notes that **patients are most motivated for treatment during an exacerbation** — so the pragmatic modern approach in a patient likely to be lost to follow-up is to **start allopurinol at a low dose under colchicine or NSAID cover rather than wait**. Two further current points: **anti-inflammatory prophylaxis should be continued for 3–6 months rather than less than 3 months** after starting urate-lowering therapy, with ongoing evaluation; and **HLA-B*5801 testing should be offered before starting allopurinol in people of Southeast Asian or African descent**, in whom the allele — carried by about **7% of those of Southeast Asian descent** — predicts severe allopurinol hypersensitivity. The examination-safe formulation is to **state the textbook rule first, then note that current guidance permits concurrent initiation under anti-inflammatory cover**.

EXAMINER TRAPS

- Diagnosing gout on a **raised serum urate**. Urate is frequently **normal or low during an acute attack**; the diagnosis rests on crystals.
- Not **aspirating the joint**, or not sending it for culture. Septic arthritis and gout can coexist, and a missed septic joint destroys it within days.
- Prescribing an **NSAID reflexively** in a 65-year-old with unknown renal function.
- Using the **old high-dose colchicine regimen** until diarrhoea occurs — obsolete and dangerous.
- **Stopping allopurinol** in a patient already established on it.
- Treating the toe and ignoring the **alcohol, the diuretic and the cardiovascular risk profile**.

QUESTION 33 · CRYSTAL ARTHROPATHY

Management of gout

7-Mark Note — 6 textbook points + 1 latest update = 7 points

PIVOT FOR THIS ANSWER

Question 32 covers the **acute attack**. This answer is weighted towards the **whole disease** — the four phases, urate-lowering therapy, treat-to-target, and the special problems of gout in the older adult. Give acute management in a single point and spend the rest on long-term control.

TEXTBOOK CORE — 6 POINTS

1. **The two goals, and the four phases they map onto. The goal of treatment is to terminate the acute attack and then prevent future attacks by considering underlying causes — for example, considering hypouricaemic therapy.** Gout progresses through **asymptomatic hyperuricaemia → acute intermittent gout → the intercritical period → chronic tophaceous gout**, in which **tophi, which are subcutaneous urate deposits on extensor surfaces, develop in the later phases and are sometimes confused with rheumatoid arthritis and its nodules**. Management differs at each phase, and the fundamental principle is that **the acute attack is treated with anti-inflammatories and the disease is cured only by lowering urate**.

2. **Acute attack — summarised. The acute phase should be managed using short-term NSAIDs, colchicine, corticotrophin or corticosteroids, with the choice based on patient comorbidities such as renal function and gastrointestinal disease.** In older adults, **low-dose colchicine or corticosteroids (intra-articular for a single joint, oral for polyarticular disease) are usually safer than NSAIDs**. **Colchicine can be used for acute management as well as for prophylaxis.** Never stop existing urate-lowering therapy during a flare.
3. **Who needs urate-lowering therapy, and which drug.** Indications: **two or more flares per year, any tophus, radiographic erosion, chronic kidney disease stage 3 or worse, urolithiasis, or a very high serum urate**. **Allopurinol is the first-line urate-lowering therapy**, and **due to the renal changes associated with ageing, allopurinol is recommended over probenecid in older individuals** — probenecid is a uricosuric and loses efficacy as GFR falls, besides risking stones. **Febuxostat**, the other xanthine oxidase inhibitor, is the alternative in allopurinol intolerance, but **is limited by the increased cardiovascular and all-cause mortality seen in studies** (the CARES trial), so it is second-line. **An xanthine oxidase inhibitor is recommended over probenecid in CKD stage 3 or worse.**
4. **How to prescribe it — start low, titrate to target, cover with prophylaxis. Start at a low dose with subsequent titration to target rather than starting high** — allopurinol **under 100 mg/day, and lower still in CKD (50 mg)**; febuxostat **under 40 mg/day**. **Adjust the dose upward every 2–5 weeks according to the uric acid level**. **ACR guidance advises that the allopurinol dose can be raised above 300 mg/day even in patients with renal impairment, provided there is adequate monitoring** — the older practice of capping the dose by creatinine clearance left most patients under-treated. **Initiate concomitant anti-inflammatory prophylaxis — colchicine, an NSAID, or low-dose prednisolone — over no prophylaxis**, and **continue it for 3–6 months rather than less than 3 months**, since urate-lowering drugs precipitate attacks by mobilising crystal deposits.
5. **Treat-to-target, and when to stop prophylaxis. Titrate to a serum urate below 6 mg/dL** — this **decreases flares and increases treatment adherence** — and to **below 5 mg/dL in tophaceous gout**, because tophi dissolve only below the saturation point. **Once the target has been achieved and maintained for 6 months, discontinue colchicine prophylaxis, unless the patient has one or more tophi on clinical examination. Urate-lowering therapy itself is continued indefinitely** — continuing it is preferred over stopping even in patients who are flare-free and tophus-free. **Asymptomatic hyperuricaemia should not, as a general rule, be treated.**
6. **Non-pharmacological management and the geriatric considerations.** Address **alcohol (especially beer and spirits), red and organ meat, shellfish, and fructose-sweetened drinks**; encourage **weight loss, hydration, low-fat dairy and vitamin C**. **Adjust contributing medications such as diuretics** — substituting a thiazide, or switching to **losartan** (mildly uricosuric) or a calcium-channel blocker for hypertension, is one of the most effective single interventions in an older patient. Geriatric-specific issues: **gout in older adults is more often polyarticular, involves the upper limbs and small hand joints, is more frequent in women after the menopause, is strongly diuretic-associated, and presents with tophi earlier** — sometimes on osteoarthritic Heberden's and Bouchard's nodes, where it is easily mistaken for nodal osteoarthritis or rheumatoid arthritis. Monitor renal function, review the whole drug list, and remember that **carpal tunnel syndrome is common in older adults and can be confused with gout, rheumatoid arthritis or pseudogout**.

Drug	Class / mechanism	Dose in the older adult	Cautions
Allopurinol	Xanthine oxidase inhibitor	Start 50–100 mg daily; titrate every 2–5 weeks; may exceed 300 mg	**HLA-B*5801** hypersensitivity (test in Southeast Asian and African descent); rash, DRESS; interacts with azathioprine
Febuxostat	Xanthine oxidase inhibitor	Start <40 mg daily	**Second-line — cardiovascular and all-cause mortality signal (CARES)**; hepatotoxicity
Probenecid	Uricosuric	500 mg once or twice daily, titrated	**Avoid in the elderly and in CKD stage >3**; urolithiasis
Colchicine	Anti-inflammatory (tubulin)	Acute: 1.2 mg then 0.6 mg at 1 h. Prophylaxis: 0.5–0.6 mg once or twice daily	Reduce in renal and hepatic impairment; **myopathy and marrow suppression with statins, clarithromycin, verapamil, diltiazem, ciclosporin**
Corticosteroid	Anti-inflammatory	Intra-articular, or prednisolone 30–35 mg for 5 days	Hyperglycaemia, delirium, fluid retention, infection risk
Pegloticase	Recombinant uricase	Specialist use, IV	Refractory tophaceous gout only; infusion reactions, anaphylaxis; **contraindicated in G6PD deficiency**

LATEST UPDATE (2023–PRESENT) — 1 POINT

7. **The two current shifts are treat-to-target with early initiation, and the head-to-head vindication of allopurinol.** The **ACR guideline's central change was the emphasis on a "treat-to-target" strategy — treating as aggressively as needed to keep serum urate below 6 mg/dL**, supported by trials published since the 2012 guideline. Alongside this, **urate-lowering therapy may now be started during a flare under anti-inflammatory cover**, capitalising on the moment the patient is most motivated. On drug choice, the **STOP-Gout trial (NEJM Evidence 2022)** found **allopurinol non-inferior to febuxostat** when both were properly dose-escalated to target, including in patients with chronic kidney disease — confirming that most "allopurinol failure" is in fact **under-dosing rather than drug failure**. The **FAST trial (Lancet 2020)** did not replicate the CARES cardiovascular mortality signal for febuxostat, so the position is now that **febuxostat is a reasonable alternative but allopurinol, correctly titrated, remains first choice**. Finally, gout is increasingly framed as a **cardiovascular risk marker**, with guideline-concordant therapy improving endothelial function and reducing intercritical inflammation — a useful closing line in a geriatrics answer.

MEMORY ANCHORS & 15-SECOND SKELETON

Skeleton: Two goals and four phases → acute attack (NSAID / colchicine / steroid, chosen by comorbidity) → who needs ULT → **allopurinol first-line, preferred over probenecid in the elderly** → start low, titrate every 2–5 weeks, cover with prophylaxis 3–6 months → **target urate <6 mg/dL (<5 with tophi)** → stop prophylaxis after 6 months at target unless tophi → diet, alcohol, diuretics, weight → geriatric pattern: polyarticular, upper limb, women, diuretic-associated, early tophi.

Numbers: target urate **<6 mg/dL**, tophaceous **<5** · allopurinol start **<100 mg** (50 in CKD), titrate every **2–5 weeks** · febuxostat start **<40 mg** · prophylaxis **3–6 months** · stop prophylaxis after **6 months** at target · ULT indicated at **≥2 flares/year** · HLA-B*5801 in **7%** of Southeast Asians.

EXAMINER TRAPS

- **Treating asymptomatic hyperuricaemia.** As a general rule, do not.
- Starting allopurinol at **300 mg** and never titrating — the commonest real-world error, and the reason most patients never reach target.
- Capping the allopurinol dose by creatinine clearance and accepting failure. **The dose may be raised above 300 mg even in renal impairment, with monitoring.**
- Omitting **anti-inflammatory prophylaxis** when starting urate-lowering therapy, then blaming the drug when a flare follows.
- Choosing **probenecid in an older patient** — allopurinol is recommended over it because of age-related renal change.
- Missing the **diuretic**. In a hypertensive older patient with gout, changing the antihypertensive may achieve more than any urate-lowering drug.

QUESTION 34 · CRYSTAL ARTHROPATHY

Pseudogout

7-Mark Note — 6 textbook points + 1 latest update = 7 points

TEXTBOOK CORE — 6 POINTS

1. **Definition and nomenclature.** Pseudogout is **acute inflammatory arthritis caused by deposition of calcium pyrophosphate dihydrate (CPPD) crystals** in articular cartilage and fibrocartilage. The current terminology is **"acute CPP crystal arthritis"**, with **chondrocalcinosis** denoting the radiographic calcification and **CPPD disease** the umbrella term. **It is predominantly a disease of the elderly** — prevalence rises steeply after 65 and chondrocalcinosis is present in a substantial minority of those over 80.
2. **Causes and associations.** Most cases are **idiopathic and age-related**, but a secondary cause must be sought in anyone presenting young or with florid disease. The classic associations — mnemonic **"5 Hs"** — are **Hyperparathyroidism, Haemochromatosis, Hypophosphatasia, Hypomagnesaemia and Hypothyroidism**; add **Wilson's disease, gout, osteoarthritis, and previous joint trauma or meniscectomy**. Attacks are precipitated by **acute illness, surgery (especially parathyroidectomy), trauma, dehydration, myocardial infarction, stroke and intra-articular hyaluronate injection** — which is why pseudogout so often appears in an already unwell hospitalised older patient.
3. **Clinical presentations — it has more than one face. (a) Acute pseudogout** — a hot, swollen, painful joint, most often the **knee, then the wrist**, developing over days rather than hours, frequently with **fever and a raised white cell count and CRP**, so it convincingly mimics sepsis. **(b) Chronic CPP crystal inflammatory arthritis** — a symmetrical polyarthritis mimicking **rheumatoid arthritis** ("pseudo-rheumatoid"). **(c) Osteoarthritis with CPPD** — affecting unusual joints for osteoarthritis such as the **metacarpophalangeal joints, wrist, elbow and shoulder**. **(d) Asymptomatic chondrocalcinosis** — an incidental radiographic finding. **(e) Crowned dens syndrome** — CPPD around the odontoid peg presenting as acute neck pain, stiffness and fever, mimicking meningitis, polymyalgia rheumatica or giant cell arteritis in an older patient. In the elderly it also presents non-specifically as **immobility, delirium, a fall or "off legs"**.
4. **Diagnosis. Joint aspiration is essential**, both to identify crystals and to exclude infection. CPP crystals are **rhomboid or rod-shaped, weakly positively birefringent under polarised light** — the mirror image of the **needle-shaped, strongly negatively birefringent** urate crystals of gout. Synovial fluid is inflammatory with **2,000–50,000 white cells/mm^3**; always send **Gram stain and culture**. Radiographs show **chondrocalcinosis** — linear or punctate calcification of the **knee menisci and hyaline cartilage, the triangular fibrocartilage of the**

wrist, and the symphysis pubis — though its presence does not prove that CPPD is causing this attack. **Ultrasound** shows hyperechoic bands within hyaline cartilage. Screen for secondary causes: **calcium, phosphate, magnesium, alkaline phosphatase, PTH, ferritin and transferrin saturation, and TSH**.

5. **The critical differential remains septic arthritis, and it may coexist.** Pseudogout in an older adult reproduces every feature of sepsis — **fever, a hot swollen joint, leucocytosis and a high CRP** — and crystals in the fluid **do not exclude infection**. **Aspirate, culture, and if there is any doubt treat empirically for sepsis while awaiting results.** Other differentials: gout, acute osteoarthritic flare, haemarthrosis in an anticoagulated patient, cellulitis, reactive arthritis and, for the polyarticular form, rheumatoid arthritis and polymyalgia rheumatica. **Carpal tunnel syndrome is common in older adults and can be confused with gout, rheumatoid arthritis or pseudogout.**
6. **Management. There is no therapy that removes CPP crystals** — this is the essential contrast with gout, where urate-lowering therapy is curative. Treatment is therefore **anti-inflammatory and supportive**. **Acute attack: joint aspiration alone is often therapeutic**; then **intra-articular corticosteroid** for a single joint, which is the **preferred first choice in the older adult**; or **short-course NSAIDs with gastroprotection** if renal function, blood pressure and gastrointestinal history permit; or **low-dose colchicine**; or **oral prednisolone 30–35 mg for 5 days** for polyarticular disease. Add rest, ice and analgesia. **Prophylaxis** for recurrent attacks (three or more a year): **low-dose colchicine 0.5 mg once or twice daily**, dose-reduced for renal function. **Treat any identified secondary cause** — correcting **hypomagnesaemia** and treating **hyperparathyroidism, haemochromatosis or hypothyroidism**. Refractory chronic inflammatory disease may need **hydroxychloroquine, methotrexate or an IL-1 inhibitor** under specialist care. Physiotherapy and joint protection preserve function.

Feature	Gout	Pseudogout
Crystal	Monosodium urate	**Calcium pyrophosphate dihydrate**
Shape	**Needle-shaped**	**Rhomboid / rod-shaped**
Birefringence	**Strongly negative** (yellow parallel to the compensator)	**Weakly positive** (blue parallel to the compensator)
Typical joint	First MTP, midfoot, ankle	**Knee, wrist**
Speed of onset	Peak in 6–24 hours	Over several days
Sex and age	Middle-aged men; older women on diuretics	**Elderly, both sexes equally**
Radiograph	Punched-out erosions with overhanging edge	**Chondrocalcinosis**
Provoking factors	Alcohol, purine load, diuretics, dehydration	Acute illness, surgery, trauma, **parathyroidectomy**
Crystal-lowering therapy	**Yes — allopurinol, treat-to-target**	**None available**

LATEST UPDATE (2023–PRESENT) — 1 POINT

7. **CPPD disease now has formal ACR/EULAR classification criteria (2023) and a defined role for imaging and IL-1 blockade.** The **2023 ACR/EULAR classification criteria for calcium pyrophosphate deposition disease** were the first for this condition: entry requires at least one episode of joint swelling, tenderness and pain in a peripheral joint, the **presence of CPP crystals in synovial fluid is a sufficient criterion**, and in their absence a weighted score combines **clinical features, age, associated metabolic disease and — importantly — imaging evidence of chondrocalcinosis on radiograph, ultrasound or dual-energy CT**. The practical consequences are three. First, **ultrasound has become a validated, bedside, radiation-free way to demonstrate crystal deposition** — useful in a frail patient in whom aspiration is difficult. Second, the criteria formally recognise **crowned dens syndrome**, which matters because it is a treatable mimic of meningitis and of giant cell arteritis in older adults. Third, **IL-1 inhibition (anakinra)** is increasingly used for acute attacks in patients in whom NSAIDs, colchicine and steroids are all contraindicated — a situation that is common in multimorbid older patients with chronic kidney disease and heart failure. The unchanged core message remains: **there is still no crystal-dissolving therapy, so management is anti-inflammatory plus treatment of the underlying metabolic cause**.

MEMORY ANCHORS

The 5 Hs of secondary CPPD: Hyperparathyroidism · **H**aemochromatosis · **H**ypophosphatasia · **H**ypomagnesaemia · **H**ypothyroidism.

Birefringence mnemonic: "Pseudogout is **P**ositive and **P**arallelogram-shaped" · gout is **N**eedle-shaped and **N**egative.

EXAMINER TRAPS

- Assuming that **finding crystals excludes infection**. It does not; the two coexist, particularly in the elderly.
- Diagnosing pseudogout from **chondrocalcinosis on the radiograph alone** — it is common and asymptomatic in the very old.
- Forgetting to screen for the **5 Hs**, especially **hyperparathyroidism and haemochromatosis**, in anyone with florid or early-onset disease.
- Offering "urate-lowering therapy" for pseudogout — **no crystal-lowering therapy exists**.
- Missing **crowned dens syndrome** in an older patient with acute neck pain, fever and a raised CRP, and treating it as meningitis or giant cell arteritis.
- Overlooking a **recent parathyroidectomy** as the precipitant.

PART VIII

Environmental and Systemic Disorders

Q35 · Q36

QUESTION 35 · ENVIRONMENTAL

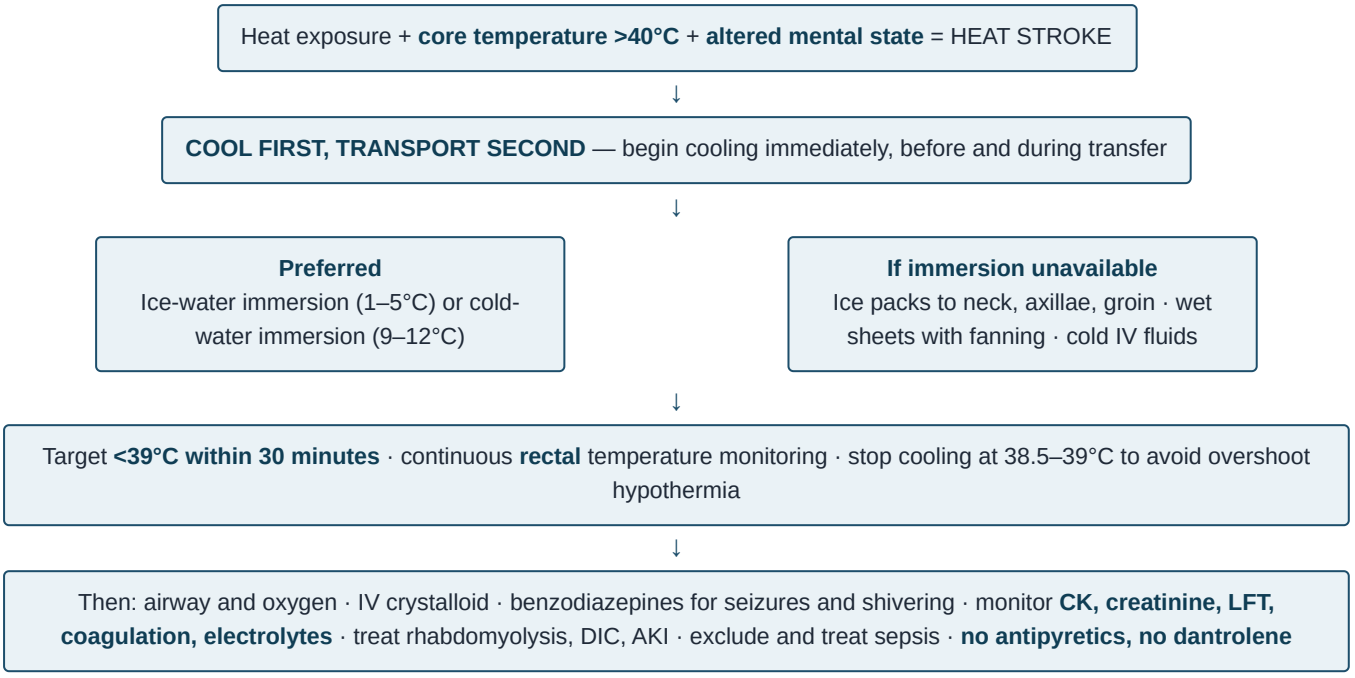
Heat stroke — clinical features, diagnostic criteria, differential diagnosis and treatment

7-Mark Note — 6 textbook points + 1 latest update = 7 points

TEXTBOOK CORE — 6 POINTS

1. **Definition and diagnostic criteria — the classic triad. Heat stroke is defined as a failure to maintain body temperature and is characterised by a core temperature above 40.6°C (105°F), severe central nervous system dysfunction — psychosis, delirium, coma — and anhidrosis with hot, dry skin. The two groups primarily affected are older adults who are chronically ill, and the young undergoing strenuous exercise** — that is, **classic (non-exertional)** and **exertional** heat stroke. **Mortality is as high as 80% once this syndrome is manifest.**
2. **Predisposing factors in older adults. There are multiple predisposing factors for heat stroke in older adults, but most often there is a prolonged heat wave.** Add the age-related and iatrogenic contributors: **reduced thirst and impaired water conservation, reduced sweating capacity, impaired cardiovascular compensation, immobility and dependence on carers, social isolation, dementia, absence of air conditioning**, and **drugs** — **anticholinergics and antihistamines (impair sweating), antipsychotics and antidepressants (impair thermoregulation), diuretics (volume depletion), beta blockers (blunt the cardiac response), and alcohol**. Chronic cardiac, respiratory, renal and neurological disease all raise the risk.
3. **Clinical features — early and late. The diagnosis requires a high level of suspicion. Early manifestations of heat exhaustion are non-specific: dizziness, weakness, a sensation of warmth, anorexia, nausea, vomiting, headache and dyspnoea. Later, severe central nervous system dysfunction and anhidrosis develop — psychosis, delirium, coma, and hot, dry skin.** In older adults the presentation is frequently just **confusion, a fall or collapse**, and the history of heat exposure must be sought actively.
4. **Differential diagnosis.** The differential is essentially **every cause of hyperthermia with altered mental state**: **sepsis and meningoencephalitis** (the commonest and most important alternative — and heat stroke may be complicated by infection); **neuroleptic malignant syndrome** (antipsychotics, lead-pipe rigidity, raised CK); **serotonin syndrome** (serotonergic drugs, clonus, hyperreflexia); **malignant hyperthermia** (anaesthetic exposure); **anticholinergic toxicity** (hot, dry skin, dilated pupils, urinary retention); **thyroid storm**; **drug withdrawal states**; **status epilepticus**; **cerebrovascular event affecting the hypothalamus**; and **stimulant or cocaine toxicity**. The distinguishing features are the **environmental history, the drug history and the muscle findings**.

5. **Complications — the multi-organ failure that determines outcome. Myocardial damage, congestive heart failure and arrhythmias; renal failure in 20–25%; cerebral oedema and seizures with diffuse and focal findings; hepatocellular necrosis, jaundice and liver failure; rhabdomyolysis with myoglobinuria; bleeding diathesis and disseminated intravascular coagulation; electrolyte disturbances; metabolic acidosis with respiratory alkalosis; infection including aspiration pneumonia and sepsis; and dehydration with shock. Once the full syndrome has developed for any length of time the prognosis is very poor.**
6. **Treatment — cool first, and cool fast. Management at this stage requires intense multisystem care, but the key is rapid specific therapy consisting of cooling to 38.9°C (102°F) within the first hour. Ice packs and ice-water immersion are superior to convection cooling with alcohol sponge baths or electric fans.** Alongside cooling: secure the airway, give **oxygen**, obtain a **rectal core temperature**, resuscitate with **intravenous crystalloid guided by cardiac status**, treat **seizures with benzodiazepines**, give **generous fluids and monitor CK and urine output for rhabdomyolysis**, correct electrolytes and coagulopathy, and treat infection. **Antipyretics — paracetamol and NSAIDs — are useless because the hypothalamic set point is normal, and are potentially harmful given hepatic and renal injury; dantrolene has no role. In view of the poor survival, efforts must be directed towards prevention: older patients should be cautioned about the dangers of hot weather, and for those at particularly high risk, temporary relocation to more protected environments should be considered. Prevention appears to be the most appropriate approach to management of temperature dysregulation in older adults** — through education about susceptibility, appropriate behaviour in extreme temperatures, and close monitoring of the most vulnerable.



LATEST UPDATE (2023–PRESENT) — 1 POINT

7. **The Society of Critical Care Medicine published the first formal Guideline for the Treatment of Heat Stroke (Crit Care Med 2025), and India's NCDC issued a national emergency cooling protocol (March 2024).** The SCCM guideline makes **active cooling recommended over passive cooling**, with **cold- or ice-water immersion achieving the fastest cooling rate and to be prioritised where available — ice-water immersion at 1–5°C or cold-water immersion at 9–12°C**. Its good-practice statements are precise and highly quotable: **clinicians may use similar strategies for either classic or exertional heat stroke**; **cooling methods should reach the target temperature within 30 minutes of recognition**; and **modalities achieving a cooling rate of at least 0.155°C per minute should be prioritised**. It also states that **there is no evidence to support pharmacological interventions affecting temperature control, so they should be avoided**. In India, the **NCDC/NPCCHH "Emergency Cooling for Severe Heat-Related Illnesses" (March 2024)** operationalises this for Indian emergency departments, including **improvised cold-water immersion using a body bag or tarpaulin** where a tub is unavailable, and the **NDMA Heat Action Plans** — with the **Ahmedabad Heat Action Plan** as the model, credited with a substantial reduction in heat mortality — provide the preventive framework: early-warning alerts, cool shelters, rescheduled outdoor work, and **active check-in on isolated older people during heatwaves and power cuts**, which is the single highest-yield preventive measure for this population.

CURRENT PRACTICE UPDATE

Aspect	Textbook	Current recommendation
Temperature threshold	>40.6°C (105°F) with anhidrosis	**>40°C with CNS dysfunction; anhidrosis is NOT required** — many patients are still sweating
Cooling target	38.9°C within the first hour	**<39°C within 30 minutes**, cooling rate ≥0.155°C/min
Method	Ice packs and ice-water immersion superior to fans	Confirmed — **cold/ice-water immersion is first choice for classic and exertional alike**
Drugs	Not addressed	**Avoid all pharmacological temperature control** — no antipyretics, no dantrolene

Exam approach: Quote the textbook triad, then state that **anhidrosis is no longer required for the diagnosis** — waiting for dry skin delays cooling and costs lives. This single correction is the highest-value line in the answer.

MEMORY ANCHORS

Skeleton: Define with the triad → two populations (classic elderly, exertional young) → predisposing factors and drugs → early non-specific then late CNS features → differential (sepsis, NMS, serotonin syndrome, anticholinergic, thyroid storm) → complications by organ system → **cool first, transport second**; immersion; target <39°C in 30 minutes → supportive care → prevention.

Numbers: core temperature **>40°C** (textbook 40.6°C / 105°F) · mortality up to **80%** · renal failure **20–25%** · cooling target **<39°C in 30 minutes** · cooling rate **≥0.155°C/min** · immersion water **1–5°C or 9–12°C**.

EXAMINER TRAPS

- Requiring **anhidrosis** before making the diagnosis. Many patients with classic heat stroke are still sweating — do not wait.
- Measuring an **oral, axillary or tympanic** temperature. Only a **rectal core temperature** is reliable.
- Giving **paracetamol**. The hypothalamic set point is normal, so it does not work, and it adds hepatotoxicity to a liver already injured.
- Investigating before cooling. **Cooling precedes CT, cooling precedes transfer.**
- Cooling past the target and producing **overshoot hypothermia** — stop at 38.5–39°C.
- Forgetting **rhabdomyolysis and DIC**, and forgetting that **sepsis may coexist**.
- Omitting **prevention**, which the textbook states is the most appropriate approach in older adults.

QUESTION 36 · SYSTEMIC

Chronic fatigue syndrome — diagnostic criteria, clinical manifestations, predisposing factors, course, prognosis and treatment

15-Mark Essay — 10 textbook points + 2 latest updates = 12 points

SOURCE NOTE

Chronic fatigue syndrome / myalgic encephalomyelitis is not covered adequately in the uploaded textbooks. Pathy's addresses *cancer-related fatigue* in detail and refers to *idiopathic chronic fatigue in older adults* being linked to impaired skeletal-muscle mitochondrial content and biogenesis signalling; Kane's addresses fatigue as a non-specific presentation. Those elements are used where they apply, and the diagnostic frameworks are taken from the **IOM/NAM 2015 criteria**, the **Fukuda 1994 CDC criteria** and **NICE NG206 (2021)**, each clearly named.

CORE ANSWER — 10 POINTS

1. **Definition.** Chronic fatigue syndrome, also called **myalgic encephalomyelitis (ME/CFS)** and, in the IOM formulation, **systemic exertion intolerance disease**, is a **chronic, disabling, multisystem illness** characterised by **profound fatigue that is not explained by ongoing exertion, is not relieved by rest, and causes a substantial reduction in previous levels of occupational, social and personal activity**, together with **post-exertional malaise** and **unrefreshing sleep**, persisting for **at least six months** (or, under NICE, three months for diagnosis) and not attributable to another disorder.
2. **Diagnostic criteria — IOM / National Academy of Medicine 2015.** Diagnosis requires **all three** of: **(i)** a **substantial reduction or impairment in the ability to engage in pre-illness levels of activity**, persisting more than six months, accompanied by fatigue that is **often profound, of new or definite onset, not the result of ongoing excessive exertion, and not substantially alleviated by rest**; **(ii) post-exertional malaise**; and **(iii) unrefreshing sleep**. **Plus at least one** of: **cognitive impairment** or **orthostatic intolerance**. Symptoms should be present for at least half the time and of at least moderate intensity.
3. **Diagnostic criteria — Fukuda (CDC) 1994, still widely quoted in examinations. Clinically evaluated, unexplained, persistent or relapsing fatigue for six months or more**, of new onset, not the result of ongoing exertion, not substantially alleviated by rest, and resulting in substantial reduction in previous activity levels — **plus four or more** of the following eight, concurrently present for six months: **impaired short-term memory or concentration; sore throat; tender cervical or axillary lymph nodes; muscle pain; multi-joint pain without swelling or redness; headaches of a new type, pattern or severity; unrefreshing sleep; and post-exertional malaise lasting more than 24 hours**.

4. **Post-exertional malaise is the cardinal feature — and the examinable one.** PEM is a **disproportionate worsening of symptoms after physical, cognitive, emotional or social exertion that was previously tolerated**, characteristically **delayed in onset by hours to days** and **disproportionately prolonged, taking days, weeks or longer to recover**. It is **not** ordinary post-exertional fatigue, and this distinction determines the entire management approach. **NICE NG206 makes post-exertional malaise mandatory for the diagnosis.**
5. **Clinical manifestations. Constitutional** — profound fatigue, PEM, unrefreshing sleep, sleep-phase disturbance. **Neurocognitive** — "brain fog", impaired concentration, word-finding and short-term memory difficulty, slowed information processing, sensory hypersensitivity to light and noise. **Autonomic** — **orthostatic intolerance, postural orthostatic tachycardia, palpitations, dizziness, nausea, temperature dysregulation**. **Musculoskeletal** — widespread myalgia, arthralgia without swelling, muscle weakness. **Immune-like** — recurrent sore throat, tender lymph nodes, flu-like malaise. **Gastrointestinal** — irritable bowel symptoms. Severity spans from those who continue reduced work to the **severely affected, who are housebound or bedbound and require assistance with all activities of daily living** — a group of about 25%.
6. **Predisposing, precipitating and perpetuating factors. Predisposing** — female sex (2–4:1), genetic susceptibility, pre-existing anxiety or depression, childhood adversity, and a personality pattern of high achievement and persistence. **Precipitating** — most characteristically an **infection**: Epstein–Barr virus, Q fever, Ross River virus, enterovirus, influenza, and now **SARS-CoV-2**; also major physical trauma, surgery, or severe psychological stress. **Perpetuating** — the **boom-and-bust cycle of over-activity followed by collapse**, deconditioning, disturbed sleep, chronic pain, comorbid mood disorder, and iatrogenic harm from inappropriate exercise prescription. Proposed mechanisms — none diagnostic — include **immune dysregulation with altered cytokine profiles, hypothalamic–pituitary–adrenal axis hypofunction, autonomic dysfunction, and impaired cellular energy metabolism**. This last is directly supported in the geriatric literature: **idiopathic chronic fatigue in older adults has been linked to impaired mitochondrial content and biogenesis signalling in skeletal muscle**.
7. **Diagnosis is clinical — investigations are to exclude, not to confirm. There is no diagnostic test.** A baseline screen excludes the treatable mimics: **full blood count, ESR and CRP, urea and electrolytes, creatinine, liver function, calcium, glucose and HbA1c, TSH, creatine kinase, ferritin, vitamin B12 and folate, vitamin D, coeliac serology, urinalysis**, and, where indicated, HIV, hepatitis serology, chest radiograph, ANA and cortisol. **NICE recommends baseline investigation to exclude alternative diagnoses and regular follow-up investigation to capture new or missed diagnoses.** The **differential in an older adult is wide and matters more than in the young**: **anaemia, hypothyroidism, diabetes, occult malignancy, heart failure, chronic kidney disease, COPD, obstructive sleep apnoea, polymyalgia rheumatica, depression, sarcopenia and frailty, undernutrition, and — above all — drug adverse effects** from beta blockers, opioids, antihistamines, benzodiazepines, antidepressants, statins and antihypertensives.
8. **The particular problem in older adults. ME/CFS characteristically begins between 20 and 50 and a new diagnosis after 65 should be made only with great caution**, because in that age group fatigue is far more likely to be the presenting feature of **occult disease, polypharmacy, depression or frailty**. In geriatric practice fatigue is therefore treated as a **syndrome requiring evaluation, not a diagnosis**. Where CFS/ME *is* genuinely present in an older person, it is usually **long-standing disease carried into later life**, in which case the clinical task is managing the **interaction of ME/CFS with accumulating comorbidity, deconditioning, sarcopenia and falls risk**. In the related and better-studied model of **cancer-related fatigue**, the textbook approach is instructive: it is a **multicomponent disturbance of neuroendocrine regulation and cytokine signalling**, and management begins with **a review of associated symptoms — pain, insomnia, depression — and a complete evaluation of possible medication side effects**, since fatigue-inducing drugs are so often the cause.
9. **Course and prognosis.** The course is **fluctuating, with relapses and remissions**, typically punctuated by PEM-driven "crashes". Onset may be **abrupt and post-infectious** or **insidious**. Prognosis is **generally poor for full recovery in adults**: **full recovery occurs in roughly 5–10%**, some degree of improvement in about **40%**, and the

remainder run a **chronic, fluctuating course**; children and adolescents do considerably better. **Favourable prognostic factors**: shorter duration before diagnosis, milder illness, absence of comorbid psychiatric disorder, a clear infective trigger, younger age, and good social and family support. **Adverse factors**: long duration, severe or bedbound disease, older age, comorbid depression or anxiety, chronic pain, ongoing pursuit of a purely psychological explanation, and — importantly — **iatrogenic deterioration from enforced exercise**. Mortality is not directly increased, but there is **excess suicide risk** in the severely affected, and profound impairment of quality of life.

10. **Treatment — there is no cure; management is supportive, individualised and multidisciplinary. (a) Energy management ("pacing")** is the cornerstone: the patient **establishes an individual "energy envelope" and stays within it, planning and prioritising activity and building in rest, rather than pushing through symptoms**. **(b) Sleep management** — sleep hygiene, treatment of a reversed sleep-wake cycle, and screening for obstructive sleep apnoea, without hypnotics where possible. **(c) Symptom-directed treatment** — analgesia for pain following the analgesic ladder with attention to constipation and delirium risk in the old; **low-dose amitriptyline or duloxetine** for pain and sleep; **increased fluid and salt, compression garments, and slow positional change** for orthostatic intolerance, with fludrocortisone or midodrine reserved for specialist use; management of irritable bowel symptoms; and treatment of coexisting depression or anxiety, which is a comorbidity to be treated in its own right and **not an explanation of the illness**. **(d) Psychological support** — **cognitive behavioural therapy may be offered as an adjunct to support coping and reduce the distress of chronic illness, but it is not a cure and must not be offered as one**. **(e) Rehabilitation and social care** — physiotherapy aimed at **maintaining, not increasing, function**; occupational therapy, aids, adaptations, home care, disability certification and carer support. **(f) Ruthless deprescribing** of fatigue-inducing drugs, and correction of anaemia, thyroid disease and nutritional deficiency. In the specific setting of **cancer-related fatigue**, where a pharmacological trial is contemplated, **methylphenidate is the best-studied agent and the usual first choice, with close monitoring**, although trials show a marked placebo response and benefit largely confined to those with severe fatigue.

Criterion set	Duration	Core requirements
Fukuda / CDC 1994	6 months	Unexplained persistent fatigue causing substantial reduction in activity, **plus 4 of 8** minor criteria
IOM / NAM 2015	6 months	**All 3**: activity reduction with profound fatigue · **post-exertional malaise** · unrefreshing sleep — **plus 1 of**: cognitive impairment *or* orthostatic intolerance
NICE NG206 (2021)	**3 months**	**All 4**: debilitating fatigue worsened by activity · **post-exertional malaise (mandatory)** · unrefreshing sleep or sleep disturbance · **cognitive difficulties**

LATEST UPDATES (2023–PRESENT) — 2 POINTS

11. **NICE NG206 reversed the previous standard of care, and the reversal has held.** The changes are the most examinable content in this topic. **Post-exertional malaise is now recognised as a core symptom and must be present for the diagnosis. ME/CFS can be confirmed after 3 months of symptoms rather than 4–6**, balanced by **baseline investigations to exclude alternative diagnoses and regular follow-up investigation to capture new or missed diagnoses. Graded exercise therapy is no longer recommended and should not be offered as a treatment for ME/CFS; people with ME/CFS should aim to remain within their energy limit and should not push through symptoms to complete tasks. Cognitive behavioural therapy is no longer considered a treatment for ME/CFS itself and should be used only as an adjunct** — the guideline states explicitly that CBT **"is not a cure for ME/CFS and should not be offered as such"**, but aims to improve wellbeing and quality of life and to reduce the distress of living with a chronic illness. **Individualised care should be provided by a multidisciplinary team.** The evidence base for graded exercise has continued to be criticised through 2025 for reliance on subjective self-report, poor recording of adverse effects, and biological data showing distinct immune and metabolic abnormalities in patients with PEM, which suggest that exercise may worsen symptoms.
12. **Long COVID has transformed the epidemiology and the research landscape.** A substantial proportion of people with long COVID meet ME/CFS criteria, so **prevalence has risen sharply** and the two conditions now share diagnostic and management pathways — **pacing and energy management have been adopted in long-COVID rehabilitation precisely because of the ME/CFS experience with graded exercise**. Two caveats to state for balance. First, **NICE itself notes that NG206 should not be assumed to apply to post-COVID-19 syndrome**, since its evidence was reviewed before the pandemic. Second, the relationship is not identical: in a comparative study, **self-reported PEM was common in long COVID (67% of one questionnaire cohort), but objectively observed PEM after standardised cardiopulmonary exercise testing was infrequent, and when present was less severe and less prolonged than in ME/CFS**. The practical geriatric implication is that **older adults with post-COVID fatigue should be assessed for PEM specifically**, since its presence dictates a pacing-based rather than a reconditioning-based rehabilitation plan — and getting this wrong causes harm.

CURRENT PRACTICE UPDATE

Aspect	Older teaching	Current recommendation (NICE NG206)
Graded exercise therapy	First-line evidence-based treatment	**Should not be offered** — risk of harm through PEM
Cognitive behavioural therapy	Curative treatment addressing illness beliefs	**Adjunct only**; explicitly "not a cure"
Duration for diagnosis	6 months (Fukuda)	**3 months**, with investigation to exclude mimics
Post-exertional malaise	One of eight minor criteria	**Mandatory core symptom**
Activity advice	Push through, increase gradually	**Stay within the energy envelope; do not push through**

Exam approach: Give Fukuda and IOM criteria for completeness, then state the NICE 2021 reversal explicitly. An answer that still recommends graded exercise therapy as first-line treatment reads as a decade out of date.

MEMORY ANCHORS & ESSAY SKELETON

Skeleton (15 marks, six blocks): (1) Define, name the three synonyms. (2) Criteria — Fukuda, IOM, NICE, in a table. (3) Clinical manifestations by system, and the severity spectrum. (4) Predisposing / precipitating / perpetuating factors, plus proposed mechanisms. (5) Diagnosis of exclusion — investigations and the wide geriatric differential; the caution about diagnosing it de novo after 65. (6) Course, prognosis with numbers, and treatment centred on pacing. Close with NICE NG206 and long COVID.

Mnemonic for the IOM criteria — "FUME + 1": Fatigue with activity reduction · **U**nrefreshing sleep · **M**alaise post-exertional · **E**verything for 6 months — **plus one** of cognitive impairment or orthostatic intolerance.

Numbers: 6 months (Fukuda, IOM) versus **3** months (NICE) · **4 of 8** Fukuda minor criteria · PEM lasting **>24 hours** · female:male **2–4:1** · onset typically **20–50 years** · **25%** severely affected · full recovery **5–10%**, improvement **~40%** · self-reported PEM in long COVID **67%**.

EXAMINER TRAPS

- Diagnosing chronic fatigue syndrome **de novo in a 75-year-old**. In geriatrics, new fatigue is **occult disease, drugs, depression or frailty until proven otherwise**. Say this explicitly — it is the geriatric marking point.
- Recommending **graded exercise therapy** as first-line treatment. This is now specifically advised against.
- Presenting CBT as **curative**, or implying the illness is psychological. Both lose marks and, in practice, lose the patient.
- Confusing **post-exertional malaise** with ordinary tiredness after exertion. The delay, the disproportion and the prolonged recovery define it.
- Ordering an exhaustive, expensive battery of tests. **Investigations exclude mimics; there is no confirmatory test.**
- Omitting the **medication review** — the single most productive intervention in an older patient with fatigue.

Night-Before Master Sheet — All 36 Questions

One row per question. Left column = the line that must appear in your answer. Right column = the current-evidence line that lifts it above a pass.

PART I — RENAL PHYSIOLOGY AND ASSESSMENT

Question	Must-write core	Update to quote
1 GFR	Peak **140** in the third decade, **−8 mL/min/1.73 m² per decade**; creatinine 1 mg/dL = GFR 120 at age 20 but **60 at age 80**; **Cockcroft–Gault for drug dosing**; one-third of older adults show no decline	**KDIGO 2024**: race-free **eGFRcr-cys** preferred, especially where creatinine is unreliable; point-of-care testing where labs are scarce
2 Renal biopsy	Same indications as the young, but **complications more serious**; nephrotic syndrome = **50% of biopsies**; crescentic GN peaks at 60–79	Biopsy-free membranous diagnosis if anti-PLA2R positive with normal function — but biopsy the atypical elderly, since amyloid coexists

PART II — FLUID, ELECTROLYTE AND ACID–BASE DISORDERS

Question	Must-write core	Update to quote
3 Fluid & electrolytes	Lead with ageing physiology: blunted thirst, concentrating **−30 mOsm/kg per decade**, free water clearance **5.9 vs 16.2 mL/min**, salt and urea wasting, hypoaldosteronism. **Hypovolaemia is always pathological in the old**	Correction cap **8–10 mEq/24 h** with DDAVP re-lowering; empagliflozin for SIAD; potassium binders before stopping RASi
4 Hyponatraemia	Serum osm → urine osm (**100**) → urine Na (**20 / 30–40**) → volume status. **Cognitive impairment even with mild hyponatraemia**	Cap **8–10 mEq/24 h** (6–8 if high risk); active re-lowering; beware **autocorrection** once the thiazide stops
5 SIADH	Four features: high urine osm, continued natriuresis, **euvolaemia**, normal renal/adrenal/thyroid. **Low uric acid**. Exclude **cerebral salt wasting**	**Empagliflozin 25 mg** raised Na by **4.1 mEq/L** in chronic SIAD; urea preferred over vaptans; deprescribing the SSRI is definitive
6 K^+ and Mg^{2+}	**Correct magnesium before potassium** (ROMK unblocking); never IV potassium in dextrose; U waves vs torsades	Deprescribe the **PPI** rather than escalate magnesium; SGLT2i incidentally raise serum magnesium
7 Potassium 5.7	**Not an emergency without ECG change.** Repeat the sample, ECG, drug review, **salt substitute**, bicarbonate for **type 4 RTA**	**KDIGO 2024**: binder *before* reducing RASi; SZC/patiromer; resonium removed from acute algorithms
8 Hypercalcaemia	Correct for albumin (**+0.8 per 1 g/dL**) → **measure PTH** → branch. Saline + **zoledronate 4 mg / pamidronate 90 mg**; calcitonin for speed (2–4 h vs 72 h)	**Endocrine Society**: **denosumab preferred to IV bisphosphonate** first-line in malignancy; combine with calcitonin if severe
9 Metabolic acidosis	**Albumin-corrected anion gap** → GOLD MARK vs urine anion gap; **type 4 RTA** in the older diabetic; Winter's formula	Keep serum bicarbonate **≥22 mEq/L** in CKD; veverimer failed VALOR-CKD, so bicarbonate remains standard

PART III — KIDNEY DISEASE

Question	Must-write core	Update to quote
10 AKI	Prerenal and postrenal are **reversible** — dehydration and the prostate. **FENa unreliable in the old.** No recovery by **3 months** = CKD	Balanced crystalloid; **STARRT-AKI** — no benefit from early dialysis; AKI alerts, nephrotoxin stewardship, post-AKI follow-up
11 CKD	Six mechanisms of progression (**HUP–PEP**); distinguish **senile hypofiltration**; conservative kidney management is an **active six-component pathway**	**KDIGO 2024**: eGFRcr-cys, KFRE risk equation, **SGLT2i regardless of diabetes**, statin for all over 50
12 Starting RRT	**AEIOU** for urgent dialysis; symptom-driven at eGFR 5–10; access at 15–20; **frailty and dependency, not age**, predict outcome	**PMNDP + AB-PMJAY + Ayushman Vay Vandana Card**; conservative kidney management as an explicit third option
13 PD vs HD	Survival **similar**; PD for cardiovascular instability and residual diuresis; median HD survival **15.6 and 11.6 months**; withdrawal **38%** of deaths	Shared decision-making across three pathways; assisted/home PD expanding in India under the national dialysis programme
14 Acute GN	Crescentic GN peaks at **60–79**; **renal vasculitis is the commonest primary cause of renal failure in the old**; **red cell casts**	**KDIGO AAV**: rituximab preferred in older adults; reduced-dose steroid taper; **avacopan** for steroid sparing
15 Nephrotic / membranous	Membranous is commonest; **20% secondary to malignancy** (lung, colon); highest thrombosis risk of the primary glomerular diseases	KDIGO: anti-PLA2R positive + normal function = no biopsy; **rituximab first-line** in moderate/high risk
16 Interstitial nephritis	Delayed hypersensitivity to **NSAIDs, diuretics, PPIs**; triad in **<10%**; **stopping the drug is the treatment**; steroids early	**Checkpoint-inhibitor AIN** — longer latency, no eosinophilia; steroid delay beyond 2 weeks predicts incomplete recovery
17 Diabetic nephropathy	**Mogensen I–V**; suspect non-diabetic disease if **no retinopathy**; RASi with creatinine rise **≤30%** acceptable	**FLOW 2024**: semaglutide ↓ kidney events **24%**; four-pillar therapy — RASi + SGLT2i + finerenone + GLP-1 RA

PART IV — DIABETES MELLITUS

Question	Must-write core	Update to quote
18 New diabetic, HbA1c 10%	**Dual therapy from the outset**: metformin + **GLP-1 RA** (weight, ASCVD, knee OA) or SGLT2i; **avoid sulphonylurea and pioglitazone**; ACEi/ARB + statin	**ADA 2026**: organ protection independent of HbA1c; obesity as a treatment target; tirzepatide; watch lean mass
19 SGLT2i / GLP-1 RA	No intrinsic hypoglycaemia; watch **volume depletion, genital infection, sarcopenia**; SGLT2i down to eGFR 20	**FLOW**: **24 / 18 / 20** (kidney / MACE / death); ADA 2026 — treat the organ, not the HbA1c
20 Alpha-glucosidase inhibitors	Block brush-border α-glucosidase → ↓ postprandial glucose; **treat hypoglycaemia with GLUCOSE, never sucrose**	Displaced internationally (no CV benefit, ACE trial) but retain an **Indian niche** for postprandial control and cost
21 Newer insulins	**Degludec and glargine U300** = flattest, least nocturnal hypoglycaemia; match the regimen to **coping ability**, not physiology	**Insulin icodec** once weekly — approved for type 2, **not** type 1 (ONWARDS 6 hypoglycaemia)

Question	Must-write core	Update to quote
22 Hypoglycaemia	Levels **70 / 54 / assistance**; **blunted autonomic warning**; presents as **falls and delirium**; sulphonylurea overdose needs 24–48 h observation	**ADA 2026**: CGM for older adults on insulin; annual screening for geriatric syndromes, hypoglycaemia and polypharmacy
23 DKA / HHS	**Fluids → Insulin → Potassium**; HHS = older type 2, higher mortality, slower correction; insulin 0.1 vs 0.05 units/kg/h	**ADA/EASD 2024**: glucose **≥200**, β-OHB **≥3.0**, pH <7.3; **anion gap removed**; euglycaemic DKA on SGLT2i
24 Diabetic foot	Neuropathy + PAD + pressure + limited joint mobility; **70%** of amputations follow an ulcer; **50%** three-year survival after amputation	**IWGDF 2023**: SINBAD for communication and audit; **non-removable knee-high offloading (total contact cast)** first choice

PART V — THYROID DISEASE

Question	Must-write core	Update to quote
25 Subclinical hypothyroidism	Treat if **TSH >10**; individualise 4.5–10; **in the oldest old it is harmless or possibly protective**; higher TSH target **6–7**	**TRUST**: 737 patients over 65 — TSH fell, but **no symptomatic, cognitive or functional benefit**
26 TSH 50 + heart failure	**Start 12.5–25 µg, titrate every 6–8 weeks**; empty stomach, 4 h from calcium/iron/PPI; lifelong; report chest pain. **The "heart failure" may be the myxoedema**	Age-adjusted TSH target (upper half of range); **no T3 or combination therapy**; amiodarone need not be stopped
27 Hashimoto's	**Commonest cause of hypothyroidism in old age**; ageing → fewer T-suppressor cells, more autoantibodies; **rapidly enlarging gland = lymphoma**	**Checkpoint-inhibitor thyroiditis**; selenium not recommended; antibodies justify surveillance, not treatment
28 Hyperthyroidism	**Apathetic, paucisymptomatic, atrial fibrillation, no goitre in one-third**; **^{131}I is the treatment of choice**; accept hypothyroidism as the planned outcome	ATA: **long-term low-dose antithyroid drugs now accepted**; 2025 network meta-analysis of the three modalities
29 Thyroid nodule	**95% benign**; risk equal for solitary and multinodular; **first test = TSH**; hot = no FNAB; **never suppress with levothyroxine**	Benign nodules rarely grow — ultrasound at 12 months then 2–5 yearly; active surveillance of low-risk microcarcinoma

PART VI — ENDOCRINE AND METABOLIC BONE DISEASE

Question	Must-write core	Update to quote
30 Testosterone	**Andropause does not exist**; treat only symptomatic, biochemically confirmed low T; target **300–400 ng/dL**; monitor haematocrit and PSA	**TRAVERSE 2023**: MACE non-inferior, but more atrial fibrillation, VTE, AKI and **fractures**
31 Osteomalacia	Low Ca + low PO_4 + **high ALP** + high PTH; bone pain with **proximal myopathy**; **Looser's zones**; replete before any bisphosphonate	**Endocrine Society 2024**: empiric vitamin D for all aged **≥75**; against routine 25(OH)D testing in the healthy; VITAL caveat

PART VII — CRYSTAL ARTHROPATHY

Question	Must-write core	Update to quote
32 Acute podagra	**Aspirate — needle-shaped, negatively birefringent; exclude sepsis.** Urate is often normal in the attack. Choose the drug by comorbidity. **Don't start ULT — but don't stop it either**	ACR: **ULT may be started during a flare under anti-inflammatory cover**; HLA-B*5801 testing in those of Southeast Asian descent
33 Management of gout	**Allopurinol first-line, preferred over probenecid in the elderly**; start low, titrate every 2–5 weeks; **target urate <6 mg/dL**; prophylaxis 3–6 months	**Treat-to-target** is the central shift; **STOP-Gout** — allopurinol non-inferior to febuxostat when properly titrated
34 Pseudogout	**Rhomboid, weakly positively birefringent**; knee and wrist; the **5 Hs**; **no crystal-lowering therapy exists**; aspiration alone is often therapeutic	**ACR/EULAR 2023 classification criteria**; ultrasound validated; anakinra when NSAIDs, colchicine and steroids are all contraindicated

PART VIII — ENVIRONMENTAL AND SYSTEMIC DISORDERS

Question	Must-write core	Update to quote
35 Heat stroke	Core **>40°C + CNS dysfunction**; mortality up to **80%**; **cool first, transport second**; immersion beats fanning; **no antipyretics, no dantrolene**	**SCCM 2025**: target **<39°C in 30 minutes**, rate ≥0.155°C/min; **NCDC India March 2024** cooling protocol; NDMA Heat Action Plans
36 Chronic fatigue syndrome	**Post-exertional malaise is the cardinal feature**; a diagnosis of exclusion; **pacing within the energy envelope**; full recovery only 5–10%	**NICE NG206**: PEM mandatory, 3-month diagnosis, **graded exercise therapy NOT recommended**, CBT adjunct only

Cross-Cutting Principles and Indian Context

SIX THREADS THAT RUN THROUGH EVERY ANSWER IN THIS BANK

1. **The ageing kidney and the ageing endocrine system explain most of this syllabus.** Blunted thirst, impaired urinary concentration and dilution, salt and urea wasting, hypoaldosteronism, poor acid handling, reduced receptor and post-receptor responsiveness. Open almost any answer with one line of that physiology and the clinical features follow logically.
2. **Atypical presentation is the geriatric signature.** Hypoglycaemia, HHS, hyponatraemia, hypercalcaemia, hypokalaemia, osteomalacia, pseudogout, thyrotoxicosis and heat stroke all present as **falls, delirium, immobility or "just ageing"** rather than as textbook syndromes. Say this once in every answer.
3. **Creatinine lies, and so does HbA1c.** Sarcopenia plus net tubular reabsorption means a normal creatinine can conceal a halved GFR; anaemia, CKD and transfusion make HbA1c falsely low. Dose every drug to the estimated GFR, and use glucose profiles or CGM where HbA1c is unreliable.
4. **Deprescribing is treatment, not the absence of it.** The thiazide in hyponatraemia and gout, the NSAID or PPI in interstitial nephritis and hypomagnesaemia, the SSRI in SIADH, the salt substitute in hyperkalaemia, the sulphonylurea in hypoglycaemia, the unnecessary levothyroxine — successfully withdrawn from half of the nursing-home residents studied.
5. **Start low, go slow, and accept an imperfect target.** Levothyroxine 12.5–25 µg, allopurinol under 100 mg, acarbose 25 mg, HbA1c 7–8%, TSH 6–7, sodium correction ≤8–10 mEq per 24 hours. The old are harmed by aggressive normalisation, and correction speed punishes more often than the number itself.
6. **Function and frailty, not chronological age, drive every decision** — renal biopsy, immunosuppression, dialysis modality, transplantation, thyroid surgery, testosterone therapy and glycaemic targets alike. Age is never, on its own, a contraindication.

INDIAN CONTEXT — READY-TO-USE LINES FOR ANY ANSWER

Programme / framework	What to write
NPHCE	National Programme for the Health Care of the Elderly — geriatric clinics, Regional Geriatric Centres, district geriatric units
NPCDCS	Opportunistic screening for diabetes and hypertension at health and wellness centres; the entry point for early CKD and diabetic foot detection
PMNDP	Pradhan Mantri National Dialysis Programme — free district-level haemodialysis in PPP mode, with policy expansion of peritoneal dialysis
AB-PMJAY / Ayushman Vay Vandana Card	Dialysis and hospital packages; the Vay Vandana Card covers **all citizens aged 70 and above irrespective of income**
NDMA Heat Action Plans · NCDC/NPCCHH	Ahmedabad HAP as the national model; NCDC "Emergency Cooling for Severe Heat-Related Illnesses" (March 2024), including improvised immersion using a body bag or tarpaulin; ambulance **108**
ICMR-NIN	Dietary reference values, calcium and vitamin D intake, and food-fortification policy — the pragmatic alternative to population-wide 25(OH)D testing
ADIP–ALIMCO · Rashtriya Vayoshri Yojana	Assistive devices including therapeutic footwear for the diabetic foot
Disease-specific Indian points	**CKD of unknown aetiology (CKDu)** in the agricultural belts of Andhra Pradesh, Odisha and Tamil Nadu · **tuberculosis** as a granulomatous cause of hypercalcaemia and a pulmonary cause of SIADH · **iodine-deficient regions** making toxic nodular goitre relatively commoner than Graves' disease · **potassium-containing "low-sodium salt"** and **aristolochic acid** in herbal preparations · **barefoot walking** as a diabetic-foot risk · **high-carbohydrate diets** justifying voglibose and acarbose · cheap **generic empagliflozin and dapagliflozin** against costly GLP-1 receptor agonists · **HLA-B*5801** in about 7% of those of Southeast Asian descent · **Kerala's community palliative care network** as the model for conservative kidney management

MD GERIATRIC MEDICINE

Consolidated Examination Answer Bank

Cardiovascular System · Respiratory Medicine & Sleep

54 Questions | 15-Mark Essays, 7-Mark and 6-Mark Notes

Primary source: Pathy's Principles and Practice of Geriatric Medicine, 6th edition (2022); Essentials of Clinical Geriatrics, 7th edition.

Answer architecture. 15-mark = **10 textbook points + 2 latest updates** (12 total). 7-mark = **6 textbook points + 1 latest update** (7 total). 6-mark = **5 textbook points + 1 latest update** (6 total). No point count is exceeded anywhere.

Structure of each answer. High-yield numbered points with bolded keywords → **Latest Updates** panel (2023–present, inline citation only) → **Current Practice Update** table wherever the textbook has been meaningfully superseded. Textbook content and current evidence are kept clearly separated throughout.

Latest evidence. 2025 AHA/ACC hypertension guideline; 2024 ESC atrial fibrillation and hypertension guidelines; 2023 ESC ACS, endocarditis, cardiomyopathy and heart failure updates; 2023 HRS physiologic pacing; 2024 ACC/AHA PAD; GOLD 2025–26; GINA 2025; BTS 2023; ATS/ERS/JRS/ALAT 2022; ACIP 2024; AASM and AGS Beers 2023; WHO EML 2023; and NEJM, Lancet, JAMA and Circulation trials to 2026.

Compilation note. This volume consolidates three separately compiled sets (Topics 25–36, 61–85 and 86–102) into a single continuously numbered document, reorganised by organ system and clinical theme. All source questions were distinct on review; no answer content has been abridged.

Prepared for: Dr. Sudarsan | MD Geriatric Medicine, India

Table of Contents

PART I — CARDIOVASCULAR SYSTEM

A. Heart Failure and the Cardiorenal Axis

1. Chronic and congestive cardiac failure — aetiology, clinical features and management, including in an 85-year-old lady 5
2. Management of refractory heart failure 6
3. Role of beta blockers in heart failure 7
4. Diastolic dysfunction — clinical presentation, diagnosis and treatment options 8
5. Case — 70-year-old man in cardiac failure with hypotension and tachypnoea: pathophysiology, differentials, investigations, and management in the ED, ward and after discharge 10
6. Cardiorenal syndrome 12

B. Ischaemic Heart Disease and Atherosclerosis

7. Unstable angina — clinical features, evaluation and management 13
8. Uses and limitations of the electrocardiogram in acute coronary syndromes 15
9. Case — advice to a 65-year-old man with diabetes, hypertension, dyslipidaemia and BMI 35, seen in clinic after two coronary stents 16
10. Drug-eluting stents and in-stent restenosis 17
11. Statins in the very old — primary and secondary prevention 18
12. Atherosclerosis and the biomarkers associated with it 19

C. Hypertension and Blood Pressure Disorders

13. Management of hypertension in a 65-year-old with dyslipidaemia and coronary artery disease 20
14. Stages of hypertension according to JNC 8, and the JNC 8 target blood pressure in people over 60 22
15. Accelerated hypertension 23
16. Isolated systolic hypertension — cut-off value, treatment and prognosis 24
17. Postural hypotension in the elderly — evaluation and management 25

D. Valvular and Myocardial Disease

18. Aortic stenosis — clinical presentation, diagnosis and management 27
19. Aortic sclerosis 28
20. Mitral regurgitation in the elderly 29
21. Infective endocarditis in the elderly 30
22. Cardiomyopathies — classification and management 31

E. Arrhythmias and Cardiac Pacing

23. Complete heart block and pacemaker indications 32
24. Sick sinus syndrome 33
25. Atrial fibrillation 34
26. CHA2DS2-VASc and HAS-BLED scores in atrial fibrillation 35
27. Tachyarrhythmias 36

F. Syncope, Falls and Autonomic Disorders

28. Head-up tilt test — indication, procedure and interpretation 37
29. Syncope — aetiology, pathogenesis, differential diagnosis and management 39
30. Drop attacks 41

G. Vascular Disease, Thrombosis and Prevention

31. Ankle brachial index and toe pressures — indications, assessment and interpretation 42
32. Direct oral anticoagulants — choice, dosing and reversal 44
33. Deep vein thrombosis — prophylaxis and treatment 44
34. Polypill 45

PART II — RESPIRATORY MEDICINE AND SLEEP

H. Respiratory Infection

35. What is community-acquired pneumonia? Etiopathogenesis, diagnosis and management 47
36. Pneumonia in the elderly 48
37. Hospital-acquired pneumonia 49
38. Case — 67-year-old man with cough, purulent expectoration, fever, tachypnoea, tachycardia and hypotension 50

I. Obstructive Airway Disease and Inhaled Therapy

39. Management of chronic obstructive airway disease 51
40. Asthma management based on GINA guidelines 52

41. Senile emphysema 53
42. Metered dose inhalers and inhaler technique in the older adult 54

J. Parenchymal and Pleural Disease

43. Diffuse parenchymal lung disease in older adults 55
44. Pleural effusion in a 70-year-old — etiopathogenesis, features, differential diagnosis, approach and management 57

K. Respiratory Failure and Critical Care

45. Respiratory failure, including acute type 2 respiratory failure 59
46. Management of ARDS 60
47. Long-term oxygen therapy and home non-invasive ventilation 61

L. Pulmonary Vascular Disease

48. Pulmonary embolism 62
49. Chronic pulmonary thromboembolism (CTEPD / CTEPH) 63

M. Thoracic Oncology

50. Bronchogenic carcinoma — types and management 64
51. Case — solitary pulmonary nodule on chest X-ray in a 30 pack-year smoker with cough 65

N. Sleep Medicine

52. Obstructive sleep apnoea — features, comorbidities, tests, management and prognosis 67
53. Sleep problems in the elderly, and the approach to insomnia in a 65-year-old recently retired professional 67
54. REM sleep behaviour disorder 69

APPENDIX — Rapid Revision Grid **71**

PART I

CARDIOVASCULAR SYSTEM

Questions 1 – 34 · 34 questions · 7 sections

A. Heart Failure and the Cardiorenal Axis

Question 1. Chronic and congestive cardiac failure — aetiology, clinical features and management, including in an 85-year-old lady

15-Mark Essay | 10 textbook points + 2 latest updates | ***Asked ×2***

Textbook Core – 10 Points

1. **Definition and classification.** Heart failure is a **clinical syndrome in which the heart is unable to deliver an output adequate for the metabolic needs of the tissues, or can do so only at raised filling pressures**. Classify by ejection fraction — **HFrEF (LVEF ≤ 40%), HFmrEF (41–49%) and HFpEF (≥ 50%)**; and by **NYHA class I–IV**. **In older patients, diastolic dysfunction rather than systolic dysfunction is the primary cause of heart failure, and it carries a marked increase in all-cause mortality.**
2. **Aetiology. More than 75% of overt heart failure in older patients is associated with hypertension or coronary heart disease.** Other causes: **degenerative calcific aortic stenosis and mitral annular calcification, atrial fibrillation, cardiomyopathy (including wild-type transthyretin amyloidosis), thyroid disease, anaemia, chronic kidney disease and cor pulmonale**. In India also consider **rheumatic valve disease**. Common **precipitants** of decompensation: infection, arrhythmia (especially new AF), myocardial ischaemia, non-adherence, high salt or fluid intake, anaemia, thyroid dysfunction, and drugs — **NSAIDs, corticosteroids, thiazolidinediones, non-dihydropyridine calcium channel blockers and negatively inotropic agents**.
3. **Why ageing predisposes.** Ageing produces **reduced β-adrenergic responsiveness with a blunted heart rate response, arterial stiffening with increased afterload, and cardiac hypertrophy**. There is **retarded left ventricular filling and a higher left ventricular diastolic pressure both at rest and on exercise**. Crucially, **older people depend more on atrial contraction than on ventricular relaxation for left ventricular filling, and are therefore more likely to develop heart failure when atrial fibrillation supervenes**. Because myocardial reserve is already used up to maintain normal resting function, **older people decompensate readily when disease is superimposed**.
4. **Clinical features. Exertional dyspnoea, orthopnoea, paroxysmal nocturnal dyspnoea, fatigue and reduced exercise tolerance**; signs of congestion — **raised JVP, basal crackles, third heart sound, cardiac enlargement, hepatomegaly and peripheral oedema**. In older patients the presentation is frequently **atypical — confusion or delirium, falls, anorexia, weight loss, nocturia, functional decline or simply "off legs"**. **Heart failure is often OVER-diagnosed in older adults: pedal and pretibial oedema alone is NOT sufficient for the diagnosis, since venous stasis produces an identical picture.** Look for the other objective signs before accepting the diagnosis.
5. **Investigations. Echocardiography determines the ejection fraction and is the key test** — NICE recommends echocardiography as the initial test in those with a previous myocardial infarction, and **natriuretic peptide (BNP or NT-proBNP)** measurement in those without. Note that **natriuretic peptides rise with age, renal impairment and atrial fibrillation, and are lowered by obesity**, so interpret with care. Add **ECG** (a completely normal ECG makes heart failure unlikely), **chest radiograph**, **full blood count, urea and electrolytes, creatinine, liver and thyroid function, glucose, HbA1c, iron studies and ferritin**. Consider **cardiac MRI or bone scintigraphy** if amyloidosis is suspected. **Specialist assessment should be considered in all.**
6. **Non-pharmacological management. Patient and carer education, daily weight monitoring with an action plan, salt and fluid moderation, smoking cessation and alcohol limitation**. **Supervised exercise training and cardiac rehabilitation** improve symptoms and quality of life at any age. Give **influenza, pneumococcal and COVID-19 vaccination**. Treat the comorbidities that drive decompensation — **hypertension, coronary artery disease, atrial fibrillation, diabetes, anaemia and iron deficiency, sleep apnoea, thyroid disease and depression**. **Multidisciplinary heart failure nurse-led follow-up reduces readmission.**
7. **Disease-modifying drugs for HFrEF.** The mainstays for systolic dysfunction in older as in younger patients are **diuretics for fluid overload, ACE inhibitors or ARBs, beta-blockers and aldosterone blockers**. **All patients with chronic symptomatic heart failure with reduced ejection fraction or left ventricular remodelling should receive an ACE inhibitor** to improve function and survival; **beta-blockers improve symptoms and survival**. **Low-dose spironolactone 12.5–50 mg reduced morbidity and mortality by 30% in NYHA class III–IV (RALES)**, with gynaecomastia as a notable adverse effect; **eplerenone reduced mortality and hospitalisation in patients with mild symptoms (EMPHASIS-HF)**, with fewer side effects. **Subgroup analyses of both MRA trials showed equal benefit in younger and older patients.** Monitor electrolytes closely at initiation and periodically thereafter. **Sacubitril–valsartan (ARNI)** was superior to enalapril in PARADIGM-HF.

8. **Diuretics, digoxin and other agents. Loop diuretics (furosemide, bumetanide) are the mainstay of symptomatic treatment of fluid retention** and show some mortality benefit versus placebo; in resistant cases give them **intravenously or combine with a thiazide (sequential nephron blockade)**. **In older people monitor renal function, electrolytes and fluid status closely to avoid hypovolaemia, hypotension and falls. Digoxin** reduces hospitalisation and improves symptoms in HFrEF in sinus rhythm but does **not improve mortality (DIG trial)**; benefit was seen at all ages, with side effects comparable to placebo in older patients. **In those over 70, start and maintain at 0.125 mg daily to achieve a level of 0.5–0.9 ng/mL**, monitoring potassium and renal function; watch for **gastrointestinal upset, heart block, arrhythmia and confusion**, and consider discontinuation once the indication has passed. **Hydralazine plus isosorbide dinitrate** is recommended in self-declared Black patients and as an alternative when ACE inhibitors and ARBs cannot be used. **Ivabradine (SHIFT)** may be added for a persisting sinus rate ≥ 70/min.
9. **Device and advanced therapy.** If symptoms persist despite optimal medical therapy, **refer to a specialist and consider cardiac resynchronisation therapy with or without a defibrillator** in those with a wide QRS and reduced ejection fraction; pooled trial data show a **survival benefit from primary-prevention ICDs that persists in older patients**, and CRT benefits older adults. **Ventricular assist devices** have been used in selected older patients. **Every device decision in the very old must weigh life expectancy, frailty, cognition and the patient's own goals — and deactivation of ICD shocks must be discussed as part of end-of-life planning.**
10. **Management of the 85-year-old lady — the geriatric answer.** (i) **Confirm the diagnosis objectively** — do not rely on ankle oedema; obtain an echocardiogram, and expect **HFpEF from hypertension, atrial fibrillation or aortic stenosis** to be the likeliest substrate. (ii) **Assess frailty, cognition, function, nutrition, continence, falls risk and social support** by comprehensive geriatric assessment. (iii) **Start low and go slow**, introducing one drug at a time with careful uptitration and close monitoring of blood pressure, renal function and electrolytes. (iv) **Review the whole drug chart** — stop NSAIDs, minimise anticholinergic and sedative burden, and address polypharmacy. (v) **Anticipate diuretic-related harm** — postural hypotension, falls, incontinence precipitating immobility, acute kidney injury and hyponatraemia; time diuretics to protect sleep and toileting. (vi) **Prioritise symptom relief, independence and quality of life over prognostic benefit** where life expectancy is short; use the **time-to-benefit** principle before adding prognostic drugs. (vii) **Recognise the family burden** — heart failure imposes a significant burden on families, requires multifaceted care, and **caregiver physical and emotional health should itself be an outcome that is assessed**. (viii) **Undertake advance care planning early**, and involve palliative care for refractory breathlessness.

Latest Updates – 2 Points (2023–Present)

11. **Four-pillar therapy and rapid sequencing.** The **2022 AHA/ACC/HFSA guideline and the 2023 ESC focused update** establish four foundational drug classes for HFrEF — **ARNI (or ACE inhibitor/ARB), beta-blocker, mineralocorticoid receptor antagonist and SGLT2 inhibitor** — to be started **together at low dose and uptitrated within 4–6 weeks (the STRONG-HF strategy) rather than sequentially over months**. **SGLT2 inhibitors (dapagliflozin, empagliflozin) are now recommended across the whole ejection fraction spectrum**, including HFpEF (EMPEROR-Preserved, DELIVER), and are notably well tolerated in older adults with no need for dose titration.
12. **New options for HFpEF and HFmrEF, which is the dominant phenotype in the very old. FINEARTS-HF (NEJM 2024)** showed that **finerenone, a non-steroidal MRA, reduced worsening heart failure events and cardiovascular death in HFmrEF and HFpEF**, with less hyperkalaemia than spironolactone. **STEP-HFpEF (NEJM 2023)** showed that **semaglutide markedly improved symptoms, physical limitation and exercise function in obesity-related HFpEF**. Together these give the first genuinely disease-modifying options for a phenotype the textbook describes as having no proven drug therapy.

Current Practice Update

Textbook	ACE inhibitor, beta-blocker, MRA and diuretic for systolic failure; "few trials in diastolic heart failure — digoxin and ARBs show minimal benefit; treat comorbidities."
Current recommendation	Four-pillar therapy including an SGLT2 inhibitor for HFrEF, started together and rapidly uptitrated; SGLT2 inhibitors plus finerenone now have proven benefit in HFpEF/HFmrEF.
Exam approach	Write "four pillars — ARNI/ACEi, beta-blocker, MRA, SGLT2 inhibitor," then add "SGLT2 inhibitors and finerenone (FINEARTS-HF 2024) have made HFpEF a treatable condition."

Question 2. Management of refractory heart failure

7-Mark Answer | 6 textbook points + 1 latest update

Textbook Core – 6 Points

1. **Definition.** Refractory (advanced or stage D) heart failure is **persistent severe symptoms at rest or on minimal exertion (NYHA class III–IV) despite optimal guideline-directed medical and device therapy**, with **objective severe cardiac dysfunction, recurrent hospitalisation or need for intravenous inotropes, and severely impaired exercise capacity**. Prognosis is poor — **one-year mortality approaches 50%**.
2. **Step 1 — confirm it is truly refractory.** Before labelling refractory failure, **verify the diagnosis and the ejection fraction, and exclude a reversible or under-treated cause**: **myocardial ischaemia, severe valve disease (especially aortic stenosis and mitral regurgitation), uncontrolled atrial fibrillation or other arrhythmia, anaemia and iron deficiency, thyroid dysfunction, infection, cardiac amyloidosis, sleep apnoea, chronic kidney disease and non-adherence**. Review the drug chart and **stop the aggravating drugs — NSAIDs, corticosteroids, thiazolidinediones, verapamil/diltiazem, class I antiarrhythmics and excessive negative inotropes**.
3. **Optimise and re-sequence medical therapy.** Ensure the patient is on **maximally tolerated four-pillar therapy**. If blood pressure limits uptitration, **reduce or stop non-prognostic vasodilators (nitrates, alpha-blockers, calcium channel blockers) first**. Add **digoxin** for symptom control and reduced hospitalisation, and **ivabradine** if the sinus rate remains ≥ 70/min. Consider **hydralazine plus isosorbide dinitrate** where ACE inhibitors or ARNIs are not tolerated. Correct **iron deficiency with intravenous ferric carboxymaltose**, which improves symptoms and reduces hospitalisation.
4. **Manage diuretic resistance.** This is the central practical problem. Steps: **restrict sodium and fluid**; **switch oral to intravenous loop diuretic** (bioavailability is unpredictable in gut oedema) as bolus or infusion; **double the dose rather than increase the frequency** (there is a threshold effect); add **sequential nephron blockade with a thiazide-type diuretic (metolazone or a thiazide) given 30 minutes before the loop diuretic**; add or continue an **MRA**; and consider **acetazolamide** or an **SGLT2 inhibitor** for additional natriuresis. **Monitor daily weight, strict fluid balance, renal function and electrolytes; a modest rise in creatinine during effective decongestion is acceptable and should not automatically stop treatment.** **Ultrafiltration** is reserved for genuinely diuretic-resistant congestion.
5. **Advanced therapies and referral.** Refer to a specialist heart failure service to consider: **cardiac resynchronisation therapy** if the QRS is broad; **revascularisation or valve intervention** (including TAVI or transcatheter edge-to-edge mitral repair) where indicated; **continuous intravenous inotropes (dobutamine, milrinone, levosimendan)** as a bridge or for palliation — these improve symptoms but **increase arrhythmia and mortality**; **left ventricular assist devices**, which have been used in older patients as destination therapy in carefully selected cases; and **cardiac transplantation**, which is essentially precluded by advanced age. **In the very old, the decision hinges on frailty, cognition, comorbidity and patient goals rather than on age alone.**
6. **Palliative care and end-of-life planning.** This is the highest-yield section for a geriatric examination. **Recognise the terminal phase** — repeated admissions, cachexia, hypotension limiting therapy, worsening renal function, refractory symptoms. Provide **opioids (low-dose morphine) for refractory breathlessness**, oxygen if hypoxaemic, treatment of anxiety and depression, and management of pain, nausea and constipation. Undertake **advance care planning, agree a ceiling of treatment and resuscitation status, and discuss ICD deactivation explicitly**. Simplify the drug regimen, **deprescribe drugs whose time-to-benefit exceeds life expectancy** (statins, some prognostic agents), continue diuretics for symptom relief, and support the **family and caregivers, whose physical and emotional health should be assessed as an outcome in their own right**.

Latest Update – 1 Point (2023–Present)

7. **Decongestion strategy has been sharpened by two randomised trials now embedded in the 2023 ESC focused update. ADVOR** showed that **adding intravenous acetazolamide 500 mg daily to loop diuretics significantly increased successful decongestion and reduced length of stay** in acute decompensated heart failure, and **CLOROTIC** showed that **adding hydrochlorothiazide to a loop diuretic improved weight loss and diuretic response**. Combined with the **STRONG-HF** strategy of rapid post-discharge uptitration with close follow-up (which reduced death and readmission), the modern approach to refractory congestion is **early combination diuretic therapy plus intensive early follow-up**, rather than escalating a loop diuretic alone.

Question 3. Role of beta blockers in heart failure

7-Mark Answer | 6 textbook points + 1 latest update

Textbook Core – 6 Points

1. **Rationale and mechanism.** Chronic heart failure activates the **sympathetic nervous system**, which is initially compensatory but ultimately causes **myocyte toxicity and apoptosis, adverse remodelling, arrhythmia, down-regulation of β_1-receptors and renin–angiotensin–aldosterone activation**. Beta-blockers **antagonise this neurohormonal activation**, producing **reverse remodelling with improved ejection fraction, reduced heart rate with improved diastolic filling and coronary perfusion, reduced myocardial oxygen demand, and antiarrhythmic protection against sudden death**.
2. **Evidence and indication. Beta-blockers improve symptoms and survival and are a mainstay of therapy for heart failure due to systolic dysfunction in older as in younger patients.** They are indicated in **all patients with HFrEF (LVEF ≤ 40%) who are clinically stable and euvolaemic**, together with an ACE inhibitor/ARNI, an MRA and an SGLT2 inhibitor. Landmark trials: **CIBIS-II (bisoprolol), MERIT-HF (metoprolol succinate), COPERNICUS (carvedilol)** and **SENIORS (nebivolol, specifically in patients aged ≥ 70)**, which showed benefit in an elderly population.
3. **Which drugs.** Only four agents have proven mortality benefit in heart failure — **bisoprolol, carvedilol, metoprolol succinate (extended release) and nebivolol**. **The benefit is not a class effect**: atenolol and short-acting metoprolol tartrate are not substitutes. **Carvedilol** also has α-blocking and antioxidant activity and is preferred where blood pressure is well maintained; **bisoprolol and nebivolol are β_1-selective** and better tolerated in coexisting airways disease; **nebivolol** additionally has nitric-oxide-mediated vasodilation and the strongest specific evidence in the old.
4. **How to prescribe — "start low, go slow".** Begin only when the patient is **euvolaemic and not requiring intravenous inotropes**. **Start at a very low dose** (bisoprolol 1.25 mg daily, carvedilol 3.125 mg twice daily, nebivolol 1.25 mg daily) and **double at intervals of not less than two weeks to the maximum tolerated or target dose**, checking heart rate, blood pressure, weight and symptoms at each step. **Warn the patient that symptoms may transiently worsen in the first weeks**. If fluid retention occurs, **increase the diuretic rather than stopping the beta-blocker**. **Some beta-blocker is better than none — a low maintained dose is preferable to withdrawal.**
5. **Adverse effects and cautions in older adults. Bradycardia and heart block** (particularly with coexisting sinus node disease, digoxin, amiodarone or verapamil), **hypotension and falls**, **fatigue**, **worsening of heart failure during uptitration**, **cold peripheries and worsening claudication**, **masking of hypoglycaemia in insulin-treated diabetes**, sleep disturbance and vivid dreams, and depression. **Contraindications:** second- or third-degree heart block without a pacemaker, symptomatic bradycardia, severe hypotension, cardiogenic shock and decompensated fluid overload. **Asthma and COPD are no longer absolute contraindications — recent evidence reassures on the safety of cardioselective beta-blockers, and they should be used when required in asthmatic and COPD patients. Never withdraw abruptly**, as rebound ischaemia and arrhythmia may result.
6. **Where the evidence is weaker.** In **HFpEF**, beta-blockers have **no proven mortality benefit** and are used chiefly for **rate control in atrial fibrillation, angina or hypertension**; excessive bradycardia may actually be harmful by limiting the chronotropic reserve that older patients already lack. In **heart failure with atrial fibrillation**, meta-analysis suggests the mortality benefit is **attenuated compared with sinus rhythm**, though beta-blockers remain useful for rate control. Their role is also limited in **cardiac amyloidosis**, where they are usually poorly tolerated.

Latest Update – 1 Point (2023–Present)

7. **The indication for beta-blockade after myocardial infarction has narrowed sharply — but the heart failure indication is untouched. REDUCE-AMI (NEJM 2024)** found that in patients after acute myocardial infarction with a **preserved ejection fraction (LVEF ≥ 50%) and no heart failure, long-term beta-blockade gave no reduction in death or recurrent infarction**, and **ABYSS (2024)** found that interruption of beta-blockade after uncomplicated MI was not inferior for hard outcomes. The examinable message is that **beta-blockers remain mandatory disease-modifying therapy in HFrEF at every age**, but should **no longer be continued indefinitely purely for a remote myocardial infarction with normal ventricular function** — a useful deprescribing opportunity in the polypharmacy-burdened older patient.

Question 4. Diastolic dysfunction — clinical presentation, diagnosis and treatment options

7-Mark Answer | 6 textbook points + 1 latest update | ***Asked ×2***

Textbook Core – 6 Points

1. **Definition and mechanism.** Diastolic dysfunction is **retarded left ventricular filling with a higher left ventricular diastolic pressure**, **present both at rest and during exercise in older persons**. It arises from **impaired active relaxation and increased passive chamber stiffness**, caused by **myocardial hypertrophy, interstitial fibrosis, arterial stiffening with increased afterload, and impaired calcium handling**. The result is that **a small increase in volume produces a large rise in filling pressure**, causing pulmonary congestion at a normal ejection fraction. When symptomatic it is called **heart failure with preserved ejection fraction (HFpEF)**.

2. **Why it dominates in old age. Diastolic dysfunction, not systolic dysfunction, is the primary cause of heart failure in older patients, and is associated with marked increases in all-cause mortality.** Age-related hypertrophy and stiffening mean that **older people depend more on atrial contraction than on ventricular relaxation for left ventricular filling**; consequently **they are far more likely to develop heart failure if atrial fibrillation ensues**, since the atrial kick is lost and diastole is shortened. **Heart failure may therefore occur in the absence of systolic dysfunction or valvular disease.**
3. **Causes and associations. Hypertension with left ventricular hypertrophy is the commonest**. Others: **coronary artery disease and ischaemia, aortic stenosis, diabetes mellitus, obesity, chronic kidney disease, atrial fibrillation, obstructive sleep apnoea, hypertrophic cardiomyopathy** and **infiltrative disease — especially wild-type transthyretin cardiac amyloidosis**, which is a commonly missed cause in the very old. Constrictive pericarditis is an important mimic.
4. **Clinical presentation.** Symptoms are **indistinguishable from systolic failure — exertional dyspnoea, orthopnoea, paroxysmal nocturnal dyspnoea, fatigue and reduced exercise tolerance**, with **raised JVP, basal crackles and peripheral oedema**. Distinguishing features: a **fourth heart sound (S4) rather than a third**, a **sustained apex beat**, a **predominance in elderly women with hypertension and obesity**, and **flash pulmonary oedema precipitated by hypertensive surges, tachycardia, atrial fibrillation, fluid or salt loading, or NSAIDs**. **The patient is exquisitely preload-dependent and decompensates in both directions — congestion with a little too much fluid, and hypotension with a little too much diuretic.**
5. **Diagnosis.** There are three requirements: **symptoms and signs of heart failure**, **a preserved ejection fraction (≥ 50%)**, and **objective evidence of raised filling pressures or diastolic dysfunction**. **Echocardiography is the key test** — look for **left ventricular hypertrophy with normal chamber size, left atrial enlargement (a marker of chronically raised filling pressure), a raised E/e′ ratio, reduced e′ tissue Doppler velocity, and an elevated tricuspid regurgitant velocity**. Support with **raised natriuretic peptides**, remembering that **BNP rises with age and renal impairment and is lowered by obesity** — so a normal level in an obese patient does not exclude HFpEF. Use the **H_2FPEF or HFA-PEFF score** where the diagnosis is uncertain, and **exercise (diastolic) stress echocardiography** for exertional symptoms with a normal resting study. Always **exclude constrictive pericarditis and cardiac amyloidosis**.
6. **Treatment.** The textbook position is that **few large randomised controlled trials of drug treatment in diastolic heart failure have been designed, and trials of digoxin and ARBs have shown minimal benefit** (CHARM-Preserved, I-PRESERVE, PEP-CHF and the ancillary DIG trial were all essentially neutral). Therefore: (i) **treat the comorbidities that drive it — hypertension, coronary artery disease, atrial fibrillation and diabetes mellitus**; (ii) use **diuretics to treat pulmonary congestion or peripheral oedema**, cautiously, since over-diuresis causes hypotension and renal impairment; (iii) use **beta-blockers to control heart rate** and lengthen diastolic filling time, and **restore or control rate in atrial fibrillation**; (iv) control **blood pressure** aggressively as the single most effective preventive measure; (v) encourage **weight loss, salt restriction and supervised exercise training**, which reliably improve exercise capacity and quality of life.

Latest Update – 1 Point (2023–Present)

7. **HFpEF is no longer an untreatable condition. SGLT2 inhibitors are now the first drug class with proven outcome benefit — EMPEROR-Preserved** and **DELIVER** showed that empagliflozin and dapagliflozin reduce cardiovascular death and heart failure hospitalisation across the preserved and mildly reduced ejection fraction range, and they are recommended for all such patients in the **2023 ESC focused update**. Building on this, **FINEARTS-HF (NEJM 2024)** showed that **finerenone reduces worsening heart failure events and cardiovascular death in HFmrEF and HFpEF**, and **STEP-HFpEF (NEJM 2023)** showed that **semaglutide produces large improvements in symptoms and exercise capacity in obesity-related HFpEF**. The examinable line is that the textbook statement "no proven drug therapy for diastolic heart failure" **is now out of date**.

Current Practice Update

Textbook	Few trials in diastolic heart failure; digoxin and ARBs show minimal benefit; treat comorbidities, use diuretics for congestion and beta-blockers for rate control.
Current recommendation	SGLT2 inhibitors are recommended for all patients with HFpEF/HFmrEF; finerenone (FINEARTS-HF) and semaglutide in obesity-related HFpEF (STEP-HFpEF) add further benefit.
Exam approach	Give the textbook management, then close with: "SGLT2 inhibitors (EMPEROR-Preserved, DELIVER) and finerenone (FINEARTS-HF 2024) are the first therapies with proven outcome benefit in HFpEF."

Question 5. Case — 70-year-old man in cardiac failure with hypotension and tachypnoea: pathophysiology, differentials, investigations, and management in the ED, ward and after discharge

15-Mark Essay | 10 textbook points + 2 latest updates

> **Recognise the syndrome in the first line:** heart failure **with hypotension** is **cardiogenic shock until proved otherwise** — this is the "cold and wet" haemodynamic profile, not simple acute pulmonary oedema. Marks are earned by naming the profile, giving a structured differential, and separating ED, ward and post-discharge care.

Textbook Core – 10 Points

1. **Underlying pathophysiology.** A primary **fall in cardiac output** (from pump failure, arrhythmia, valve failure or mechanical complication) causes **systemic hypoperfusion**. Compensatory **sympathetic activation and renin–angiotensin–aldosterone activation** raise heart rate and systemic vascular resistance, which **further increases afterload and myocardial oxygen demand while reducing coronary perfusion** — a **vicious spiral of ischaemia → worsening contractility → lower output → more ischaemia**. Simultaneously, **raised left ventricular filling pressure is transmitted to the pulmonary capillaries, causing interstitial and alveolar oedema**; this reduces compliance and impairs gas exchange, and the resulting **hypoxaemia and metabolic (lactic) acidosis drive the tachypnoea**. Systemic hypoperfusion causes **acute kidney injury, hepatic dysfunction, delirium and a systemic inflammatory response with inappropriate vasodilatation**.

2. **Haemodynamic profiling at the bedside.** Classify immediately by **congestion ("wet or dry") and perfusion ("warm or cold")**:

Profile	Findings	Approach
Warm and wet	Congested, well perfused, normal or high BP	Diuretic ± vasodilator
Cold and wet	**Congested AND hypoperfused — cool peripheries, narrow pulse pressure, mottling, oliguria, confusion, raised lactate**	**Inotrope/vasopressor first, then diuresis once perfused — this patient**
Cold and dry	Hypoperfused, not congested	Cautious fluid challenge, then inotrope

   Cardiogenic shock = systolic BP < 90 mmHg for > 30 minutes (or vasopressor need) with **evidence of end-organ hypoperfusion** and adequate filling.

3. **Differential diagnosis — cardiac causes to consider. Acute myocardial infarction with pump failure** (the commonest cause); **mechanical complications of MI — acute mitral regurgitation from papillary muscle rupture, ventricular septal rupture, free wall rupture with tamponade**; **tachyarrhythmia or bradyarrhythmia including complete heart block**; **critical aortic stenosis**; **acute severe valvular regurgitation from infective endocarditis**; **myocarditis**; **Takotsubo cardiomyopathy**; **cardiac tamponade**; and **decompensation of known heart failure**.

4. **Differential diagnosis — non-cardiac mimics that must be excluded. Sepsis and septic shock with myocardial depression** (fever, warm periphery, high lactate, source of infection); **massive pulmonary embolism** (right ventricular failure with clear lungs, raised JVP, hypoxia with hypocapnia); **hypovolaemia and gastrointestinal bleeding**; **tension pneumothorax**; **aortic dissection** with tamponade or acute aortic regurgitation; **anaphylaxis**; **drug toxicity — beta-blocker, calcium channel blocker or digoxin overdose**; **adrenal crisis and severe hypothyroidism (myxoedema)**; and **severe anaemia or thyrotoxicosis causing high-output failure**.

5. **Investigations that discriminate between them.**

Test	What it rules in or out
12-lead ECG (immediately)	STEMI, NSTEMI, arrhythmia, complete heart block, right heart strain (S1Q3T3) in PE, low voltage in tamponade
Bedside echocardiography	**The single most discriminating test** — LV/RV function, regional wall motion, valve rupture, VSD, tamponade, aortic stenosis, IVC size (excludes hypovolaemia)
Troponin, natriuretic peptide	Myocardial injury; a normal BNP essentially excludes heart failure as the cause of breathlessness
ABG with lactate	Hypoxia, acidosis, degree of hypoperfusion; hypocapnia with hypoxia suggests PE
Chest radiograph, lung ultrasound	Pulmonary oedema (B-lines, Kerley B, upper lobe diversion), pneumothorax, consolidation, widened mediastinum
FBC, U&E, creatinine, LFT, glucose, TFT, CRP, cultures	Sepsis, anaemia, AKI, hepatic congestion, thyroid disease
CT pulmonary angiogram / CT aorta	Pulmonary embolism; aortic dissection — only once stabilised
Coronary angiography	Definitive if ACS-related shock — should not be delayed

6. **Emergency Department management — immediate stabilisation. ABCDE**, monitored bed, two cannulae, arterial line if shocked, urinary catheter for hourly output. **Sit the patient up** and give **oxygen to target SpO_2 94–98%**; escalate to **CPAP or non-invasive ventilation**, which reduces preload and afterload, improves oxygenation and reduces intubation; intubate if exhausted, obtunded or acidotic despite NIV. **Do NOT give nitrates or high-dose diuretic first in a hypotensive patient** — nitrates are contraindicated at systolic BP < 90 mmHg. Instead: give a **cautious 250 mL fluid challenge only if the IVC is collapsed and there is no pulmonary oedema**; otherwise start **noradrenaline** to restore a mean arterial pressure ≥ 65 mmHg, then add an **inotrope (dobutamine or levosimendan)** to restore perfusion. Once perfused, give **intravenous furosemide** for congestion. Correct **arrhythmia (urgent DC cardioversion for haemodynamically unstable tachyarrhythmia; pacing for complete heart block)**, and if ACS is the cause arrange **immediate primary PCI**.
7. **Ward or coronary care management.** Continue **continuous monitoring of rhythm, blood pressure, saturation, hourly urine output, daily weight and strict fluid balance**, with **daily renal function and electrolytes**. **Wean vasopressors and inotropes as perfusion recovers**, then **titrate diuretics to a negative fluid balance**, adding a **thiazide or acetazolamide for diuretic resistance**. Treat the precipitant — **revascularisation, antibiotics, rate or rhythm control, correction of anaemia and thyroid dysfunction**. **Reintroduce and uptitrate disease-modifying therapy as soon as the patient is off inotropes and euvolaemic**: ACE inhibitor or ARNI, beta-blocker, MRA and SGLT2 inhibitor. Provide **VTE prophylaxis, early mobilisation, pressure-area care, delirium prevention, nutrition and dietitian input**. Refer for **device therapy (ICD/CRT) assessment** once the ejection fraction has been reassessed at 3 months on optimal therapy.
8. **Geriatric considerations throughout.** A 70-year-old may have **reduced β-adrenergic responsiveness, arterial stiffening, diastolic dysfunction and limited renal reserve**, so he is **exquisitely preload-sensitive** and tolerates neither over-filling nor over-diuresis. **Give fluid in 250 mL aliquots with reassessment**. Actively look for and prevent **acute kidney injury, delirium, falls, hospital-acquired infection, pressure sores, constipation and deconditioning**. **Review the whole drug chart** — stop NSAIDs and other precipitants; review anticholinergic and sedative burden. Undertake a **comprehensive geriatric assessment**, and **discuss ceilings of care, escalation and resuscitation status early** with the patient and family.
9. **Discharge planning. Discharge only when the patient is euvolaemic, on stable oral therapy for at least 24 hours, with stable renal function.** Provide: a **written management plan with daily weights and an action plan for a weight gain of more than 2 kg in three days**; **education on salt and fluid moderation, medication purpose and side effects**; a **reconciled and simplified drug list**; **enrolment in cardiac rehabilitation**; **influenza, pneumococcal and COVID-19 vaccination**; and **social and functional support with an occupational therapy home assessment**. Arrange **heart failure nurse follow-up within 7 days and clinic review within 2 weeks**, with **early uptitration of the four pillars — multidisciplinary nurse-led follow-up is the intervention that most reliably reduces readmission**.
10. **Longer-term follow-up and prognosis. Repeat echocardiography at 3 months on optimal therapy** to guide ICD/CRT referral. Monitor **renal function and potassium after each dose change**. Screen and treat **iron deficiency (intravenous iron), anaemia, atrial fibrillation, sleep apnoea, depression and frailty**. **Secondary prevention** if ischaemic — antiplatelet therapy, statin, blood pressure and glycaemic control, smoking cessation. Counsel on **driving restrictions and vaccination**. Prognosis after cardiogenic shock remains poor, so **advance care planning should be revisited**, and the **caregiver's physical and emotional health should be assessed as part of ongoing review**.

Latest Updates – 2 Points (2023–Present)

11. **DanGer Shock (NEJM 2024)** — the first adequately powered randomised trial of mechanical circulatory support in cardiogenic shock. In **STEMI-related cardiogenic shock**, a **microaxial flow pump (Impella CP) reduced 180-day all-cause mortality compared with standard care**, though at the cost of significantly more **bleeding, limb ischaemia, haemolysis and renal replacement therapy**. This is the first positive mortality trial in this field (intra-aortic balloon counterpulsation was neutral in IABP-SHOCK II), and it supports early referral of selected patients to a **shock team at a centre with mechanical circulatory support** — while the complication profile makes patient selection critical in the frail older patient.
12. **Culprit-lesion-only revascularisation and rapid post-discharge uptitration. CULPRIT-SHOCK** established that in shock complicating multivessel disease, **culprit-lesion-only PCI is superior to immediate multivessel PCI** (less death and renal replacement therapy) — staged treatment of remaining lesions is the correct approach. After stabilisation, **STRONG-HF** showed that **high-intensity uptitration of guideline-directed therapy to full dose within 2 weeks of discharge, with four follow-up visits in 2 months, significantly reduced death and heart failure readmission at 180 days**, and this benefit was preserved in older patients — making the **first two weeks after discharge the highest-value period of the whole admission**.

Question 6. Cardiorenal syndrome

7-Mark Answer | 6 textbook points + 1 latest update

Textbook Core – 6 Points

1. **Definition.** Cardiorenal syndrome (CRS) is a group of disorders of the heart and kidneys in which **acute or chronic dysfunction of one organ induces acute or chronic dysfunction of the other**. It is **bidirectional**, and its recognition matters because the presence of renal dysfunction is one of the strongest independent predictors of mortality in heart failure, and because it constrains the very treatments (diuretics, ACE inhibitors, MRAs) that heart failure requires.
2. **Classification (Ronco).**

Type	Name	Mechanism and typical example
Type 1	Acute cardiorenal	**Acute heart failure → acute kidney injury** (acute decompensated heart failure, cardiogenic shock, acute coronary syndrome)
Type 2	Chronic cardiorenal	**Chronic heart failure → progressive chronic kidney disease**
Type 3	Acute renocardiac	**Acute kidney injury → acute cardiac dysfunction** (fluid overload, hyperkalaemic arrhythmia, uraemic pericarditis)
Type 4	Chronic renocardiac	**Chronic kidney disease → cardiac disease** (LV hypertrophy, accelerated atherosclerosis, uraemic cardiomyopathy)
Type 5	Secondary	**A systemic disease damaging both organs** — sepsis, diabetes, amyloidosis, SLE, cirrhosis

3. **Pathophysiology.** The traditional model of a "low forward flow" reducing renal perfusion is now known to be secondary. **The dominant mechanism is renal VENOUS CONGESTION — a raised central venous pressure is transmitted back to the renal veins, raising renal interstitial and intracapsular pressure, reducing the transrenal perfusion gradient and hence the glomerular filtration rate.** Elevated intra-abdominal pressure from ascites and visceral oedema compounds this. Additional mechanisms: **neurohormonal activation (renin-angiotensin-aldosterone and sympathetic overactivity) causing sodium and water retention and efferent arteriolar constriction**; **systemic inflammation and oxidative stress**; **endothelial dysfunction with reduced nitric oxide**; **anaemia and iron deficiency, forming the "cardiorenal anaemia syndrome"**; and **drug-induced injury from NSAIDs, contrast media and over-diuresis**. In the older patient **reduced renal reserve, nephrosclerosis, and reduced GFR that is masked by low muscle mass** make all of this more likely.
4. **Clinical features and assessment.** Features of **congestion** — raised JVP, peripheral and sacral oedema, hepatomegaly and ascites, weight gain, orthopnoea — coexisting with features of **renal dysfunction** — rising creatinine and urea, oliguria, hyperkalaemia, metabolic acidosis, anaemia. **The critical bedside distinction is between the CONGESTED patient (raised JVP, dilated IVC, oedema — needs MORE decongestion) and the HYPOVOLAEMIC over-diuresed patient (flat JVP, postural drop, dry mucosae — needs LESS diuretic)**, because the creatinine rises in both. Assess with **daily weight, strict fluid balance, JVP, postural blood pressure, IVC diameter and lung ultrasound B-lines**. Investigations: **urea, electrolytes, creatinine and eGFR, urinalysis with urine protein-creatinine ratio, urine sodium, full blood count and iron studies, natriuretic peptides, renal ultrasound, echocardiography**, and review for nephrotoxins. **Note that a rise in creatinine during effective decongestion ("pseudo-worsening renal function") is common, is haemodynamic rather than structural, and is NOT associated with worse outcome provided congestion is being relieved**.

5. **Management — decongestion. Intravenous loop diuretics are the cornerstone.** Give as a **bolus or continuous infusion** at **1–2.5 times the usual home oral dose**; oral bioavailability is unreliable in gut oedema. For **diuretic resistance**, use **sequential nephron blockade** — add a **thiazide-type diuretic (metolazone or hydrochlorothiazide) 30 minutes before the loop diuretic**, add an **MRA**, and consider **acetazolamide**. Restrict sodium and fluid. **Ultrafiltration** is reserved for genuinely diuretic-resistant congestion; it removes isotonic fluid but has not shown superiority over stepped pharmacological care (CARRESS-HF). **Renal replacement therapy** for refractory hyperkalaemia, acidosis, uraemia or fluid overload. Monitor **daily weight, fluid balance, renal function and electrolytes**.
6. **Management — disease-modifying therapy and the drugs to keep or stop. Do NOT reflexively stop ACE inhibitors, ARBs or MRAs for a modest creatinine rise** — a rise of up to about 30% with stable potassium is acceptable and these drugs confer long-term cardiac and renal benefit; halve the dose and monitor rather than stopping outright. Do stop them for a rise beyond 30%, refractory hyperkalaemia or symptomatic hypotension. **Stop NSAIDs and other nephrotoxins, and minimise contrast exposure.** Continue or start an **SGLT2 inhibitor**, which reduces both heart failure and renal endpoints and may cause a small early reversible "dip" in eGFR that should not prompt discontinuation. Treat **anaemia and iron deficiency with intravenous iron**. Manage **hyperkalaemia** with dietary advice and a **potassium binder (patiromer or sodium zirconium cyclosilicate)** so that RAAS inhibition can be maintained. Treat the precipitant — infection, arrhythmia, ischaemia, obstruction. Involve **nephrology and cardiology jointly**, and in the frail older patient with advanced dual organ failure, undertake **advance care planning and consider a conservative, symptom-focused pathway rather than dialysis**.

Latest Update – 1 Point (2023–Present)

7. **Combination diuretic therapy up front, and non-steroidal MRAs, have changed cardiorenal management.** The **ADVOR trial** showed that **adding intravenous acetazolamide 500 mg daily to loop diuretics significantly increased successful decongestion and shortened hospital stay** in acute decompensated heart failure with volume overload, and **CLOROTIC** showed that adding hydrochlorothiazide improved weight loss and diuretic response — both are now reflected in the **2023 ESC heart failure focused update**, which endorses **early combination diuretic therapy for congestion rather than escalating a loop diuretic alone**. In parallel, **FIDELIO-DKD and FIGARO-DKD** established that **finerenone reduces both renal progression and cardiovascular events in chronic kidney disease with type 2 diabetes with less hyperkalaemia than spironolactone**, and **FINEARTS-HF (NEJM 2024)** extended this benefit to **HFmrEF and HFpEF** — giving, for the first time, a single agent that protects both organs in the cardiorenal patient.

B. Ischaemic Heart Disease and Atherosclerosis

Question 7. Unstable angina — clinical features, evaluation and management

15-Mark Essay | 10 textbook points + 2 latest updates | ***Asked ×2***

Textbook Core – 10 Points

1. **Definition and place in the ACS spectrum.** Unstable angina is **myocardial ischaemia at rest or on minimal exertion, without acute myocyte necrosis** — that is, **ischaemic symptoms with a normal (non-elevated) troponin**. It sits between stable angina and non-ST-elevation myocardial infarction (NSTEMI); together they form the **non-ST-elevation acute coronary syndromes (NSTE-ACS)**. The classical triad of presentations is **rest angina lasting more than 20 minutes, new-onset severe angina, or a crescendo pattern of previously stable angina**.
2. **Pathogenesis.** The usual mechanism is **disruption of an atheromatous plaque — rupture, fissuring, erosion or haemorrhage — with superimposed non-occlusive platelet-rich thrombus** causing a sudden reduction in coronary flow. Contributory mechanisms are **vasospasm, distal embolisation and inflammation**. In the older patient, **type 2 myocardial ischaemia** from an **oxygen supply–demand mismatch** is equally common and is precipitated by **sepsis, hypotension, tachyarrhythmia, anaemia, hypoxia, thyrotoxicosis and organ dysfunction** — this must be identified because the treatment is entirely different.
3. **Clinical features and the geriatric pitfall.** Typical features are **retrosternal chest tightness or heaviness radiating to the jaw, neck or left arm, with sweating, nausea and dyspnoea**. **In older people typical cardiac chest pain is less common and, when present, is usually less well defined or less severe.** Ischaemia may be **asymptomatic or present atypically with dyspnoea, fatigue, dizziness, syncope, confusion, lethargy or altered mental state**, especially in the frail. Because they are physically less active, **angina in this age group is less often related to exertion**. Signs may include a new S4, transient mitral regurgitation murmur, or signs of heart failure.

4. **Differential diagnosis.** Cardiac — **NSTEMI, STEMI, aortic dissection, pericarditis, myocarditis, Takotsubo, severe aortic stenosis**. Respiratory — **pulmonary embolism, pneumonia, pneumothorax, pleurisy**. Gastrointestinal — **epigastric pain may be misdiagnosed as peptic ulcer disease, and burning nocturnal or post-prandial pain as gastro-oesophageal reflux or hiatus hernia**. Musculoskeletal — **shoulder or back pain may be misdiagnosed as osteoarthritis**, costochondritis, herpes zoster. Also consider anxiety and panic disorder.
5. **Initial evaluation — ECG and biomarkers.** Obtain a **12-lead ECG within 10 minutes** and repeat it serially and with pain. Look for **ST depression, transient ST elevation and T-wave inversion**; **dynamic changes are the key finding** and confer high risk. **Note that the resting ECG is less valuable in the elderly because of common age-related changes, and a normal ECG does not exclude ACS.** Measure **high-sensitivity troponin at presentation and at 1–3 hours**: unstable angina is defined by an **absence of significant rise and fall**. Remember that troponin is **chronically elevated in renal impairment, heart failure, atrial fibrillation, sepsis, pulmonary embolism and old age**, so it is the **dynamic change, not the absolute level, that indicates infarction**.
6. **Further evaluation. Bedside echocardiography** for regional wall motion abnormality, ejection fraction, valve disease and mechanical complications. Baseline **full blood count (anaemia is a common precipitant and modifies antithrombotic choice), renal function, electrolytes, glucose, HbA1c, lipids, thyroid function and CRP**; **chest radiograph**. **The exercise treadmill test has limited diagnostic value in old age because exercise capacity is insufficient to induce ischaemia; pharmacological stress testing and imaging modalities are useful in older as in younger people.** Options include **stress echocardiography, myocardial perfusion imaging, stress cardiac MRI and CT coronary angiography**.
7. **Risk stratification.** Use the **GRACE score** (age, heart rate, systolic BP, creatinine, Killip class, cardiac arrest, ST deviation, troponin) for ischaemic risk and the **TIMI score** as a simpler alternative; use the **CRUSADE score** or **ARC-HBR criteria** for bleeding risk. **Very-high-risk features mandating immediate (< 2 h) invasive strategy: haemodynamic instability or cardiogenic shock, recurrent or refractory chest pain despite treatment, life-threatening arrhythmia, mechanical complication, acute heart failure, or recurrent dynamic ST changes**. **High risk (invasive within 24 h):** raised troponin, dynamic ST/T changes, GRACE score > 140. **Age is heavily weighted in every risk score, so almost all older patients are classified as high risk — but they are also at the highest bleeding risk, which is the central tension of the answer.**
8. **Immediate medical management. Anti-ischaemic: sublingual then intravenous nitrates** (avoid if hypotensive, in aortic stenosis or with recent phosphodiesterase-5 inhibitors), **beta-blockers** unless there is heart failure, bradycardia or cocaine use, and **a rate-limiting calcium channel blocker** if beta-blockers are contraindicated. **Oxygen only if SpO_2 < 90%. Antiplatelet: aspirin 150–300 mg loading then 75 mg daily indefinitely**, plus a **$P2Y_{12}$ inhibitor — ticagrelor or clopidogrel**; **prasugrel is contraindicated over 75 years, under 60 kg, or with prior stroke or TIA**. **Anticoagulant: fondaparinux 2.5 mg subcutaneously daily is preferred for its favourable bleeding profile**, or enoxaparin, or unfractionated heparin — all **renally dose-adjusted**. **Add a statin (high-intensity) and an ACE inhibitor**, and a proton pump inhibitor for gastroprotection with dual antiplatelet therapy.
9. **Invasive strategy and revascularisation. Coronary angiography with PCI** is the definitive treatment for high-risk patients, timed by risk as above, with **radial access preferred because it substantially reduces bleeding and mortality** — particularly valuable in older patients. Use **current-generation drug-eluting stents**. Choose **CABG** for left main or complex three-vessel disease, especially with diabetes, after Heart Team discussion. **During treatment of ACS, older people are more prone to side effects such as stroke, acute kidney injury and bleeding complications**, so contrast volume should be minimised with pre- and post-hydration, and antithrombotic doses carefully adjusted for weight and renal function. **Suboptimal timing — that is, excessively late referral — has a significant negative impact on both procedural risk and late outcome**, so decisions should not be made on a "wait and see" basis.
10. **Secondary prevention and geriatric care planning.** Long-term therapy: **aspirin indefinitely plus a $P2Y_{12}$ inhibitor for 12 months** (shortened to **1–3 months** in high bleeding risk, then single antiplatelet therapy), **high-intensity statin ± ezetimibe**, **ACE inhibitor**, and **beta-blocker where the ejection fraction is reduced**. Add **blood pressure and glycaemic control, smoking cessation, weight and diet management, and cardiac rehabilitation — in old age, cardiac rehabilitation following ischaemic events improves survival and enhances quality of life, general well-being and mental health**, and improves physical and cognitive function. Incorporate **multimorbidity, polypharmacy, physical and cognitive function, frailty and life expectancy** into decision-making, and adopt a **patient-centred rather than disease-centred plan** that focuses on **quality rather than quantity of life**. For frail patients with poor cognitive and physical function or limited life expectancy, aggressive treatment may not achieve meaningful goals, and **symptom palliation or end-of-life planning may be more appropriate**.

Latest Updates – 2 Points (2023–Present)

11. **SENIOR-RITA (NEJM 2024)** — the largest randomised trial of invasive versus conservative management in **NSTE-ACS in patients aged 75 and over** (median age 82, half frail). An invasive strategy **did NOT reduce the primary composite of cardiovascular death or non-fatal myocardial infarction, but did significantly reduce non-fatal myocardial infarction and subsequent revascularisation**, without excess procedural harm. The examinable message is nuanced and highly quotable: **routine invasive management should not be assumed beneficial for hard outcomes in the very old, but it remains reasonable for symptom control and to prevent recurrent events — the decision must be individualised by frailty, comorbidity and patient preference, not by chronological age**.
12. **2023 ESC Guidelines for Acute Coronary Syndromes** unified STEMI and NSTE-ACS into a **single guideline with a continuum-based approach**. Key changes: the **"invasive within 24 hours" recommendation for high-risk NSTE-ACS was downgraded to Class IIa** following neutral timing trials; **routine pre-treatment with a $P2Y_{12}$ inhibitor before angiography in NSTE-ACS is not recommended** (Class III); **abbreviated DAPT of 1–3 months followed by $P2Y_{12}$ monotherapy is endorsed in high-bleeding-risk patients**; and there is a dedicated section recommending that management in **older, frail patients be guided by comorbidity, frailty and estimated life expectancy**.

Current Practice Update

Textbook	Early invasive strategy for high-risk NSTE-ACS; pre-treatment with dual antiplatelet therapy before angiography; 12 months of DAPT.
Current recommendation	2023 ESC — no routine $P2Y_{12}$ pre-treatment in NSTE-ACS; 24-hour invasive timing downgraded to IIa; abbreviated DAPT in high bleeding risk; SENIOR-RITA tempers the invasive default in the very old.
Exam approach	Write "2023 ESC no longer recommends routine $P2Y_{12}$ pre-treatment in NSTE-ACS, and SENIOR-RITA (2024) shows the invasive strategy in over-75s reduces recurrent MI but not death — individualise by frailty."

Question 8. Uses and limitations of the electrocardiogram in acute coronary syndromes

7-Mark Answer | 6 textbook points + 1 latest update

Textbook Core – 6 Points

1. **Primary use — immediate triage.** The 12-lead ECG must be obtained and interpreted **within 10 minutes of first medical contact**. Its principal function is **to separate ST-elevation myocardial infarction, which requires immediate reperfusion, from non-ST-elevation ACS, which is managed with antithrombotic therapy and risk-stratified angiography**. Because **the greatest benefit from reperfusion occurs within the first two to three hours after symptom onset**, the ECG is the single time-critical investigation in the whole pathway.
2. **STEMI criteria and localisation. New ST elevation at the J point in two contiguous leads: ≥ 1 mm in all leads except V2–V3, where the threshold is ≥ 2 mm in men ≥ 40 years, ≥ 2.5 mm in men < 40, and ≥ 1.5 mm in women. New left bundle branch block with ischaemic symptoms is treated as a STEMI equivalent.** Territory localisation: **II, III, aVF = inferior (right coronary); V1–V4 = anteroseptal (LAD); I, aVL, V5–V6 = lateral (circumflex)**. **Always record right-sided (V4R) leads in inferior infarction to detect right ventricular involvement, and posterior leads (V7–V9) when there is ST depression in V1–V3.**
3. **NSTE-ACS findings and serial recording.** Look for **ST depression (horizontal or downsloping ≥ 0.5 mm), transient ST elevation and T-wave inversion**. **Dynamic changes — appearing with pain and resolving with relief — are the most specific and confer high risk.** A single ECG is therefore inadequate: **repeat at 15–30 minute intervals, with every episode of pain, and after any change in symptoms**. Continuous ST-segment monitoring is preferable where available.
4. **Additional uses beyond diagnosis.** The ECG gives **prognostic information** — the extent of ST deviation and the number of leads involved are components of the **GRACE risk score**. It detects **arrhythmic and conduction complications** — atrial fibrillation, ventricular tachycardia, and **complete heart block, which most often complicates inferior infarction**. It suggests **mechanical and other complications** such as pericarditis (widespread saddle-shaped ST elevation with PR depression) and left ventricular aneurysm (persistent ST elevation weeks later). It also provides a **baseline against which subsequent tracings are compared**, which is why obtaining old ECGs is so valuable.

5. **Limitations in the older patient. The resting ECG is less valuable in older people because of common age-related changes.** Confounders that mask or mimic ischaemia: **left bundle branch block, paced rhythm, left ventricular hypertrophy with strain, atrial fibrillation, digoxin effect, electrolyte disturbance and pre-existing Q waves or T-wave abnormalities**. **A normal ECG does NOT exclude acute coronary syndrome** — up to a third of NSTEMIs have a normal or non-diagnostic tracing, and the **left circumflex territory and the posterior wall are electrocardiographically "silent"**. Conversely the ECG cannot distinguish **type 1 (plaque rupture) from type 2 (supply–demand mismatch) infarction**, which is a very common and important distinction in the old.
6. **Practical consequences.** Because of these limitations the ECG must always be interpreted **together with the clinical context, serial high-sensitivity troponin measurement and echocardiography**. **Compare with a previous tracing wherever one exists.** Where left bundle branch block or a paced rhythm is present, apply the **Sgarbossa (or modified Smith-Sgarbossa) criteria** and use **urgent echocardiography** to look for a regional wall motion abnormality. **Never withhold reperfusion from a patient with a convincing history and evolving symptoms merely because the ECG is non-diagnostic — repeat it, and escalate to imaging or angiography.**

Latest Update – 1 Point (2023–Present)

7. The **2023 ESC ACS guideline** formally recognises **"STEMI-equivalent" patterns that indicate acute coronary occlusion despite the absence of classical ST elevation**, and recommends an **immediate invasive strategy for these patients**. They include: **de Winter's sign** (upsloping ST depression at the J point in the precordial leads with tall symmetrical T waves — proximal LAD occlusion); **posterior infarction** (ST depression in V1–V3 with tall R waves, confirmed on posterior leads V7–V9); **ST elevation in aVR with widespread ST depression** (left main or severe three-vessel disease); and **isolated right ventricular infarction on V4R**. This reflects the wider shift in emphasis from "STEMI versus NSTEMI" toward the **occlusion myocardial infarction (OMI) concept**, and it is precisely the kind of high-yield detail examiners reward.

Question 9. Case — advice to a 65-year-old man with diabetes, hypertension, dyslipidaemia and BMI 35, seen in clinic after two coronary stents

7-Mark Answer | 6 textbook points + 1 latest update

Frame the answer as structured secondary prevention. This man has established ASCVD with four modifiable risk factors and fresh stents — every point should name a target and an action.

Textbook Core – 6 Points

1. **Antiplatelet therapy — the most urgent point. Aspirin 75 mg daily lifelong**, plus a **$P2Y_{12}$ inhibitor (ticagrelor, prasugrel or clopidogrel) for 12 months after an acute coronary syndrome** or 6 months after elective PCI; shorten to **1–3 months if bleeding risk is high**, then continue single antiplatelet therapy. **Emphasise in the strongest terms that dual antiplatelet therapy must NOT be stopped prematurely — stent thrombosis carries about 40% mortality.** Advise him to carry a **stent card**, to **consult his cardiologist before any surgical or dental procedure**, and to report bleeding rather than stopping the tablets himself. Add a **proton pump inhibitor** for gastroprotection.
2. **Lipids.** He has established ASCVD, so target **LDL-cholesterol below 55 mg/dL (1.4 mmol/L) with at least a 50% reduction from baseline**. Start or continue a **high-intensity statin — atorvastatin 40–80 mg or rosuvastatin 20–40 mg**. If the target is not met, **add ezetimibe 10 mg**, then consider a **PCSK9 inhibitor or bempedoic acid**. Check a **lipid profile and liver function at 6–12 weeks**. Explain that the statin is taken for **plaque stabilisation, not for the cholesterol number alone**, and counsel on muscle symptoms so that he reports rather than silently stops the drug.
3. **Blood pressure.** Target **below 130/80 mmHg** given diabetes and coronary disease, avoiding a diastolic pressure below 70 mmHg (the "J-curve" concern in coronary disease). Preferred agents in this patient: an **ACE inhibitor or ARB** (cardioprotective, renoprotective in diabetes) plus a **beta-blocker** if the ejection fraction is reduced or angina persists, and a **calcium channel blocker or thiazide-like diuretic** as needed. Advise **home blood pressure monitoring** and **single-pill combinations** to reduce tablet burden and improve adherence.
4. **Diabetes.** Target **HbA1c around 7% (53 mmol/mol)**, individualised and relaxed if there is hypoglycaemia risk or frailty. Because he has established cardiovascular disease and obesity, **choose glucose-lowering agents for their cardiovascular benefit, not merely for glycaemic control — metformin as background, plus an SGLT2 inhibitor (cardiovascular and renal benefit, heart failure prevention) and/or a GLP-1 receptor agonist (atherosclerotic benefit plus substantial weight loss). Avoid pioglitazone (fluid retention and heart failure) and minimise sulfonylureas and insulin because of hypoglycaemia**. Arrange annual **retinal screening, foot examination and urine albumin-creatinine ratio**.

5. **Weight, diet and exercise.** A BMI of 35 is **class II obesity**: aim for an initial **5–10% weight loss**. Advise a **Mediterranean-style or DASH diet**, salt below 5 g/day, reduced refined carbohydrate and saturated fat, more fruit, vegetables, whole grains, nuts and fish, and **alcohol limitation**. **Refer to a structured cardiac rehabilitation programme — this is one of the most strongly evidence-based interventions after PCI, improving survival, quality of life, physical and cognitive function and mental health.** Build up to **150 minutes per week of moderate aerobic activity plus resistance training twice weekly**. Screen for **obstructive sleep apnoea**, which is highly likely at this BMI.
6. **General advice, safety-netting and follow-up. Absolute smoking cessation** with pharmacological support. Give **influenza, pneumococcal and COVID-19 vaccination**. Teach him to **recognise recurrent angina and to use sublingual GTN, with clear instructions to call emergency services if pain persists beyond 15 minutes or is severe**. Advise on **driving restrictions** after PCI per local regulations, and on **safe resumption of sexual activity** (nitrates must never be combined with phosphodiesterase-5 inhibitors). **Screen for depression and anxiety**, which are common and worsen outcomes. Discuss **adherence explicitly** and simplify the regimen. Arrange **follow-up at 4–6 weeks** with repeat lipids, renal function, electrolytes, HbA1c and blood pressure, and **reassess the ejection fraction if it was reduced**.

Latest Update – 1 Point (2023–Present)

7. **SELECT (NEJM 2023)** — in patients with **established cardiovascular disease and overweight or obesity but WITHOUT diabetes**, weekly **semaglutide 2.4 mg reduced major adverse cardiovascular events by 20%**, establishing **obesity itself as a modifiable cardiovascular target and pharmacological weight loss as secondary prevention in its own right**. In a patient who also has diabetes, the parallel evidence from **SUSTAIN-6 and LEADER (GLP-1 receptor agonists)** and **EMPA-REG OUTCOME, CANVAS and DECLARE-TIMI 58 (SGLT2 inhibitors)** supports using these agents for cardiovascular protection. Current ACC/AHA and ESC guidance therefore recommends an **SGLT2 inhibitor and/or a GLP-1 receptor agonist in patients with type 2 diabetes and established ASCVD, independently of HbA1c**.

Question 10. Drug-eluting stents and in-stent restenosis

7-Mark Answer | 6 textbook points + 1 latest update

Textbook Core – 6 Points

1. **Stent types.** A **bare-metal stent (BMS)** is a bare metallic scaffold that prevents acute recoil but has a high restenosis rate of **20–30%**. A **drug-eluting stent (DES)** has three components — a **metal platform**, an **antiproliferative drug** and a **polymer carrier**. Current-generation DES use **thin-strut cobalt-chromium or platinum-chromium platforms** eluting **everolimus, zotarolimus or sirolimus** from **biocompatible or biodegradable polymers**.
2. **Mechanism and benefit of DES.** The antiproliferative drug inhibits **vascular smooth muscle proliferation and neointimal hyperplasia**, reducing **restenosis and target-lesion revascularisation to under 5%**. Current-generation DES also have **lower stent thrombosis rates than BMS**, so **DES are now the default for virtually all patients, including the elderly and those at high bleeding risk** — BMS are essentially obsolete.
3. **In-stent restenosis (ISR) — definition and mechanism.** ISR is **renarrowing of ≥ 50% of the stented segment**. Two mechanisms: **neointimal hyperplasia** (dominant with BMS, occurs early at 3–12 months) and **neoatherosclerosis** — new atherosclerotic plaque forming inside the stent (typical of DES, occurs late, beyond 1 year, and may rupture). Usually presents as **recurrent stable angina**, but up to a third present as an **acute coronary syndrome**.
4. **Risk factors for ISR. Patient factors** — diabetes mellitus, chronic kidney disease, continued smoking. **Lesion factors** — small vessel diameter (< 2.5 mm), long lesions, **bifurcation and ostial** lesions, heavily calcified lesions, chronic total occlusion. **Procedural factors** — **stent under-expansion or malapposition**, geographic miss, residual dissection, stent fracture. **Premature discontinuation of dual antiplatelet therapy** is the key modifiable factor for stent thrombosis.
5. **Evaluation and treatment of ISR.** Coronary angiography with **intravascular imaging (IVUS or OCT)** to define the mechanism — under-expansion needs high-pressure or cutting/scoring balloon dilatation, whereas neointimal hyperplasia needs antiproliferative therapy. Definitive options: **drug-coated balloon (DCB)** or **implantation of a second DES**; plain balloon angioplasty alone has an unacceptably high recurrence rate. **CABG** is considered for recurrent or diffuse multi-vessel ISR.

6. **Antiplatelet therapy and geriatric considerations. Dual antiplatelet therapy (aspirin + a $P2Y_{12}$ inhibitor)** is given for **6 months after elective PCI** and **12 months after an acute coronary syndrome**, shortened to **1–3 months** in patients at **high bleeding risk (ARC-HBR criteria — age ≥ 75, anaemia, CKD, prior bleeding, oral anticoagulation)**, followed by single antiplatelet therapy. **Never stop DAPT prematurely — stent thrombosis carries about 40% mortality.** In the very old prefer **clopidogrel**; **prasugrel is contraindicated over 75 years, under 60 kg, or with prior stroke/TIA**. If oral anticoagulation is also needed, use **triple therapy for only about a week, then a DOAC plus clopidogrel**.

Latest Update – 1 Point (2023–Present)

7. The **AGENT IDE trial (2024)** showed that a **paclitaxel-coated balloon** was **superior to conventional balloon angioplasty** for coronary in-stent restenosis, reducing target-lesion failure — leading to the **first FDA approval of a coronary drug-coated balloon in the United States (2024)**. DCB treatment avoids adding another metal layer, which is particularly attractive in older patients with recurrent ISR and high bleeding risk. In parallel, the **2023 ESC ACS guideline** endorses **abbreviated DAPT (1–3 months) followed by $P2Y_{12}$ inhibitor monotherapy** in high-bleeding-risk patients.

Current Practice Update

Textbook	Choice between bare-metal and drug-eluting stents, with DES avoided when prolonged DAPT is undesirable; ISR treated with repeat stenting.
Current recommendation	Current-generation DES for all patients regardless of bleeding risk, with the duration of DAPT shortened instead; drug-coated balloons are a guideline-supported first-line option for ISR.
Exam approach	State "modern DES with abbreviated DAPT has replaced the BMS strategy in high-bleeding-risk elderly patients" — this single line is highly mark-earning.

Question 11. Statins in the very old — primary and secondary prevention

7-Mark Answer | 6 textbook points + 1 latest update

Textbook Core – 6 Points

1. **Rationale. Atherosclerotic cardiovascular disease is the leading cause of death and disability in adults aged 75 and above**, and **age is the strongest single predictor of cardiovascular risk**. Because absolute risk rises steeply with age, **the absolute benefit of lipid lowering can be greater in older than in younger patients** even though the relative risk reduction is similar. Statins act by **HMG-CoA reductase inhibition**, with additional plaque-stabilising and anti-inflammatory effects.
2. **Secondary prevention — evidence is clear.** In established ASCVD (previous myocardial infarction, stroke or TIA, peripheral arterial disease, or revascularisation), statins **reduce major vascular events and mortality at all ages**. At least **moderate-intensity statin therapy is recommended regardless of age**, and existing therapy should **not be stopped merely because the patient has grown older**. This is the strongest examination point.
3. **Primary prevention — evidence is weaker.** Older adults, particularly those over 75, were **under-represented in the major trials**. **PROSPER** (pravastatin, ages 70–82) reduced coronary events but **not stroke and not mortality**; **ALLHAT-LLT** showed no benefit in older hypertensive patients. Standard risk calculators (Pooled Cohort Equations, SCORE2) **lose specificity above 75** because age dominates the score. Decisions must therefore be **individualised**.
4. **Time-to-benefit and the geriatric decision.** The average **time to benefit is about 2.5 years** for prevention of ASCVD events. **A statin is inappropriate when estimated life expectancy is shorter than the time to benefit.** Weigh **frailty, multimorbidity, cognition, polypharmacy, functional status and patient preference**, and use **shared decision-making**. Consider **deprescribing** in advanced dementia, terminal malignancy, severe frailty or when goals of care become purely palliative.
5. **Adverse effects and interactions. Statin-associated muscle symptoms** (myalgia, weakness — a cause of falls and reduced mobility; rarely rhabdomyolysis), **transaminitis**, and **new-onset diabetes**. Risk rises with **CYP3A4 interactions** — **amiodarone, verapamil, diltiazem, macrolides, azole antifungals, ciclosporin** — so **avoid high-dose simvastatin**. **Pravastatin, rosuvastatin and pitavastatin** are least CYP3A4-dependent. Evidence overall shows **neutral or even protective cognitive effects**; statins do not cause dementia.
6. **Practical prescribing. Start low and go slow** — moderate intensity (**atorvastatin 10–20 mg** or **rosuvastatin 5–10 mg**) is usually sufficient. Check **baseline LFT and CK**, exclude hypothyroidism, and repeat lipids at **6–12 weeks**. If the LDL target is not met, **add ezetimibe 10 mg** rather than escalating the statin dose — this is better tolerated in the old. For statin intolerance, try an alternative statin, alternate-day dosing, or **bempedoic acid / PCSK9 inhibitors** in selected high-risk patients.

Latest Update – 1 Point (2023–Present)

7. **2025 National Lipid Association / American Geriatrics Society Expert Clinical Consensus (JAGS 2025)** on hypercholesterolaemia in adults over 75 without ASCVD: LDL-C remains an **independent predictor of ASCVD beyond age 75**; it is **reasonable to initiate a statin for primary prevention when LDL-C is 70–189 mg/dL in the absence of life-limiting illness**; **coronary artery calcium scoring** can refine risk when the decision is uncertain; and **deprescribing is appropriate in those with life-limiting disease**. The **STAREE trial** (atorvastatin 40 mg versus placebo in ~10,000 Australians aged ≥ 70, co-primary endpoint **disability-free survival**) will settle the primary prevention question — **results awaited**.

Current Practice Update

Textbook	Statin benefit in the very old is uncertain, particularly for primary prevention; treatment individualised.
Current recommendation	2025 NLA/AGS consensus gives an explicit LDL-C threshold (70–189 mg/dL), supports CAC-guided decisions, and formalises deprescribing criteria.
Exam approach	Write "secondary prevention — statin regardless of age; primary prevention — individualise using time-to-benefit (~2.5 years) and the 2025 NLA/AGS consensus."

Question 12. Atherosclerosis and the biomarkers associated with it

7-Mark Answer | 6 textbook points + 1 latest update | ***Asked ×2***

Textbook Core – 6 Points

1. **Definition and burden.** Atherosclerotic cardiovascular disease is the **leading cause of death and disability in adults aged 75 and older**. Atherosclerosis is a **slow-developing chronic inflammatory disease that begins as early as the first decades of life, at the level of the intimal tunica of large-calibre arteries**. The presence of atherosclerosis has been positively correlated with age at death even in **ancient mummified populations**, suggesting it is an **inherent component of human ageing** — but genetic and environmental factors modulate its severity and speed of progression, among which **lipid disorders, in particular hypercholesterolaemia, are foremost**.
2. **Pathogenesis.** The sequence is: **endothelial injury and dysfunction** (from shear stress at branch points, hypertension, smoking, hyperglycaemia, oxidised LDL) → **increased permeability with subendothelial retention and oxidation of apoB-containing lipoproteins → monocyte adhesion and migration, differentiation into macrophages and uptake of oxidised LDL to form foam cells → fatty streak → smooth muscle migration and proliferation with fibrous cap formation → fibrofatty plaque with a necrotic lipid core → plaque rupture, fissuring, erosion or haemorrhage with superimposed thrombosis**, producing the acute event. In ageing, **cellular senescence primes the vessel and drives established disease forward**, and **"inflammageing"** — chronically elevated inflammatory markers — contributes through inhibition of growth factors, increased catabolism and disturbed homeostatic signalling.
3. **Risk factors. Non-modifiable:** age (the strongest single predictor of cardiovascular risk), male sex, family history and genetic predisposition, ethnicity. **Modifiable: dyslipidaemia, hypertension, diabetes mellitus, smoking, obesity and visceral adiposity, physical inactivity, chronic kidney disease**. **Emerging and geriatric-specific: frailty appears to be a cardiovascular risk factor unique to older people, sharing a pathogenetic link with ischaemic heart disease through chronic inflammation and haematological and endocrine factors**; and **loneliness, social isolation, depression and dementia** are further risk factors that increase cardiovascular events in old age.
4. **Established lipid biomarkers.**

Biomarker	Significance
LDL-cholesterol	The **causal driver and primary treatment target**; remains independently associated with ASCVD beyond the age of 75
Non-HDL-cholesterol	Total minus HDL — captures **all atherogenic apoB particles**; superior in diabetes, obesity and hypertriglyceridaemia; needs no fasting sample
Apolipoprotein B	Directly counts **atherogenic particle number**; more accurate than LDL-C when triglycerides are high or LDL-C is low
Lipoprotein(a)	**Genetically determined, largely unmodifiable by diet or statins**; causal for ASCVD and for calcific aortic stenosis; measure **once in a lifetime**
Triglycerides, HDL-C	HDL-C is a risk marker, not a treatment target — HDL-raising therapies have failed to improve outcomes

5. **Inflammatory and other circulating biomarkers. High-sensitivity CRP** — an established marker of residual inflammatory risk; a strong association exists between increasing age and increasing systemic inflammation as measured by CRP. **Interleukin-6** — an upstream driver of the inflammatory pathway and a core marker of inflammageing and frailty. **Lp-PLA2 and myeloperoxidase** — markers of plaque inflammation and instability. **Homocysteine** — associated with risk, but lowering it with B vitamins does not reduce events. **Fibrinogen and D-dimer** — thrombotic risk. **NT-proBNP and high-sensitivity troponin** — markers of subclinical myocardial injury and strain that predict events in older adults, and are increasingly used as "newer biomarkers" of cardiovascular risk. **Note that in older people a raised troponin and BNP frequently reflect chronic myocardial stress rather than acute events, so trends matter more than absolute values.**
6. **Imaging biomarkers and clinical use. Coronary artery calcium (CAC) score on non-contrast CT** is the most useful risk-refining test — a **score of zero confers a very low short-term risk** and can justify withholding or deferring a statin, whereas a high score reclassifies the patient upward. **Carotid intima-media thickness and carotid plaque on ultrasound**, **ankle brachial index** and **CT coronary angiography with plaque characterisation** are alternatives. The practical role of all these markers is to **refine risk when the decision to treat is uncertain** — this matters especially in older adults, in whom **standard risk calculators lose specificity because chronological age dominates the score**. **Management remains risk-factor modification: statin therapy to LDL targets, blood pressure and glycaemic control, smoking cessation, weight loss and exercise.**

Latest Update – 1 Point (2023–Present)

7. **Two biomarkers have moved from research interest to guideline recommendation.** First, **Lipoprotein(a)**: **ESC/EAS and 2025 AHA/ACC guidance now recommend measuring Lp(a) at least once in every adult's lifetime** for risk stratification, and the first targeted therapies — **pelacarsen (Lp(a)HORIZON), olpasiran and lepodisiran** — are in phase 3 outcome trials, with pelacarsen lowering Lp(a) by around 80%. Second, **residual inflammatory risk**: pooled analyses of over 30,000 statin-treated patients (Ridker et al., **Lancet 2023**) showed that **high-sensitivity CRP predicted future cardiovascular events and death more strongly than LDL-cholesterol did**, supporting the dual-target concept — **lower LDL for the lipid pathway and target inflammation with colchicine (LoDoCo2, COLCOT) for the inflammatory pathway**. In parallel, the **2025 AHA/ACC hypertension guideline has adopted the PREVENT risk equations**, which for the first time incorporate **eGFR, urine albumin-creatinine ratio and HbA1c** into routine cardiovascular risk estimation.

C. Hypertension and Blood Pressure Disorders

Question 13. Management of hypertension in a 65-year-old with dyslipidaemia and coronary artery disease

15-Mark Essay | 10 textbook points + 2 latest updates

Textbook Core – 10 Points

1. **Definition and the importance of the problem.** Hypertension is defined as a **systolic blood pressure of 140 mmHg or greater and/or a diastolic blood pressure of 90 mmHg or greater**. It is the **major risk factor for stroke, heart failure and coronary artery disease in older adults, all of which are important contributors to mortality and functional disability**. Because it is remediable and its control reduces the incidence of coronary heart disease and stroke, **increased efforts at detection and treatment are indicated**. In this patient, hypertension, dyslipidaemia and established coronary disease act multiplicatively, so **global cardiovascular risk reduction — not blood pressure alone — is the objective**.
2. **Accurate diagnosis and measurement.** Confirm with **repeated measurements on at least two occasions** using a validated device and correct cuff size, after 5 minutes seated rest, with the arm supported at heart level. **Measure in both arms initially and use the higher reading. Always measure standing blood pressure at 1 and 3 minutes to detect orthostatic hypotension, which is common in this age group and limits how aggressively treatment can be titrated.** Confirm the diagnosis with **home blood pressure monitoring or 24-hour ambulatory monitoring**, which also detects **white-coat hypertension, masked hypertension and non-dipping**. Exclude **pseudohypertension** from rigid calcified arteries.
3. **Evaluation — secondary causes and target-organ damage.** Screen for secondary hypertension where there is resistant or accelerated hypertension or a suggestive feature: **renal parenchymal disease, renovascular disease (renal bruit, flash pulmonary oedema, AKI on ACE inhibitor), primary aldosteronism (hypokalaemia), obstructive sleep apnoea, thyroid disease, phaeochromocytoma, Cushing's syndrome**, and **drugs — NSAIDs, corticosteroids, decongestants, ciclosporin, erythropoietin, liquorice**. Assess target-organ damage: **ECG and echocardiography for left ventricular hypertrophy, urine albumin-creatinine ratio and eGFR, fundoscopy**, and carotid or peripheral vascular assessment.

4. **Baseline investigations and risk assessment. Full blood count, urea, electrolytes, creatinine and eGFR, fasting glucose and HbA1c, fasting lipid profile, uric acid, liver and thyroid function, urinalysis and ACR, ECG**. Because he already has **established coronary artery disease, he is by definition at very high cardiovascular risk** — a risk calculator is unnecessary, and he qualifies immediately for **intensive blood pressure lowering and high-intensity statin therapy**.
5. **Blood pressure targets in this patient.** Aim for a **systolic pressure below 130 mmHg**, and generally **not below 120 mmHg**, with a **diastolic pressure of 70–80 mmHg**. **In coronary artery disease, be cautious about lowering the diastolic pressure below about 70 mmHg, because coronary perfusion occurs in diastole and excessive lowering may worsen ischaemia — the "J-curve" phenomenon.** Targets must be **individualised**: relax them in the presence of **frailty, symptomatic orthostatic hypotension, falls, cognitive impairment or limited life expectancy**. **Standing blood pressure, not seated, should be used to guide titration in patients with postural symptoms.**
6. **Non-pharmacological management.** These measures lower blood pressure and also improve lipids and glycaemia. **Salt restriction to under 5 g of sodium chloride daily** (an important and achievable target in the Indian diet); **DASH or Mediterranean dietary pattern** rich in fruit, vegetables, whole grains and low-fat dairy; **weight reduction**; **at least 150 minutes per week of moderate aerobic exercise plus resistance training**; **alcohol moderation**; **smoking cessation**; **stress reduction and adequate sleep**; and **withdrawal of drugs that raise blood pressure, especially NSAIDs**.
7. **Choice of antihypertensive drugs — driven by the coronary disease.** The comorbidity dictates the choice (a "compelling indication"):

Agent	Role in this patient
Beta-blocker	**First choice** — antianginal, reduces myocardial oxygen demand, mandatory if there is prior MI with reduced ejection fraction or heart failure
ACE inhibitor or ARB	**First choice** — reduces cardiovascular events in coronary disease (HOPE, EUROPA), regresses LV hypertrophy, renoprotective
Dihydropyridine calcium channel blocker (amlodipine)	Excellent add-on; effective in isolated systolic hypertension and in angina; ankle oedema is the limiting effect
Thiazide-like diuretic (indapamide, chlorthalidone)	Effective add-on; watch **hyponatraemia, hypokalaemia, gout and postural hypotension** in the old
Spironolactone 25 mg	Drug of choice for **resistant hypertension** (PATHWAY-2) — monitor potassium and renal function

   Avoid short-acting nifedipine, alpha-blockers as first-line therapy (postural hypotension), and the combination of an ACE inhibitor with an ARB. Verapamil and diltiazem must be avoided if there is heart failure or if a beta-blocker is co-prescribed.
8. **Prescribing principles in the older adult. Start low and go slow** — begin with a single low-dose agent or a low-dose combination, and **review at 4-weekly intervals**. **Prefer adding a second low-dose drug to maximising the dose of one, and prefer single-pill combinations, which improve adherence and reduce tablet burden.** Check **renal function and electrolytes 1–2 weeks after starting or increasing an ACE inhibitor, ARB, diuretic or MRA**. At every visit, **recheck standing blood pressure, ask about dizziness and falls, and reconcile the medication list**. Deprescribe or reduce doses if the patient becomes frail, loses weight, or develops symptomatic hypotension.
9. **Concurrent management of the dyslipidaemia.** With established coronary artery disease the LDL target is **below 55 mg/dL (1.4 mmol/L) with at least a 50% reduction from baseline**. Give a **high-intensity statin (atorvastatin 40–80 mg or rosuvastatin 20–40 mg)**, **add ezetimibe 10 mg** if the target is not reached, and consider a **PCSK9 inhibitor or bempedoic acid** thereafter. **Intensive LDL lowering has been shown to be beneficial in high-risk older patients with established cardiovascular disease.** Watch for **CYP3A4 interactions** with amlodipine, diltiazem, verapamil and amiodarone — **avoid high-dose simvastatin**. Check **lipids and liver function at 6–12 weeks** and address **statin-associated muscle symptoms** promptly rather than allowing silent discontinuation.
10. **Complete the secondary prevention package and follow up.** Add **aspirin 75 mg daily** (secondary prevention — established coronary disease), **sublingual GTN for angina with clear instructions**, and consider an **SGLT2 inhibitor or GLP-1 receptor agonist** if diabetes coexists. Arrange **cardiac rehabilitation**, **influenza, pneumococcal and COVID-19 vaccination**, and **screening for and treatment of obstructive sleep apnoea, depression and anaemia**. Follow up **monthly until controlled, then 3–6 monthly**, with **annual renal function, electrolytes, lipids and ECG**. Reinforce **adherence at every visit** — non-adherence is the commonest cause of apparent resistant hypertension. Add **Indian context**: high dietary salt, low potassium intake, early-onset and more aggressive ASCVD, and a large treatment gap — **API and Lipid Association of India consensus** recommend intensive risk factor control in this group.

Latest Updates – 2 Points (2023–Present)

11. **2025 AHA/ACC multisociety High Blood Pressure Guideline** — the first major US update since 2017, and **co-authored by the American Geriatrics Society**. Key changes: a **universal treatment target of below 130/80 mmHg with further encouragement to reach a systolic pressure below 120 mmHg**, supported by SPRINT, **STEP** (which specifically randomised patients aged 60–80), **ESPRIT** and **BPROAD**; replacement of the Pooled Cohort Equations with the **PREVENT risk calculator**; a strong endorsement of **single-pill combination therapy** and of **out-of-office (home or ambulatory) measurement for both diagnosis and titration**; a new Class IIb recommendation for **renal denervation** in uncontrolled hypertension; and **explicit individualised exceptions to the target for institutionalised patients, those with limited life expectancy and the frail**.
12. **2024 ESC Guidelines for Elevated Blood Pressure and Hypertension** introduced a **new intermediate category of "Elevated BP" (systolic 120–139 or diastolic 70–89 mmHg)**, in which treatment is considered for those at high cardiovascular risk, and set a **general on-treatment target of systolic 120–129 mmHg where tolerated**. Crucially for geriatric practice, the guideline recommends that treatment be guided by **tolerability rather than by chronological age**, mandates **routine screening for orthostatic hypotension**, and applies a **less intensive, symptom-guided target in patients with significant frailty or limited life expectancy**.

Question 14. Stages of hypertension according to JNC 8, and the JNC 8 target blood pressure in people over 60

7-Mark Answer | 6 textbook points + 1 latest update

Important framing point to state at the start of the answer: JNC 8 (2014) did not create a staging system. It was a treatment-threshold and target guideline; the stages that are conventionally quoted belong to **JNC 7 (2003)**. Stating this explicitly earns marks — many candidates conflate the two.

Textbook Core – 6 Points

1. **Classification (JNC 7 stages, retained for reference).** The textbook definition is a **systolic pressure of 140 mmHg or greater and/or a diastolic pressure of 90 mmHg or greater**.

Category	Systolic (mmHg)		Diastolic (mmHg)
Normal	< 120	and	< 80
Prehypertension	120–139	or	80–89
Stage 1 hypertension	**140–159**	or	**90–99**
Stage 2 hypertension	**≥ 160**	or	**≥ 100**
Isolated systolic hypertension	**≥ 140**	and	**< 90**

2. **What JNC 8 actually did.** JNC 8 (published in **JAMA, 2014**) was an **evidence-based panel report addressing nine questions on thresholds, targets and drug choice**, deliberately restricted to **randomised controlled trial evidence**. It **did not define stages, did not address measurement technique, secondary hypertension, lifestyle modification or resistant hypertension**, and was **not formally endorsed by the NHLBI**.
3. **The JNC 8 recommendation for people over 60 — the core of the question. In the general population aged 60 years and older, initiate pharmacological treatment when the blood pressure is ≥ 150/90 mmHg, and treat to a goal of below 150/90 mmHg (Grade A, strong recommendation).** This **relaxation of the systolic threshold from 140 to 150 mmHg** was the single most controversial and most examinable element of JNC 8, and was based chiefly on **HYVET, SHEP and Syst-Eur**. The panel added a **corollary (expert opinion)**: if treatment already achieves a lower systolic pressure without adverse effects on health or quality of life, **treatment need not be adjusted upwards**. A **minority of the panel dissented**, arguing the evidence was insufficient to raise the threshold above 140 mmHg.
4. **Other JNC 8 thresholds and targets.**

Group	Threshold to treat	Goal
General population **≥ 60 years**	**≥ 150/90**	**< 150/90**
General population < 60 years	≥ 140/90	< 140/90
Diabetes (any age)	≥ 140/90	< 140/90
Chronic kidney disease (any age)	≥ 140/90	< 140/90

Note that JNC 8 **relaxed the diabetes and CKD targets from the previous 130/80 mmHg**.

5. **JNC 8 drug recommendations.** In the **general non-Black population, including those with diabetes**, first-line treatment may be a **thiazide-type diuretic, calcium channel blocker, ACE inhibitor or ARB**. In the **general Black population, including those with diabetes**, first-line treatment should be a **thiazide-type diuretic or calcium channel blocker**. In **chronic kidney disease of any race, an ACE inhibitor or ARB should be included** for renal protection. **Beta-blockers were NOT recommended as first-line therapy** (based on the LIFE trial), which was another notable departure from JNC 7. Titration strategies: maximise the first drug, add a second before maximising the first, or start with two drugs.
6. **Criticisms and the geriatric perspective.** The over-60 target of 150/90 mmHg was criticised as **too permissive**, particularly for patients with coronary artery disease or previous stroke, and it was **overturned by SPRINT (2015)**, which showed that targeting a systolic pressure below 120 mmHg reduced cardiovascular events and all-cause mortality, including in a prespecified subgroup aged 75 and over. **The enduring, examinable geriatric principle from JNC 8 is nevertheless correct and still current: blood pressure targets in older people must be individualised and balanced against orthostatic hypotension, falls, polypharmacy, frailty and quality of life.** The important practical caveat is that **SPRINT measured unattended automated office blood pressure, which reads roughly 5–10 mmHg lower than conventional office measurement**.

Latest Update – 1 Point (2023–Present)

7. **JNC 8 is now obsolete and has been superseded twice over.** The **2025 AHA/ACC multisociety guideline** (co-authored by the American Geriatrics Society) **retires and replaces the 2017 ACC/AHA guideline and sets a universal target of below 130/80 mmHg, with encouragement to reach a systolic pressure below 120 mmHg**, supported by SPRINT, **STEP** (in patients aged 60–80), **ESPRIT** and **BPROAD**. It also introduces the **PREVENT risk calculator** in place of the Pooled Cohort Equations, endorses **single-pill combinations** and **home/ambulatory measurement**, and provides **explicit individualised exceptions for institutionalised patients, the frail and those with limited life expectancy**. In parallel the **2024 ESC guideline** introduced the intermediate category of **"Elevated BP" (120–139/70–89 mmHg)** and a target of **120–129 mmHg systolic where tolerated**.

Current Practice Update

Textbook / JNC 8	Age ≥ 60 — treat at ≥ 150/90 mmHg to a goal of < 150/90 mmHg; diabetes and CKD goal < 140/90 mmHg.
Current recommendation	2025 AHA/ACC — universal target < 130/80 mmHg with encouragement toward < 120 mmHg systolic; 2024 ESC — 120–129 mmHg systolic where tolerated; both individualise for frailty.
Exam approach	Answer the question as asked (JNC 8: < 150/90 over 60), then add one line: "JNC 8 has been superseded — SPRINT, STEP and the 2025 AHA/ACC guideline now recommend < 130/80 mmHg, individualised for frailty."

Question 15. Accelerated hypertension

7-Mark Answer | 6 textbook points + 1 latest update

Textbook Core – 6 Points

1. **Definitions and terminology. Accelerated hypertension** is a **severe elevation of blood pressure (usually diastolic > 120–130 mmHg) with grade III retinopathy — flame haemorrhages, cotton-wool spots and hard exudates**. **Malignant hypertension** is the same picture **with grade IV retinopathy, that is, papilloedema**. The modern classification is more useful:

Term	Definition	Management
Hypertensive urgency	Severe hypertension (usually ≥ 180/120) **WITHOUT acute target-organ damage**	**Oral** agents, lower over 24–48 h, outpatient or short observation
Hypertensive emergency	Severe hypertension **WITH acute, ongoing target-organ damage**	**Intravenous** therapy in a monitored setting

The presence of acute target-organ damage, not the absolute number, defines the emergency.

2. **Pathophysiology.** A rapid rise in blood pressure overwhelms **vascular autoregulation**, causing **endothelial injury, loss of vasomotor tone and increased permeability**. The histological hallmarks are **fibrinoid necrosis of arterioles and proliferative (onion-skin) endarteritis**. This triggers a self-perpetuating cycle: ischaemia → renin–angiotensin activation → further vasoconstriction and pressure natriuresis → volume depletion → more renin release. In the brain, breakthrough of cerebral autoregulation produces **vasogenic cerebral oedema (hypertensive encephalopathy, PRES)**; in small vessels it causes **microangiopathic haemolytic anaemia with thrombocytopenia**.

3. **Causes. Essential hypertension that is untreated or where treatment has been abruptly stopped** (the commonest scenario, and abrupt clonidine or beta-blocker withdrawal is a classic trigger). Secondary causes are disproportionately represented: **renal parenchymal disease, renovascular disease (bilateral renal artery stenosis, fibromuscular dysplasia), primary aldosteronism, phaeochromocytoma, Cushing's syndrome, coarctation, systemic sclerosis renal crisis, and eclampsia**. Drugs and toxins: **cocaine, amphetamines, sympathomimetics and decongestants, ciclosporin and tacrolimus, erythropoietin, NSAIDs, corticosteroids, VEGF inhibitors and tyrosine kinase inhibitors, MAOI–tyramine interaction, and steroid or liquorice excess**.
4. **Clinical features by target organ. Eye** — blurred vision, visual loss; grade III/IV retinopathy on fundoscopy. **Brain** — **hypertensive encephalopathy** with headache, nausea, vomiting, visual disturbance, confusion, seizures and coma; also **intracerebral haemorrhage, subarachnoid haemorrhage and ischaemic stroke**. **Heart** — **acute pulmonary oedema, acute coronary syndrome, aortic dissection**. **Kidney** — **acute kidney injury with haematuria, proteinuria and red cell casts**. **Blood** — **microangiopathic haemolytic anaemia with schistocytes and thrombocytopenia**. **In older patients, presentation may be non-specific — delirium, falls or unexplained deterioration — so fundoscopy and urinalysis must be performed in every severely hypertensive older patient.**
5. **Investigations. Fundoscopy is mandatory and is the single most important bedside investigation.** Also: **urinalysis and urine microscopy for haematuria, proteinuria and casts; urea, electrolytes and creatinine; full blood count with a blood film for schistocytes; LDH, haptoglobin and reticulocyte count; ECG and troponin; chest radiograph; and echocardiography**. Imaging as indicated: **CT or MRI brain** for neurological features (PRES shows posterior white-matter oedema), **CT aortogram** for suspected dissection. Investigate for **secondary causes once stabilised** — renal ultrasound with Doppler, plasma aldosterone-to-renin ratio, plasma or urinary metanephrines, and a full drug and substance history.
6. **Management. Hypertensive urgency:** treat with **oral agents (amlodipine, an ACE inhibitor, a beta-blocker) and reduce the pressure gradually over 24–48 hours**, with close outpatient follow-up. **Never use sublingual short-acting nifedipine — the uncontrolled fall in pressure precipitates stroke and myocardial infarction, a particular danger in the older patient with impaired cerebral autoregulation. Hypertensive emergency:** admit to a monitored bed with **intra-arterial pressure monitoring** and use titratable **intravenous** agents. The general rule is to **reduce the mean arterial pressure by no more than 20–25% in the first hour, then to about 160/100 mmHg over the next 2–6 hours, and to normal over 24–48 hours**. **Important exceptions:** in **aortic dissection**, reduce systolic pressure to **< 120 mmHg and heart rate < 60/min within 20 minutes** using a **beta-blocker (esmolol or labetalol) FIRST, then a vasodilator**; in **acute ischaemic stroke**, do not lower unless > 220/120 mmHg (or thrombolysis is planned, when the target is < 185/110 mmHg). Drug choice by context: **labetalol or nicardipine** for encephalopathy and stroke; **glyceryl trinitrate** for acute pulmonary oedema or ACS; **esmolol plus a vasodilator** for dissection; **phentolamine** for phaeochromocytoma; **labetalol or hydralazine with magnesium** for eclampsia. **Avoid diuretics unless there is pulmonary oedema**, since these patients are often volume-depleted from pressure natriuresis.

Latest Update – 1 Point (2023–Present)

7. The **2024 ESC hypertension guideline and the 2025 AHA/ACC guideline** both reinforce a shift away from treating a number toward **treating the presence of acute target-organ damage**. Specific practice points: for **hypertensive urgency without organ damage there is no benefit and potential harm from rapid parenteral lowering** — oral therapy with follow-up within days is preferred, and **routine admission is discouraged**; the **2025 AHA/ACC guideline provides drug- and condition-specific intravenous protocols** (labetalol, nicardipine, clevidipine, esmolol, enalaprilat, fenoldopam) rather than a single generic target; and both guidelines **advise against short-acting oral or sublingual agents in any setting**. The 2025 guideline also adds a Class IIb recommendation for **renal denervation** in patients whose hypertension remains uncontrolled despite optimal therapy or who cannot tolerate medication.

Question 16. Isolated systolic hypertension — cut-off value, treatment and prognosis

7-Mark Answer | 6 textbook points + 1 latest update

Textbook Core – 6 Points

1. **Definition and cut-off. Isolated systolic hypertension (ISH) is defined as a systolic pressure of 140 mmHg or greater WITH a diastolic pressure of less than 90 mmHg.** It is the **commonest form of hypertension in older adults**, accounting for the majority of hypertension after the age of 60, and its prevalence rises steadily with age. A **wide pulse pressure (systolic minus diastolic, normally < 60 mmHg)** is its defining haemodynamic feature and is itself a strong independent predictor of cardiovascular events.

2. **Pathophysiology.** ISH results from **age-related stiffening of the large conduit arteries** — degeneration and fragmentation of **elastin** with increased **collagen** deposition, calcification, endothelial dysfunction and reduced nitric oxide availability. Loss of the aortic "Windkessel" cushioning function means the **stroke volume is ejected into a rigid tube, raising systolic pressure**, while **faster pulse-wave velocity causes the reflected wave to return during systole rather than diastole, further augmenting systolic pressure and lowering diastolic pressure**. Contributory factors: high salt intake, obesity, diabetes, chronic kidney disease, and high-output states such as anaemia, thyrotoxicosis, aortic regurgitation, arteriovenous fistula and Paget's disease.
3. **Clinical assessment and pitfalls.** Most patients are asymptomatic. Confirm with repeated measurements and **home or ambulatory monitoring**. **Three specific pitfalls in the old: (i) always measure standing blood pressure, since orthostatic hypotension coexists in up to a third and limits treatment; (ii) exclude pseudohypertension from rigid calcified arteries, suggested by a palpable radial artery despite cuff occlusion (Osler's sign) and by high readings without target-organ damage; (iii) look for white-coat and masked hypertension.** Assess target-organ damage — **ECG and echocardiography for left ventricular hypertrophy, eGFR and urine ACR, fundoscopy**.
4. **Treatment thresholds and targets.** Treat when the systolic pressure is **persistently ≥ 140 mmHg** (or lower in high-risk patients), aiming for a **systolic pressure below 130 mmHg where tolerated**. **The critical caution is that lowering systolic pressure inevitably lowers diastolic pressure further, and a diastolic pressure below about 60–65 mmHg may compromise coronary perfusion, which occurs in diastole — the "J-curve".** Therefore titrate to **symptoms and standing blood pressure**, accept a higher systolic target in those with symptomatic postural hypotension, and relax targets in **frailty, dementia, recurrent falls or limited life expectancy**. Begin with **lifestyle measures — salt restriction below 5 g daily, weight reduction, DASH-type diet, aerobic exercise, alcohol moderation and withdrawal of NSAIDs**.
5. **Drug treatment. Thiazide-like diuretics (indapamide, chlorthalidone) and dihydropyridine calcium channel blockers (amlodipine) are first-line**, because they are the agents proven in the ISH trials and they specifically reduce arterial stiffness and central pressure. **ACE inhibitors and ARBs** are effective alternatives and are preferred with diabetes, chronic kidney disease, heart failure or previous myocardial infarction. **Beta-blockers are less effective in ISH** — by slowing the heart they increase stroke volume and, through wave reflection, may raise central aortic pressure — so they are used only for a compelling indication such as angina, prior MI or rate control. **Start with one low-dose agent (or a low-dose single-pill combination), review 4-weekly, and add rather than maximise.** Monitor **electrolytes and renal function, and check for hyponatraemia, hypokalaemia, gout and postural dizziness**.
6. **Prognosis.** ISH is **not a benign consequence of ageing**. **Systolic pressure and pulse pressure are stronger predictors of cardiovascular events in older adults than diastolic pressure**, and ISH carries an increased risk of **stroke, heart failure, coronary events, atrial fibrillation, chronic kidney disease, cognitive decline and vascular dementia**. The benefit of treatment is well established: **SHEP** reduced stroke by about 36%, **Syst-Eur** reduced stroke by about 42% and also reduced incident dementia, and **HYVET**, in patients aged 80 and over, showed reductions in stroke, heart failure and **all-cause mortality** — establishing that **treating hypertension is worthwhile even in the very old**. The counterweight is that over-treatment causes **falls, syncope, fractures, acute kidney injury and electrolyte disturbance**, so the outcome depends on careful individualised titration.

Latest Update – 1 Point (2023–Present)

7. **Intensive systolic lowering is now supported by trials conducted specifically in older adults.** The **STEP trial** randomised Chinese patients **aged 60–80** to a systolic target of 110–130 versus 130–150 mmHg and found **a significant reduction in cardiovascular events in the intensive group, with no increase in falls, syncope or acute kidney injury**; **ESPRIT** (2024) and **BPROAD** (2024, in diabetes) reached concordant conclusions. On this basis the **2025 AHA/ACC guideline** sets a universal target of **below 130/80 mmHg with encouragement toward below 120 mmHg systolic**, and the **2024 ESC guideline** targets **120–129 mmHg systolic where tolerated** — both explicitly stating that **chronological age alone should not raise the target, but frailty, symptomatic orthostatic hypotension and limited life expectancy should**.

Question 17. Postural hypotension in the elderly — evaluation and management

7-Mark Answer | 6 textbook points + 1 latest update

Textbook Core – 6 Points

1. **Definition and prevalence.** Orthostatic (postural) hypotension is a **sustained fall in systolic blood pressure of at least 20 mmHg, or in diastolic pressure of at least 10 mmHg, within 3 minutes of standing** (or of head-up tilt to 60°). Variants: **initial OH** (a transient fall within 15 seconds), **delayed OH** (beyond 3 minutes, requiring prolonged standing or tilt to detect), and **post-prandial hypotension** (a fall within 2 hours of a meal). **It is very common: a postural fall of more than 20 mmHg is found in about 30% of a large elderly population, and a fall of more than 40 mmHg in 10%. Baroreceptor responses become more sluggish with age**, giving an increased tendency to postural syncope.
2. **Causes — drugs first. Drugs are the commonest and most reversible cause.** Implicated agents include **ganglion blockers, diuretics, phenothiazines, antihistamines, antidepressants, benzodiazepines, barbiturates and antiparkinsonian drugs**; cardiovascular drugs are particularly important — **nitrates, beta-blockers and nifedipine** — and **donepezil may increase the risk of syncope**. Also alpha-blockers, ACE inhibitors, ARBs, opioids and alcohol. **Non-neurogenic causes: volume depletion (dehydration, haemorrhage, diarrhoea, vomiting, poor oral intake), sepsis, anaemia, adrenal insufficiency, prolonged bed rest and deconditioning, varicose veins, and haemodialysis** — orthostatic hypotension and syncope are common in the elderly following haemodialysis. **Neurogenic causes: autonomic neuropathy (diabetes, amyloidosis, Guillain–Barré, Shy–Drager/multiple system atrophy), Parkinson's disease, dementia with Lewy bodies, pure autonomic failure and spinal cord disease, particularly traumatic cervical cord lesions**.
3. **Clinical features. Light-headedness, dizziness, blurred or tunnel vision, "coat-hanger" ache across the neck and shoulders, generalised weakness, fatigue and cognitive slowing on standing**, progressing to **presyncope and syncope**. **In the older patient the presentation is frequently NOT dizziness but unexplained falls, drop attacks, transient confusion or functional decline, with no recall of loss of consciousness — so orthostatic hypotension must be actively sought in every faller. Symptoms are typically worst on rising in the morning, after meals, in hot weather, after exertion, after alcohol, and during intercurrent illness.**
4. **Evaluation.** Measure blood pressure and heart rate **supine after 5 minutes rest, then at 1 and 3 minutes of standing**; extend to 5–10 minutes if delayed OH is suspected. **A compensatory rise in heart rate suggests a non-neurogenic cause such as volume depletion, whereas a blunted or absent heart rate response suggests neurogenic autonomic failure.** Perform a **head-up tilt table test** if standing measurements are negative but the history is suggestive. Take a complete **drug history** and examine for **dehydration, anaemia, parkinsonism, peripheral neuropathy and cognitive impairment**. Investigations: **full blood count, urea, electrolytes, creatinine, glucose and HbA1c, B12, morning cortisol or short Synacthen test, thyroid function, ECG** and, where relevant, serum and urine electrophoresis for amyloid. **Exclude treatable conditions such as Addison's disease.** Screen for **supine hypertension**, which frequently coexists and complicates treatment.
5. **Non-pharmacological management — always first. Review the medication**; **it is not always possible to discontinue the relevant drugs — for example in Parkinson's disease the effect of dopaminergic drugs in aggravating the postural fall must be balanced against their therapeutic benefit.** Then: **avoid factors causing vasodilatation — high temperatures, marked exertion and alcohol**; eat **small frequent meals rather than large ones** (to limit post-prandial splanchnic pooling), and reduce refined carbohydrate; **avoid dehydration** and liberalise salt and fluid intake where not contraindicated. **Change posture slowly and reverse the change temporarily if symptoms supervene.** Teach **physical counter-manoeuvres — crossing the legs, tensing the calf muscles and abdominal compression**, which transiently ameliorate symptoms. **Elastic compression stockings** (or an abdominal binder) contribute significantly, though patients may need help putting them on and they are hard to tolerate in hot weather. **Sleeping with a slight head-up tilt** helps, possibly by expanding plasma volume, but more than about 10° is poorly tolerated. Encourage patients to **remain as active as possible**, since chronic exposure to low blood pressure appears to improve cerebral autoregulation.
6. **Drug treatment.** Add only if the above measures fail. **Most drugs are used outside their licence and the benefits may be limited. Fludrocortisone 100–200 µg at night is usually started first**, acting by salt and water retention — monitor for **hypokalaemia, oedema, heart failure and supine hypertension**. **Ephedrine**, a sympathomimetic, can be added at **15 mg three times daily, increasing if necessary to 30 mg three times daily**; higher doses cause **tremor, tachycardia and agitation**. Other agents used include **midodrine** (an α_1-agonist, given three times daily with the last dose at least 4 hours before lying down), **desmopressin** for nocturnal polyuria and morning postural hypotension, and **erythropoietin** where there is anaemia. **Supine hypertension is the main risk of all drug treatment** — manage it by head-up sleeping, avoiding daytime recumbency, and a short-acting nocturnal antihypertensive if needed. **Cardiac pacing does not help.** Finally, because **the risks of serious injury from falls are very high in this population**, the patient must understand the risks and take precautions to avoid attacks wherever possible.

Latest Update – 1 Point (2023–Present)

7. The **2024 ESC hypertension guideline** makes **routine screening for orthostatic hypotension a standard part of hypertension assessment in older adults**, recommending that standing blood pressure be measured at every visit and that **treatment be titrated to the standing rather than the seated pressure** where postural symptoms exist. Importantly, it also states that **orthostatic hypotension is not in itself a reason to leave hypertension untreated** — analyses from SPRINT and pooled trial data showed that intensive blood pressure lowering did **not** increase orthostatic hypotension with symptoms, and that **treating the underlying hypertension often improves rather than worsens orthostatic tolerance**. In parallel, the **AGS Beers Criteria 2023** continue to list drugs to avoid in patients with a history of syncope or falls, reinforcing medication review as the first therapeutic step.

D. Valvular and Myocardial Disease

Question 18. Aortic stenosis — clinical presentation, diagnosis and management

7-Mark Answer | 6 textbook points + 1 latest update | ***Asked ×3***

Textbook Core – 6 Points

1. **Epidemiology and aetiology. Acquired calcific degenerative aortic stenosis is the commonest valve disease in the elderly, with a prevalence of 2.8% in those aged 75 and over**, and it is found at autopsy in about 4–6% of those over 65. **Aortic valve cusp sclerosis or calcification is present in 26–29% of adults older than 65.** Isolated aortic stenosis is commoner in men except over the age of 80, where women predominate. Degenerative calcification was formerly thought to be passive calcium deposition, but is now known to share the **pathophysiology of vascular atherosclerosis** — lipid deposition, inflammatory cell infiltration, basement membrane disruption and calcification, a concept termed **"valvulo-metabolic risk"**. Other causes: **bicuspid valve** (presents in the 50s–60s) and **rheumatic disease**, still important in India.
2. **Clinical presentation.** The usual presentation in older adults is **fatigue, syncope, angina pectoris and congestive heart failure**; exertional dyspnoea is often the earliest symptom. **The development of symptoms — angina, syncope or heart failure — identifies the critical point in the natural history, because symptomatic aortic stenosis without intervention has only a 20% three-year survival.** Other features: exertional dizziness, sudden death, and **gastrointestinal bleeding from angiodysplasia with acquired von Willebrand disease (Heyde's syndrome)**. Note that **the asymptomatic state is difficult to establish in the elderly because of a gradual reduction in activity or a sedentary lifestyle** — patients unconsciously limit themselves.
3. **Physical signs and their unreliability in the old.** The classical findings are a **slow-rising, low-volume pulse (pulsus parvus et tardus), a narrow pulse pressure, a heaving sustained apex beat, a soft or absent second heart sound, an S4, and an ejection systolic murmur radiating to the carotids**. **However, the typical murmur and pulse of aortic stenosis may be modified in older adults: systemic hypertension may shorten the systolic murmur, giving it the character of an aortic sclerosis murmur, and loss of vascular elasticity may modify the pulse pressure so that the typical pulse contour is absent.** The murmur may also be maximal at the apex and fail to radiate (Gallavardin phenomenon), mimicking mitral regurgitation. **Physical examination alone is therefore not reliable for diagnosing aortic stenosis in older adults**, and because systolic murmurs are so common in this age group, differentiating mitral regurgitation, aortic sclerosis and aortic stenosis by auscultation is a genuine challenge.
4. **Diagnosis. The addition of Doppler flow studies to echocardiography has greatly improved diagnostic accuracy.** Severe aortic stenosis is defined by an **aortic valve area < 1.0 cm² (or < 0.6 cm²/m² indexed), a peak transvalvular velocity ≥ 4.0 m/s, and a mean gradient ≥ 40 mmHg**. Be alert to **low-flow, low-gradient severe aortic stenosis** (small valve area with a low gradient) — with a **reduced ejection fraction**, use **dobutamine stress echocardiography** to distinguish true severe stenosis from pseudo-severe; with a **preserved ejection fraction**, use the **calcium score on CT**. **Left ventricular catheterisation remains the most reliable method of assessing aortic stenosis in older adults but should be reserved for patients who are symptomatic (angina, syncope or dyspnoea) and in whom surgery is contemplated.** An **exercise test is recommended to determine whether an apparently asymptomatic patient is truly asymptomatic** — but is **contraindicated once symptoms are present**. Add **BNP** and coronary angiography before intervention.
5. **Management — general. There is no effective medical therapy that alters the natural history; valve replacement is the only definitive treatment.** Medical care is supportive: cautious diuretics for congestion, control of hypertension (avoid excessive vasodilatation), rate control and prompt cardioversion of atrial fibrillation, and treatment of coexisting coronary disease. **Avoid nitrates and high-dose vasodilators in severe stenosis** because a fixed cardiac output makes profound hypotension likely. Statins do not slow progression. **Follow asymptomatic patients with serial echocardiography** and educate them to report symptoms immediately.

6. **Intervention — SAVR versus TAVI. Survival of the elderly patient after successful surgical aortic valve replacement is similar to that of the general population and enables return to an independent, active life.** Operative mortality approaches that of younger patients, and **it is not until patients reach their 80s that age alone becomes a risk factor**; five-year survival for UK octogenarians undergoing SAVR is about 65% (about 55% with concomitant CABG). **Once symptomatic aortic valve disease is diagnosed in a patient aged 80 or more, early referral for valve replacement avoids the hazards of falling LV function, loss of contractile reserve, urgent surgery and sudden death. TAVI** improves five-year survival compared with medical treatment in inoperable patients, is equivalent to SAVR in high-risk patients with possible peri-procedural advantage, and is now used in intermediate and lower-risk patients; **the choice between SAVR and TAVI must be made by the Heart Team** on clinical, anatomical and technical grounds. Considerations favouring one over the other include the **lower rate of paravalvular leak with SAVR, the higher pacemaker requirement with TAVI, and uncertain long-term TAVI valve durability**. A **bioprosthesis is generally preferred over a mechanical valve above 65 years**, since mortality from thromboembolism and anticoagulant-related haemorrhage is three times higher with mechanical valves in this group. **Balloon aortic valvuloplasty is no longer a definitive option** — mortality 3–10%, stroke 10–25%, 66% restenosis within six months, and fewer than 20% surviving a year — but retains a role as a **bridge** in very sick patients. Finally, **identify futility**: life expectancy under two years from malignancy, severe airways disease, established dementia or severe frailty, an STS score > 15%, dialysis dependence or home oxygen dependence all predict that even TAVI will not benefit the patient.

Latest Update – 1 Point (2023–Present)

7. **EARLY TAVR (NEJM 2024)** challenged the long-standing rule of watchful waiting in asymptomatic disease. In patients with **asymptomatic severe aortic stenosis and a normal ejection fraction**, **early TAVI significantly reduced the composite of death, stroke or unplanned cardiovascular hospitalisation compared with clinical surveillance**, driven largely by fewer unplanned hospitalisations — and notably **26% of the surveillance group required valve replacement within six months**, underlining how quickly these patients declare themselves. This supports a strategy of **earlier intervention in selected asymptomatic patients**, and is particularly relevant in older adults in whom, as the textbook notes, **the "asymptomatic" state is unreliable because activity is self-limited**.

Question 19. Aortic sclerosis

7-Mark Answer | 6 textbook points + 1 latest update

Textbook Core – 6 Points

1. **Definition.** Aortic sclerosis is **focal thickening, fibrosis and calcification of the aortic valve leaflets WITHOUT significant obstruction to left ventricular outflow** — by convention a **peak transvalvular velocity below 2.5 m/s with normal leaflet excursion**. It is the **earliest, non-obstructive stage of the same disease process that culminates in calcific degenerative aortic stenosis**.
2. **Prevalence.** It is very common. **Aortic valve cusp sclerosis or calcification is present in 26% of adults older than 65**, and in the Cardiovascular Health Study the prevalence was **29% of the elderly**, rising to about **40–50% of those over 80**. Roughly **10–15% progress to haemodynamically significant aortic stenosis over 5–10 years**, at an average rate of about 1–2% per year.
3. **Pathogenesis — an atherosclerosis-like process.** Degenerative aortic valve disease was for many years thought to result from the **passive accumulation of calcium on the surface of the valve leaflet**; it is now established that its **aetiology has a pathophysiology similar to that of vascular atherosclerosis**. Early stages resemble active atherosclerotic inflammation — **basement membrane disruption, lipid deposition, inflammatory cell infiltration and calcification**, with later osteoblastic differentiation of valve interstitial cells. Shared risk factors — **age, male sex, smoking, hypertension, diabetes, dyslipidaemia (particularly raised Lp(a)), chronic kidney disease and visceral obesity** — give rise to the concept of **valvulo-metabolic risk**. Note that **only about 50% of patients with degenerative aortic valve disease have significant coronary disease, and most patients with coronary disease do not have valve disease**, so additional factors must be involved.
4. **Clinical features.** Aortic sclerosis is **asymptomatic by definition** and is usually found incidentally. The sign is an **ejection systolic murmur, loudest at the aortic area, typically early-peaking, short and grade 1–2/6**, and crucially it is accompanied by a **normal carotid upstroke, a normal pulse pressure and a normally preserved (often accentuated) aortic component of the second heart sound**. There is no left ventricular hypertrophy attributable to the valve.

5. **Distinguishing sclerosis from stenosis.** This is the crux of the answer, and **it can be difficult in older adults on clinical grounds alone**.

Feature	Aortic sclerosis	Aortic stenosis
Symptoms	None	Angina, syncope, heart failure
Carotid upstroke	**Normal**	**Slow-rising, low volume (parvus et tardus)**
Murmur peak	Early systolic, short	Mid-to-late peaking, long, harsh
Second heart sound (A2)	Normal or accentuated	Soft, single or absent
Apex beat	Normal	Sustained, heaving
Peak velocity / gradient	**< 2.5 m/s**, no gradient	**≥ 2.5 m/s**; severe if ≥ 4.0 m/s or mean gradient ≥ 40 mmHg

   Two textbook caveats make the clinical distinction unreliable: **systemic hypertension may shorten the systolic murmur of stenosis, giving it the character of a sclerosis murmur**, and **loss of vascular elasticity may modify the pulse pressure so that the typical pulse contour of stenosis is absent**. **Doppler echocardiography is therefore mandatory in any older patient with a systolic murmur.** Bedside manoeuvres also help differentiate the systolic murmurs of the elderly: the murmurs of both sclerosis and stenosis **increase after a premature ventricular contraction and with amyl nitrite and squatting, and decrease with Valsalva**, whereas the murmur of **hypertrophic obstructive cardiomyopathy increases with Valsalva and decreases with squatting**, and **mitral regurgitation does not increase after a premature beat**.

6. **Prognostic significance and management. Aortic sclerosis is NOT a benign incidental finding: it is associated with an increase in the risk of death from cardiovascular causes and the risk of myocardial infarction, even in the absence of haemodynamically significant obstruction of left ventricular outflow — an approximately 50% relative increase in cardiovascular death and myocardial infarction in the Cardiovascular Health Study. It should therefore be treated as a marker of systemic atherosclerotic burden and a trigger for comprehensive cardiovascular risk assessment: assess and treat blood pressure, lipids, diabetes, smoking, weight and renal function. No treatment retards progression of the valve lesion itself — statins have been shown not to slow progression despite the atherosclerotic pathogenesis, since 60–70% of patients with degenerative valve disease are already dyslipidaemic and on statins yet still progress rapidly, suggesting that targeting the entire metabolic risk rather than lipids alone may be more effective. Management is therefore risk factor modification plus surveillance: repeat echocardiography every 3–5 years, or sooner if symptoms develop or the murmur changes. Antibiotic prophylaxis for infective endocarditis is not recommended for aortic sclerosis.**

Latest Update – 1 Point (2023–Present)

7. **Lipoprotein(a) has emerged as the strongest causal lipid driver of calcific aortic valve disease**, and it is the one modifiable factor that distinguishes valve disease from ordinary atherosclerosis. Mendelian randomisation and cohort data show that **elevated Lp(a) and the LPA risk genotype are causally associated with aortic valve calcification, incident aortic stenosis and faster progression from sclerosis to stenosis** — which explains why statins, which do not lower Lp(a), have repeatedly failed to slow valve disease. Current **ESC/EAS and 2025 AHA/ACC guidance recommends measuring Lp(a) at least once in every adult**, and **Lp(a)-lowering agents (pelacarsen in Lp(a)HORIZON, olpasiran, lepodisiran) are in phase 3 trials**, offering the first prospect of disease-modifying therapy for calcific aortic valve disease.

Question 20. Mitral regurgitation in the elderly

6-Mark Answer | 5 textbook points + 1 latest update

Textbook Core – 5 Points

1. **Definition and classification.** Mitral regurgitation (MR) is **systolic backflow from the left ventricle into the left atrium** through an incompetent mitral valve. Classify as **PRIMARY (degenerative/organic) — a structural abnormality of the leaflets, chordae or annulus**, versus **SECONDARY (functional) — a structurally normal valve failing because of left ventricular or left atrial remodelling**. This distinction determines the entire management pathway. Also classify by **acute versus chronic**.

2. **Causes in older adults. Primary: mucoid (myxomatous) degeneration with mitral valve prolapse** — mucoid degeneration affects mainly the mitral valve, allowing the leaflet to stretch under normal intracardiac pressure and prolapse into the left atrium in systole; it is found in about **1% of autopsies over the age of 65**. Also **mitral annular calcification**, a disease of older adults found in **9% of autopsies over 50, rising strikingly with age and particularly in women — from 3.2% under 70 to 44% over 90** — which **often results in mitral insufficiency or conduction abnormalities and rarely stenosis, is an important contributor to congestive heart failure in older adults, and is a site for endocarditis**. Other primary causes: **infective endocarditis, rheumatic disease (important in India), and chordal rupture**, which is the usual mechanism of death directly from mucoid degeneration. **Secondary: ischaemic MR after myocardial infarction, dilated cardiomyopathy, and atrial functional MR from long-standing atrial fibrillation with left atrial and annular dilatation** — increasingly recognised as a distinct and common entity in the very old.
3. **Clinical features. Chronic MR** is often long asymptomatic, then presents with **exertional dyspnoea, fatigue, orthostatic intolerance, palpitations and atrial fibrillation**, and finally with **pulmonary hypertension and right heart failure**. **Acute severe MR** — from papillary muscle rupture after myocardial infarction or from endocarditis — presents as **abrupt pulmonary oedema and cardiogenic shock, often with only a soft or absent murmur** because atrial and ventricular pressures rapidly equalise. Signs of chronic MR: a **pansystolic murmur at the apex radiating to the axilla, a soft S1, a third heart sound, a displaced hyperdynamic apex beat** and, in mitral valve prolapse, a **mid-systolic click with a late systolic murmur** — though the murmur of prolapse can occur at any time in systole. Distinguishing features on manoeuvres: **the murmur of mitral regurgitation does NOT increase after a premature ventricular contraction and decreases with amyl nitrite and Valsalva**, unlike the aortic murmurs. **Mitral insufficiency caused by mucoid degeneration is usually well tolerated and rarely requires surgery**; with mitral annular calcification the regurgitation is **usually mild to moderate**, and **as many as two-thirds of patients with mitral annular calcification present with an apical systolic murmur of mitral regurgitation**.
4. **Investigations. Echocardiography is the best technique for diagnosing mitral annular calcification** and for assessing MR overall — it defines the **mechanism, severity (regurgitant volume, effective regurgitant orifice area, vena contracta), left ventricular size and ejection fraction, left atrial size and pulmonary artery pressure**. **Transoesophageal echocardiography** is used to define anatomy before intervention and to detect endocarditis. Add **ECG** (atrial fibrillation, left atrial enlargement, conduction abnormality with annular calcification), **chest radiograph**, **BNP**, **exercise echocardiography** for exertional symptoms with apparently moderate MR, and **coronary angiography** before surgery. **In severe chronic MR the ejection fraction is spuriously flattered by the low-impedance leak, so an EF below 60% already indicates significant left ventricular dysfunction.**
5. **Management. Acute severe MR** is a surgical emergency: stabilise with **afterload reduction (nitroprusside), diuretics and an intra-aortic balloon pump if hypotensive**, and proceed to urgent surgery. **Chronic primary MR:** there is no medical therapy that alters the natural history — **surgery (mitral valve repair in preference to replacement) is indicated for symptomatic severe MR, or for asymptomatic severe MR with an ejection fraction ≤ 60%, an end-systolic diameter ≥ 40 mm, new atrial fibrillation or pulmonary hypertension**. **Chronic secondary MR:** the first step is **optimal guideline-directed heart failure therapy plus cardiac resynchronisation and revascularisation where indicated**, because functional MR frequently improves with successful treatment of the ventricle. **Surgery is indicated only after this has failed**, and there is a well-established survival benefit from **correcting coexistent moderate or greater ischaemic mitral regurgitation at the time of CABG surgery**. Additional geriatric points: **anticoagulate for atrial fibrillation**; note that **mitral annular calcification carries a higher incidence of cerebral embolism, so anticoagulation with warfarin may be indicated**; **surgery for annular calcification is usually indicated only if endocarditis is superimposed**; and **a bioprosthesis is preferred over 70 years for a valve in the mitral position**. Subacute bacterial endocarditis prophylaxis is no longer recommended for mitral valve prolapse.

Latest Update – 1 Point (2023–Present)

6. **Transcatheter edge-to-edge repair (TEER, "MitraClip") is now established for older patients who are unsuitable for surgery.** Building on COAPT, two 2024 randomised trials in **secondary MR with heart failure** — **RESHAPE-HF2** and **MATTERHORN (both NEJM 2024)** — showed that **TEER added to guideline-directed medical therapy reduced heart failure hospitalisation and improved symptoms and quality of life**, with MATTERHORN additionally showing non-inferiority to surgery for the composite of death, hospitalisation, reintervention and assist device implantation, but with **significantly fewer major adverse events**. Because it is performed percutaneously without cardiopulmonary bypass, TEER is **especially attractive in the frail elderly**, and current ACC/AHA and ESC valve guidance recommends it for appropriately selected patients with severe symptomatic MR at high or prohibitive surgical risk, after Heart Team assessment.

Question 21. Infective endocarditis in the elderly

7-Mark Answer | 6 textbook points + 1 latest update

Textbook Core – 6 Points

1. **Definition and burden.** Infective endocarditis (IE) is infection of the **endocardium**, usually of the heart valves. In people over 65 the incidence is about **four times** that of the general population and mortality is about **twice** that of younger patients.
2. **Predisposing factors. Undiagnosed degenerative (calcific) aortic valve disease** and increased use of **invasive procedures and implanted devices** (pacemakers, prosthetic valves, intravascular catheters) are the main reasons for the rise. Other risks are **diabetes** and **gastrointestinal and genitourinary tract cancers**. Mitral annular calcification and mucoid mitral valve degeneration also predispose. In India, **rheumatic valvular disease** remains an important substrate.
3. **Microbiology changes with age.** The proportion due to **Staphylococcus aureus falls with age**, while **enterococci** and **Streptococcus bovis (gallolyticus)** emerge as major pathogens. Isolation of **S. bovis mandates colonoscopy** to look for colonic neoplasm. Viridans streptococci still predominate where rheumatic heart disease is common; coagulase-negative staphylococci cause device-related IE.
4. **Atypical presentation.** Fever may be **absent or low grade**. The older patient often presents with **delirium, anorexia, weight loss, falls, fatigue or unexplained anaemia** with a raised ESR/CRP. A **new or changing murmur** may be the only sign. Classical peripheral stigmata (Osler nodes, Janeway lesions, Roth spots, splinter haemorrhages) are **uncommon**, so diagnosis is frequently delayed.
5. **Diagnosis.** Take **three sets of blood cultures** from separate sites before antibiotics. **Transthoracic echocardiography** first; proceed to **transoesophageal echocardiography** if TTE is negative, or if there is a prosthetic valve or intracardiac device. Apply the **modified Duke criteria**. Send serology for culture-negative causes (Coxiella burnetii, Bartonella, Brucella).
6. **Management. Prolonged intravenous bactericidal antibiotics for 4–6 weeks**, guided by culture and sensitivity, with careful **renal-adjusted aminoglycoside dosing**. **Surgery** is indicated for heart failure, uncontrolled infection/abscess, prosthetic valve dehiscence, and large mobile vegetations with embolism — **age alone is not a contraindication**, though frailty raises operative risk. **Antibiotic prophylaxis strategy should take age into account** in global risk assessment before invasive gastrointestinal or genitourinary procedures.

Latest Update – 1 Point (2023–Present)

7. **2023 ESC Guidelines on Infective Endocarditis** adopt the **2023 Duke–ISCVID criteria**, which add **cardiac CT angiography** and **^{18}F-FDG PET/CT** as major imaging criteria (especially valuable for prosthetic valve and device infection in older adults). They also endorse **partial oral step-down antibiotic therapy** after at least 10 days of intravenous treatment in stabilised left-sided IE (**POET trial**), and **outpatient parenteral antibiotic therapy** — both reduce prolonged hospitalisation and deconditioning in frail elderly patients.

Current Practice Update

Textbook	Diagnosis by blood cultures, echocardiography and the modified Duke criteria; full course of intravenous antibiotics.
Current recommendation	2023 ESC / Duke–ISCVID criteria include cardiac CT and FDG-PET/CT as major criteria; partial oral therapy and OPAT are accepted in stabilised patients.
Exam approach	Write the modified Duke criteria, then add one line: "2023 ESC now incorporates the Duke–ISCVID criteria with advanced imaging and permits partial oral therapy (POET)."

Question 22. Cardiomyopathies — classification and management

7-Mark Answer | 6 textbook points + 1 latest update

Source note: Cardiomyopathy is **not covered adequately as a standalone topic in the uploaded textbook** (Pathy's refers to dilated and hypertrophic cardiomyopathy mainly as causes of sudden cardiac death; Essentials of Clinical Geriatrics covers idiopathic hypertrophic subaortic stenosis). Points 1–6 below are constructed from those textbook references supplemented by current standard guidance, which is clearly labelled.

Textbook Core – 6 Points

1. **Definition and classification.** A cardiomyopathy is a **myocardial disorder with structural and functional abnormality not explained by coronary disease, hypertension or valve disease**. Morphological types: **Dilated (DCM)**, **Hypertrophic (HCM)**, **Restrictive (RCM)**, **Arrhythmogenic right ventricular (ARVC)**, and **unclassified** (Takotsubo, left-ventricular non-compaction).

2. **Geriatric relevance. Dilated and hypertrophic cardiomyopathy are important causes of sudden cardiac death** in older people after coronary disease. **Wild-type transthyretin cardiac amyloidosis (ATTR-wt)** is the commonest restrictive cardiomyopathy of old age — suspect it in **HFpEF with left ventricular hypertrophy but low-voltage ECG**, bilateral carpal tunnel syndrome, lumbar canal stenosis, biceps tendon rupture and intolerance of antihypertensives.
3. **Dilated cardiomyopathy.** Dilated left ventricle with **systolic dysfunction**. Causes: ischaemic, **alcohol**, chemotherapy (**anthracyclines, trastuzumab**), thyroid disease, tachycardia-mediated, myocarditis, peripartum. Treatment is **four-pillar heart failure therapy — ARNI or ACE inhibitor, beta-blocker, MRA and SGLT2 inhibitor** — titrated slowly, with diuretics for congestion.
4. **Hypertrophic cardiomyopathy.** Asymmetric septal hypertrophy with **dynamic left ventricular outflow tract obstruction**; in older adults it may be mistaken for aortic stenosis and is described as **idiopathic hypertrophic subaortic stenosis (IHSS)**. The murmur **increases with Valsalva and standing** and **decreases with squatting**; a **bisferiens pulse** with no aortic regurgitant murmur is a clue. Diagnosis is by **echocardiography**. Therapy relies on **beta-blockers**; **avoid digoxin, diuretics, nitrates and vasodilators** which worsen obstruction. **Atrial fibrillation is poorly tolerated** and may need cardioversion.
5. **Restrictive cardiomyopathy. Impaired ventricular filling with normal or near-normal ejection fraction** and biatrial enlargement. Causes: **amyloidosis**, haemochromatosis, radiation, sarcoidosis and **endomyocardial fibrosis** (relevant in India). Diagnosis: echocardiography with **apical sparing on strain imaging**, cardiac MRI, **^{99m}Tc-PYP/DPD bone scintigraphy** for ATTR, and exclusion of a monoclonal protein (serum free light chains, immunofixation). Must be distinguished from **constrictive pericarditis**.
6. **Management principles in older adults.** Treat heart failure cautiously with attention to **preload dependence**; control and anticoagulate **atrial fibrillation**; correct anaemia and thyroid dysfunction. **ICD for sudden death prevention** only after weighing life expectancy, frailty and patient wishes. In amyloidosis **avoid digoxin, calcium channel blockers and high-dose beta-blockers**; use **tafamidis** as disease-modifying therapy in ATTR-CM. Genetic counselling and family screening for inherited forms.

Latest Update – 1 Point (2023–Present)

7. The **2023 ESC Guidelines on Cardiomyopathies** — the first standalone ESC cardiomyopathy guideline — adopt a **phenotype-based approach** with multimodality imaging and genetic testing for all patients and relatives. For the older patient, disease-modifying therapy for **ATTR cardiac amyloidosis** has expanded rapidly: after tafamidis (ATTR-ACT), the **ATTRibute-CM** trial (acoramidis, NEJM 2024) and **HELIOS-B** (vutrisiran, NEJM 2025) both reduced death and cardiovascular hospitalisation; and **mavacamten**, a cardiac myosin inhibitor (EXPLORER-HCM), is now approved for symptomatic obstructive HCM.

E. Arrhythmias and Cardiac Pacing

Question 23. Complete heart block and pacemaker indications

7-Mark Answer | 6 textbook points + 1 latest update

Textbook Core – 6 Points

1. **Definition.** Third-degree (complete) heart block is **complete failure of conduction between atria and ventricles**, producing regular atrial activity with an **independent escape rhythm**. The escape is usually ventricular — **wide QRS at 30–40 beats per minute**. A nodal or junctional escape (narrow QRS, faster) means the block is higher, within the AV node or bundle of His. **The lower the origin of the escape rhythm, the less reliable it is.**
2. **Causes in the elderly. Idiopathic age-related fibrosis of the conduction system** (Lev and Lenègre disease) is commonest. Others: **ischaemic heart disease and inferior myocardial infarction**, drugs (**beta-blockers, digoxin, verapamil, diltiazem, amiodarone**), **hyperkalaemia**, calcific aortic valve disease and post-TAVI, infiltrative disease (**sarcoidosis, amyloidosis**), **neuromuscular disease** (myotonic dystrophy type 1, Kearns–Sayre syndrome), and cardiac surgery or AV junction ablation.
3. **Clinical features. Stokes–Adams attacks** (abrupt syncope without warning), presyncope, **falls**, fatigue, dyspnoea, angina and heart failure. Signs: slow regular pulse, **cannon 'a' waves** in the JVP, **variable intensity of the first heart sound** and a wide pulse pressure. Complete heart block is a classic cardiac cause of syncope in old age.
4. **Class I indications for permanent pacing.** (i) **Acquired complete heart block not attributable to a reversible or physiological cause — regardless of symptoms**. (ii) Complete heart block due to **neuromuscular disease with conduction disorder** (myotonic dystrophy type 1, Kearns–Sayre). (iii) **Symptomatic complete heart block occurring as a consequence of guideline-directed drug therapy that must be continued**.

5. **Related Class I / IIa indications. Mobitz type II second-degree block** not attributable to reversible causes should be paced **regardless of symptoms** (Class I); so should **unresolving advanced second-degree block after cardiac surgery or AV junction ablation** (Class I). Pacing is **reasonable (Class IIa)** in **infiltrative cardiomyopathy such as cardiac sarcoidosis with Mobitz type II block**, and in **Mobitz type I block when symptoms are clearly attributable to the block**.
6. **Pacemaker choice and complications. DDD (dual-chamber)** pacing preserves AV synchrony and is preferred in complete heart block with sinus rhythm; **VVI/R** is used when there is permanent atrial fibrillation. Pacing that maintains physiological ventricular activation (**CRT or His-bundle pacing**) should be considered when the **ejection fraction is below 50% and ventricular pacing is expected more than 40% of the time** (Class IIa). Complications: **pneumothorax 1–2% with the subclavian approach but under 0.1% with cephalic or axillary access**, tamponade under 0.2%, infection under 1%, lead displacement; early complications occur in about **7% within two weeks** and later complications in up to 10%. Risk factors are **female sex and low body mass index** — **increasing age itself is not associated with higher complication risk**. In cachectic patients with thin skin or skeletal deformity, a **sub-pectoral** position reduces erosion.

Latest Update – 1 Point (2023–Present)

7. The **2023 HRS/APHRS/LAHRS Guideline on Cardiac Physiologic Pacing** establishes **conduction system pacing** as a recommended alternative to conventional right-ventricular pacing when a **high ventricular pacing burden is anticipated** — as in complete heart block. **Left bundle branch area pacing (LBBAP)** has largely superseded His-bundle pacing because of better capture thresholds and lead stability. **Leadless pacemakers** (Micra AV2, Aveir) are an option for AV block in frail older patients with poor venous access, thin skin or high infection risk.

Current Practice Update

Textbook	Choice lies between VVI/R, AAI, DDD and CRT; direct His-bundle pacing is "increasingly popular" but technically challenging.
Current recommendation	2023 HRS physiologic pacing guideline — left bundle branch area pacing is the preferred conduction system pacing technique; leadless devices are established for selected patients.
Exam approach	Give the classical mode selection, then add: "2023 HRS guideline now recommends conduction system pacing (LBBAP) when high ventricular pacing burden is expected."

Question 24. Sick sinus syndrome

7-Mark Answer | 6 textbook points + 1 latest update

Textbook Core – 6 Points

1. **Definition.** Sick sinus syndrome (**sinus node dysfunction**) is a spectrum of **inappropriate sinus bradycardia, sinus arrest or pause, sinoatrial exit block, chronotropic incompetence**, and **tachycardia-bradycardia syndrome**. Dysfunction may progress until **no sinus beats occur** and a junctional escape rhythm takes over. It is **primarily a disease of older people**, resulting from **degenerative or ischaemic change** that may also involve the AV node.
2. **Causes. Idiopathic age-related fibrosis** of the sinus node is commonest. Others: **ischaemic heart disease**, drugs (**beta-blockers, verapamil, diltiazem, digoxin, amiodarone, ivabradine, lithium, donepezil**), **hypothyroidism**, hyperkalaemia, infiltrative disease (amyloid, sarcoid), obstructive sleep apnoea, and cardiac surgery. **Always exclude a drug cause before implanting a pacemaker.**
3. **Clinical features. Recurrent syncope** from sinus pauses with an inadequate escape rhythm, presyncope, dizziness, **unexplained falls**, fatigue, confusion, and **exercise intolerance from chronotropic incompetence**. In **tachy-brady syndrome**, palpitations from atrial fibrillation or flutter are followed by a **long post-tachycardia pause** and syncope on termination. Many older patients are asymptomatic. **Both paroxysmal and chronic atrial fibrillation are commonly associated** with sinus node disease.
4. **Diagnosis.** Resting ECG, then **ambulatory Holter monitoring**; if symptoms are infrequent use **prolonged external monitoring or an implantable loop recorder**. **Symptom–rhythm correlation is essential** — an abnormal ECG without symptoms does not justify pacing. **Exercise testing** demonstrates chronotropic incompetence. Electrophysiological study (sinus node recovery time) is rarely required. Check **thyroid function, electrolytes and the drug chart**.
5. **Prognosis. Mortality appears to be unaffected by sinus node dysfunction itself**; survival is determined by **associated pathology such as ischaemic heart disease**. The main morbidity is **syncope with injury** and **thromboembolic stroke from associated atrial fibrillation** — assess stroke risk and anticoagulate accordingly, independently of the pacing decision.

6. **Management and pacing indications.** First **withdraw culprit drugs** and correct reversible causes. Pacing is **Class I** when (i) **symptoms are directly attributable to sinus node dysfunction**, and (ii) **sinus bradycardia develops secondary to guideline-directed treatment of another condition which must be continued**. Pacing **should be considered (Class IIa)** in **tachy–brady syndrome where symptoms are attributable to the bradycardia**, and **rate-responsive pacing** should be considered for **symptomatic chronotropic incompetence**. **AAI pacing** is used only in pure sinus node disease with no existing or expected disease elsewhere in the conduction system; otherwise **DDD(R)** is preferred, since VVI pacing causes pacemaker syndrome and more atrial fibrillation.

Latest Update – 1 Point (2023–Present)

7. The **2023 HRS/APHRS/LAHRS Cardiac Physiologic Pacing Guideline** reinforces that in sinus node dysfunction with intact AV conduction the aim is to **preserve intrinsic ventricular activation** — use **atrial-based (AAI/DDD) pacing** and enable **algorithms that minimise unnecessary right-ventricular pacing**, since a high right-ventricular pacing burden increases atrial fibrillation and heart failure. Conduction system pacing is reserved for those who also need ventricular pacing.

Question 25. Atrial fibrillation

7-Mark Answer | 6 textbook points + 1 latest update

Textbook Core – 6 Points

1. **Definition and epidemiology.** Atrial fibrillation (AF) is the **most common sustained cardiac arrhythmia**, characterised by **uncoordinated atrial activation with consequent loss of atrial mechanical function**. It is now regarded as a **multifactorial disease** caused by an interplay of cardiovascular and metabolic risk factors, systemic inflammation, and anatomical and electrical substrates. In 2016 more than **43 million people** were estimated to have AF; the **lifetime risk at age 55 is about 35%**. It is **1.5 times more common in men**, though onset is later in women (65 versus 60 years).
2. **Risk factors. All the classical cardiovascular risk factors are significantly associated with incident AF.** Each **10 mmHg rise in systolic blood pressure increases the risk of developing AF by 11–16%**; in diabetes there is a **dose–response relationship between HbA1c and incident AF**; and there is a **strong association between increasing age and increasing systemic inflammation as measured by C-reactive protein**. Others: heart failure, valvular disease (especially rheumatic mitral disease in India), obesity, obstructive sleep apnoea, alcohol, thyrotoxicosis, chronic kidney disease and cardiac surgery. **The risk of incident AF is directly related to the total burden of risk factors, rising progressively with the number present.**
3. **Clinical features and consequences.** Presentation may be with **palpitations, breathlessness, fatigue, chest pain, dizziness or syncope**, but AF is frequently **asymptomatic and detected incidentally** — or presents first as **stroke, heart failure or falls**. In older people the loss of the atrial contribution to ventricular filling is critical, because **they depend more on atrial contraction than on ventricular relaxation for left ventricular filling and therefore decompensate rapidly into heart failure**. Consequences: **up to 30% of all ischaemic strokes are due to AF**; **20–30% of AF patients develop heart failure**; there is an **up to 60% relative increase in risk of significant cognitive decline**; **more than 60% report impaired quality of life**; and AF causes an **up to 3.5-fold increase in cardiovascular and all-cause death**. The pulse is **irregularly irregular** with an apex–radial deficit — but note that **frequent atrial ectopic beats, common in the old, can produce a pulse that is confused with AF**, so confirm with an ECG and obtain a transthoracic echocardiogram if not previously done.
4. **Diagnosis and assessment.** Confirm with a **12-lead ECG showing absent P waves, an irregularly irregular QRS rhythm and a fibrillatory baseline**. For paroxysmal AF use **Holter monitoring, extended patch monitoring, an implantable loop recorder or a smartphone/wearable single-lead ECG**. Classify as **first-diagnosed, paroxysmal, persistent, long-standing persistent or permanent**. Investigate with **echocardiography** (left atrial size, valve disease, ejection fraction, left ventricular hypertrophy), **thyroid function, full blood count, renal and liver function, electrolytes**, and screen for **sleep apnoea and alcohol excess**.
5. **Anticoagulation — the priority.** Assess stroke risk with **CHA_2DS_2-VASc** and bleeding risk with **HAS-BLED**. Anticoagulate men scoring ≥ 2 and women ≥ 3. **A high HAS-BLED score is not a reason to withhold anticoagulation — it identifies modifiable risk factors to correct and prompts earlier review. DOACs are preferred over warfarin** in non-valvular AF; **apixaban has the best geriatric profile** (lowest major and gastrointestinal bleeding, safest in chronic kidney disease). **Warfarin is mandatory in mechanical prosthetic valves and moderate-to-severe rheumatic mitral stenosis.** Falls risk is a poor reason to withhold treatment — a patient would need to fall about **295 times a year** for the bleeding risk to outweigh the stroke benefit. Consider **left atrial appendage occlusion** when anticoagulation is absolutely contraindicated. In practice virtually every patient aged 75 or over already scores ≥ 2, so **anticoagulation is the default**.

6. **Rate versus rhythm control and integrated care. Rate control** is the usual first strategy in the elderly, with a resting target of **< 110/min (lenient) tightened to < 80/min if symptoms persist**: use a **beta-blocker or a rate-limiting calcium channel blocker (diltiazem, verapamil — avoid in heart failure)**, with **digoxin** as an add-on, particularly in the sedentary or hypotensive, at reduced dose with level monitoring. **Rhythm control** is indicated for persistent symptoms despite rate control, recent-onset AF, AF-related heart failure, or a young patient: options are **DC cardioversion (with 3 weeks of anticoagulation before and 4 weeks after, or a TOE-guided approach), antiarrhythmic drugs (amiodarone is the most usable in structural heart disease but has thyroid, hepatic, pulmonary, ocular and neurological toxicity in long-term use; flecainide and propafenone only with structurally normal hearts), and catheter ablation**, which is more effective than drugs and is reasonable in selected fitter older patients. **Pace-and-ablate (AV node ablation with pacing)** is an option for refractory rapid AF. Manage the whole patient with the **ABC pathway — Avoid stroke with anticoagulation, Better symptom control, Comorbidity and risk factor management**, which has been shown to improve outcomes.

Latest Update – 1 Point (2023–Present)

7. The **2024 ESC Guidelines for the Management of Atrial Fibrillation** made three examinable changes. First, **CHA_2DS_2-VASc has been replaced by CHA_2DS_2-VA — sex category is removed (maximum 7)**, with anticoagulation recommended at a score of ≥ 2 and considered at 1, on the grounds that female sex is an age-dependent risk modifier rather than an independent risk factor. Second, the ABC pathway has been reframed as the **AF-CARE pathway** — **C**omorbidity and risk factor management, **A**void stroke and thromboembolism, **R**educe symptoms by rate and rhythm control, and **E**valuation with dynamic reassessment — deliberately placing **comorbidity management first**. Third, following **EAST-AFNET 4** and **CASTLE-AF**, there is stronger support for **early rhythm control within 12 months of diagnosis** and for **catheter ablation as first-line therapy in paroxysmal AF and in AF with heart failure and reduced ejection fraction**.

Current Practice Update

Textbook	CHA_2DS_2-VASc with sex category; ABC pathway; rate control as the default strategy.
Current recommendation	2024 ESC — CHA_2DS_2-VA (sex removed); AF-CARE pathway with comorbidity management first; early rhythm control and first-line ablation in selected patients.
Exam approach	Draw CHA_2DS_2-VASc (still examinable), then add: "2024 ESC has replaced it with CHA_2DS_2-VA and the ABC pathway with AF-CARE."

Question 26. CHA2DS2-VASc and HAS-BLED scores in atrial fibrillation

7-Mark Answer | 6 textbook points + 1 latest update

Textbook Core – 6 Points

1. **Purpose. CHA_2DS_2-VASc** estimates the annual risk of **ischaemic stroke and systemic thromboembolism** in non-valvular atrial fibrillation and decides who needs **oral anticoagulation**. **HAS-BLED** estimates **major bleeding risk** on anticoagulation. Atrial fibrillation causes **up to 30% of all ischaemic strokes** and a **3.5-fold increase** in cardiovascular and all-cause death.
2. **CHA_2DS_2-VASc components (maximum 9).**

Letter	Risk factor	Score
C	Congestive heart failure / LV dysfunction	1
H	Hypertension	1
A_2	**Age ≥ 75 years**	**2**
D	Diabetes mellitus	1
S_2	**Prior Stroke / TIA / thromboembolism**	**2**
V	Vascular disease (prior MI, PAD, aortic plaque)	1
A	Age 65–74 years	1
Sc	Sex category (female)	1

3. **Interpretation of CHA_2DS_2-VASc. Men ≥ 2 and women ≥ 3** → oral anticoagulation **recommended**. Men = 1, women = 2 → anticoagulation **should be considered**. Men = 0, women = 1 → **no antithrombotic therapy**. **Aspirin is not an acceptable alternative to anticoagulation.** Practically, almost every patient aged 75 or over already scores 2 or more — **age alone makes anticoagulation the default in geriatric practice**.

4. **HAS-BLED components (maximum 9). H**ypertension (uncontrolled systolic BP > 160 mmHg) 1; **A**bnormal renal **or** liver function, 1 each; **S**troke 1; **B**leeding history or predisposition (including anaemia) 1; **L**abile INR (time in therapeutic range < 60%) 1; **E**lderly (age > 65) 1; **D**rugs (antiplatelets, NSAIDs) **or** alcohol excess, 1 each.
5. **Interpretation of HAS-BLED.** A score of **≥ 3 indicates high bleeding risk**. **A high HAS-BLED score is NOT a reason to withhold anticoagulation.** Its purpose is to (i) identify and **correct modifiable risk factors** — control blood pressure, stop NSAIDs and unnecessary antiplatelets, limit alcohol, review renal function, avoid labile INR — and (ii) **schedule earlier and more frequent review**.
6. **Geriatric considerations. Falls risk is a very poor reason to withhold anticoagulation** — a patient would need to fall roughly **295 times a year** for the bleeding risk to outweigh the stroke benefit. Assess **frailty, cognition, adherence, swallowing, renal function and drug interactions** instead. Use the integrated **ABC pathway** (**A**void stroke with anticoagulation, **B**etter symptom control, **C**omorbidity and risk factor management), which improves outcomes in anticoagulated patients.

Latest Update – 1 Point (2023–Present)

7. The **2024 ESC Guidelines for the Management of Atrial Fibrillation** replaced CHA_2DS_2-VASc with the **CHA_2DS_2-VA score** — **sex category has been removed** (maximum 7) because female sex is an age-dependent risk modifier rather than an independent risk factor. Anticoagulation is **recommended at a score ≥ 2** and **should be considered at a score of 1**. The ABC pathway has been reframed as the **AF-CARE pathway** (**C**omorbidity and risk factor management, **A**void stroke and thromboembolism, **R**educe symptoms by rate and rhythm control, **E**valuation and dynamic reassessment).

Current Practice Update

Textbook	CHA_2DS_2-VASc including sex category; sex-specific thresholds (men ≥2, women ≥3); ABC pathway.
Current recommendation	2024 ESC — CHA_2DS_2-VA (sex removed, maximum 7), single threshold of ≥2 for both sexes; AF-CARE pathway.
Exam approach	Draw the full CHA_2DS_2-VASc table (still examinable), then write one line: "2024 ESC has replaced this with CHA_2DS_2-VA by removing sex category."

Question 27. Tachyarrhythmias

7-Mark Answer | 6 textbook points + 1 latest update

Textbook Core – 6 Points

1. **Definition and classification.** A tachyarrhythmia is a rhythm with a **ventricular rate above 100 beats per minute**. Classify first by **QRS width** — this is the practical bedside division:

Narrow complex (QRS < 120 ms) — supraventricular	Broad complex (QRS ≥ 120 ms)
Sinus tachycardia; **atrial fibrillation**; atrial flutter; atrial tachycardia; **multifocal atrial tachycardia** (classically in COPD); AV nodal re-entrant tachycardia (AVNRT); AV re-entrant tachycardia (AVRT)	**Ventricular tachycardia (monomorphic or polymorphic)**; **torsades de pointes**; **ventricular fibrillation**; SVT with aberrant conduction or pre-existing bundle branch block; pre-excited AF; paced rhythm

Any broad-complex tachycardia in an older patient with structural heart disease must be treated as ventricular tachycardia until proved otherwise.

2. **Epidemiology and substrate in old age. Abnormal heart rhythm increases with age even in those with normal hearts — the prevalence of atrial and ventricular ectopy reaches up to 100% in older people**, and **ventricular arrhythmias can be found in 70–80% of people over 60**. Age-related **fibrosis of the conduction system and atrial myocardium, chamber dilatation, ischaemic scar and hypertrophy** provide the substrate. For a ventricular tachyarrhythmia to develop, both a **substrate** — structural (infarcted or hypertrophic myocardium creating myocytes of differing refractory periods and a re-entrant circuit) or electrical (early or delayed after-depolarisations) — and a **trigger** are required; the trigger is typically transient, such as **electrolyte imbalance, acute ischaemia or an antiarrhythmic drug** (as in torsades de pointes). **Channelopathies such as congenital long QT and Brugada syndrome are uncommon in older age groups.**
3. **Clinical features. Palpitations, breathlessness, chest pain, dizziness, presyncope, syncope** or, in sustained VT, **cardiac arrest**. In older patients presentation is frequently atypical — **falls, confusion, fatigue, decompensated heart failure or an incidental finding**. Assess **haemodynamic stability** immediately, looking for the adverse features: **shock, syncope, myocardial ischaemia and heart failure**. Examine for **cannon 'a' waves and variable intensity of the first heart sound, which indicate AV dissociation and therefore VT**.

4. **Diagnosis.** Obtain a **12-lead ECG during the tachycardia wherever possible**, plus a rhythm strip; a **previous ECG in sinus rhythm** is invaluable for comparison. ECG features favouring **VT** over SVT with aberrancy: **AV dissociation, capture and fusion beats, QRS > 140 ms, extreme (northwest) axis, concordance across the precordial leads, and a history of previous myocardial infarction** — the last being the single most useful clinical discriminator. For intermittent arrhythmia use **Holter monitoring, an event recorder, an implantable loop recorder or wearable ECG**. Always check **potassium, magnesium, calcium, renal and thyroid function, troponin and digoxin level**, review the drug chart for **QT-prolonging agents**, and obtain an **echocardiogram**. **Interpretation of asymptomatic rhythm disturbance is difficult in the old, since minor asymptomatic dysrhythmias are common and may be entirely incidental — symptom–rhythm correlation is essential.**

5. **Acute management.** The decision is driven by **haemodynamic stability**.

   UNSTABLE (shock, syncope, ischaemia, heart failure) → **immediate synchronised DC cardioversion** under sedation (unsynchronised defibrillation for VF or pulseless VT)
   STABLE narrow-complex → **vagal manoeuvres → adenosine 6 mg then 12 mg rapid IV bolus** (contraindicated in asthma); if AF or flutter, **rate control with a beta-blocker or diltiazem**
   STABLE broad-complex → treat as VT: **amiodarone 300 mg IV over 20–60 min then 900 mg/24 h**; correct electrolytes
   Torsades de pointes → **IV magnesium sulphate 2 g**, stop the offending drug, correct K^+, overdrive pacing or isoprenaline

   Never give verapamil or diltiazem to a broad-complex tachycardia — if it is VT, the result may be cardiovascular collapse. Always **correct potassium and magnesium and treat the underlying trigger** — ischaemia, sepsis, hypoxia, thyrotoxicosis, anaemia or drug toxicity.

6. **Long-term management and prognosis. Treat the underlying disease** — revascularisation for ischaemia, optimal heart failure therapy, correction of electrolytes, withdrawal of proarrhythmic and QT-prolonging drugs. **Beta-blockers are the antiarrhythmic backbone**; **amiodarone** is the most usable agent in structural heart disease but carries thyroid, hepatic, pulmonary, corneal and neurological toxicity in long-term use; **flecainide and propafenone are restricted to structurally normal hearts**. **Catheter ablation** is curative for AVNRT, AVRT and atrial flutter and is effective for scar-related VT. **ICD** for secondary prevention after sustained VT or aborted sudden cardiac death, and for primary prevention with a reduced ejection fraction — **pooled trial data show the survival benefit of the primary-prevention ICD persists in older patients, but the decision must weigh life expectancy, frailty and patient wishes, and ICD deactivation must be discussed as part of end-of-life planning**. On prognosis: **ventricular ectopy and non-sustained VT in the absence of heart disease are not associated with increased coronary disease or mortality and do not require antiarrhythmic drugs**; there is **no association between the extent or complexity of ventricular ectopy and coronary disease in the absence of left ventricular dysfunction, and prognosis is good**. By contrast, **polymorphic VT is a risk marker even without heart disease**, and after myocardial infarction **frequent ectopy (> 10/hour), runs of ectopy or non-sustained VT with a reduced ejection fraction are independently associated with new coronary events and mortality**. Ventricular arrhythmias in the **first 24–48 hours after acute myocardial infarction do not indicate continuing risk**.

Latest Update – 1 Point (2023–Present)

7. **Wearable and consumer devices have become a routine part of arrhythmia diagnosis.** Following large screening studies (**Apple Heart, Fitbit Heart, mSToPS, LOOP**), **smartwatch and single-lead handheld ECG devices are now accepted for symptom–rhythm correlation and for confirming AF, and the 2024 ESC AF guideline explicitly addresses device-detected atrial high-rate episodes**. The key examinable nuance comes from **NOAH-AFNET 6 (NEJM 2023)** and **ARTESiA (NEJM 2023)**, which studied **subclinical device-detected atrial fibrillation**: anticoagulation produced **only a small reduction in stroke that was largely offset by an increase in major bleeding**. The conclusion is that **device-detected subclinical AF is NOT equivalent to clinical AF, and should not automatically trigger anticoagulation** — a decision that requires individual assessment of episode duration, stroke risk and bleeding risk, which is particularly important given how frequently incidental arrhythmia is now detected in older people.

F. Syncope, Falls and Autonomic Disorders

Question 28. Head-up tilt test — indication, procedure and interpretation

7-Mark Answer | 6 textbook points + 1 latest update

Textbook Core – 6 Points

1. **Principle.** Head-up tilt testing reproduces the haemodynamic stress of standing under controlled, monitored conditions. Passive upright tilt causes **venous pooling of about 500–800 mL of blood in the lower limbs and splanchnic bed**, reducing venous return and cardiac output. In a susceptible individual this triggers the **Bezold–Jarisch reflex** — vigorous contraction of an underfilled ventricle activates mechanoreceptors, producing **paradoxical sympathetic withdrawal and vagal activation with vasodilatation and/or bradycardia**, and hence syncope. **Increasing use has been made of tilt testing to obtain evidence supporting a suspected diagnosis of vasovagal syncope and to investigate the underlying mechanisms.**
2. **Indications.** (i) **Unexplained recurrent syncope** after cardiac causes have been excluded, particularly where **vasovagal syncope is suspected but the history is not diagnostic**. (ii) To distinguish **reflex syncope from orthostatic hypotension** and to demonstrate **delayed orthostatic hypotension** that standing measurements miss. (iii) **Unexplained falls or drop attacks in the elderly, where there is no recall of loss of consciousness**. (iv) To differentiate syncope from **epilepsy or psychogenic pseudosyncope** — the **semiology of tilt-induced psychogenic pseudosyncope** is characteristic, and testing may be combined with video-EEG. (v) To demonstrate **susceptibility for the purpose of patient education and counter-manoeuvre training**. (vi) In suspected **postural orthostatic tachycardia syndrome** or autonomic failure. **Contraindications:** severe aortic stenosis, critical coronary or cerebrovascular stenosis, hypertrophic obstructive cardiomyopathy, pregnancy and inability to stand safely.
3. **Procedure.** The patient is **fasted for 2–4 hours**, and vasoactive drugs are withheld where safe. They lie on an **electrically driven tilt table with a footboard**, secured with straps, with **continuous ECG and beat-to-beat non-invasive blood pressure monitoring** and resuscitation facilities available. There is a **supine stabilisation phase of 5–20 minutes**, then **passive tilt to 60–70° for 20–45 minutes**. If nothing occurs, a **provoked (drug) phase** follows: **the proportion of positive tilt tests is increased following the administration of glyceryl trinitrate** (400 µg sublingual, tilt maintained for a further 15–20 minutes), or isoprenaline may be used. **Carotid sinus massage** is often performed as part of the same assessment, supine and erect, since **carotid sinus syndrome is a common cause of syncope in the elderly and is not confined to those with a history of fainting on neck movement**; it carries a very low complication risk despite underlying atheroma, but **must not be performed if there is a carotid bruit unless ultrasound has excluded severe stenosis**. The table is returned to horizontal as soon as syncope or presyncope occurs. **The test has been shown to be safe in the elderly, including with glyceryl trinitrate provocation, over 10 years of experience.**
4. **Interpretation — a positive test.** The test is positive when **symptoms are reproduced together with hypotension and/or bradycardia**. **Provocation of the patient's habitual attack is diagnostically very suggestive.** Classify the response (VASIS classification):

Type	Response	Implication
Type 1 — Mixed	Heart rate falls but not below 40/min for > 10 s, with hypotension	Commonest pattern
Type 2A — Cardioinhibitory without asystole	Heart rate < 40/min for > 10 s, no asystole > 3 s; BP falls after the heart rate	—
Type 2B — Cardioinhibitory with asystole	**Asystole > 3 seconds**	**May benefit from pacing**
Type 3 — Vasodepressor	BP falls with heart rate fall < 10% from peak	Pacing will not help
Orthostatic hypotension	Progressive BP fall ≥ 20/10 mmHg without bradycardia	Treat as OH
POTS	Heart rate rise > 30/min (or to > 120/min) without hypotension	—
Psychogenic pseudosyncope	Apparent unconsciousness with **normal blood pressure and heart rate**	Characteristic; confirms non-syncopal attack

5. **Limitations. The major difficulty in interpreting the results is the occurrence of positive tests in a significant minority of control subjects, which limits the positive predictive value.** Sensitivity is around 50–70% and specificity around 80–90% for passive tilt, with sensitivity improving but specificity falling after nitrate provocation. **Reproducibility is imperfect** — a negative test does not exclude reflex syncope. Crucially, **the type of response on tilt does not reliably predict the mechanism of the patient's spontaneous attacks**, which is why an implantable loop recorder is often preferred where the diagnosis will change management. The test is also **time-consuming and labour-intensive**, and in frail older patients prolonged standing may be poorly tolerated. It is also worth noting that a **postural fall in blood pressure of more than 20 mmHg is present in about 30% of a large elderly population**, so an abnormal result must always be interpreted alongside the clinical history.

6. **Clinical use of the result.** A positive test **confirms susceptibility, allows patient education, and justifies conservative treatment** — recognition of prodromal symptoms, avoidance of triggers (heat, prolonged standing, dehydration, alcohol, large meals), **physical counter-pressure manoeuvres (leg crossing, calf tensing, abdominal compression)**, adequate salt and fluid intake, and compression stockings. Drug options where these fail include **fludrocortisone, midodrine and, in some patients, beta-blockers**. **Pacing is reserved for documented cardioinhibitory responses with prolonged asystole in older patients with recurrent injurious syncope** — but note that **pacing does not improve hypotension in patients with severe orthostatic hypotension**, so it is not a treatment for the vasodepressor form. In **carotid sinus syndrome with a cardioinhibitory response, pacing reduces falls (SAFE PACE)**. Finally, **the risks of serious injury from falls in an elderly population are very high**, so the patient must be counselled to take precautions to avoid attacks.

Latest Update – 1 Point (2023–Present)

7. **The diagnostic role of tilt testing has narrowed as prolonged rhythm monitoring has become easier and cheaper.** Current practice, consistent with the **2018 ESC syncope guideline and the Canadian Cardiovascular Society clinical practice update**, is that in **recurrent unexplained syncope where a cardiac mechanism is suspected, an implantable loop recorder gives a far higher diagnostic yield than tilt testing and should be used early rather than as a last resort**, because — unlike tilt — it documents the mechanism of the **spontaneous** attack. Tilt testing has correspondingly been repositioned as a test principally used to **demonstrate susceptibility to reflex syncope for patient education, to unmask delayed orthostatic hypotension, and to diagnose psychogenic pseudosyncope** — the last being an indication where it remains uniquely valuable, especially when combined with **video and EEG recording**. Alongside this, **smartphone and smartwatch single-lead ECG recordings** are increasingly used to capture symptom–rhythm correlation before more invasive testing is arranged.

Question 29. Syncope — aetiology, pathogenesis, differential diagnosis and management

15-Mark Essay | 10 textbook points + 2 latest updates

Textbook Core – 10 Points

1. **Definition.** Syncope is a **transient loss of consciousness due to global cerebral hypoperfusion**, characterised by **rapid onset, short duration and spontaneous complete recovery**. It is one cause of the wider syndrome of **transient loss of consciousness (T-LOC)**. Cerebral perfusion fails when systemic blood pressure falls, which occurs through a fall in **cardiac output**, in **peripheral vascular resistance**, or both. **In older people syncope frequently presents as a history of falls or drop attacks with no recall of loss of consciousness**, so the diagnosis is systematically under-recognised.
2. **Classification and aetiology.**

Category	Causes
Reflex (neurally mediated)	**Vasovagal** (fear, trauma, pain, emotion); **carotid sinus syndrome**; **situational — micturition, cough, defaecation, swallow, post-prandial**; oculocardiac and neck pressure; **glossopharyngeal neuralgia and IXth/Xth cranial nerve disease**; neck tumours
Orthostatic hypotension	**Autonomic neuropathy** (diabetes, Shy–Drager/multiple system atrophy, Guillain–Barré); **spinal cord disease, especially traumatic cervical cord lesions**; **drugs**; **old age itself**; low venous pressure (haemorrhage, dehydration, prolonged standing)
Cardiac	**Dysrhythmias** — bradyarrhythmia (**sick sinus syndrome, complete heart block with Stokes–Adams attacks**), asystole, **paroxysmal tachyarrhythmia such as ventricular tachycardia**; **ventricular outflow obstruction** (aortic stenosis, HOCM), especially on exertion; also PE, tamponade, dissection, myocardial infarction
Respiratory	**Coughing, trumpeting, weight lifting, hyperventilation** — all raise intrathoracic pressure and impair venous return; anoxia
Cerebrovascular	**Vertebrobasilar transient ischaemic attacks** — but **syncope as a manifestation of cerebrovascular disease is unlikely in the absence of additional symptoms suggestive of posterior circulation TIAs**

3. **Pathogenesis of the individual mechanisms.** In **vasovagal syncope**, venous pooling in the upright posture reduces ventricular filling; vigorous contraction of the underfilled ventricle activates mechanoreceptors and triggers **paradoxical sympathetic withdrawal with vagal activation (Bezold–Jarisch reflex)**, producing vasodilatation and/or bradycardia. In **micturition syncope** the trigger is the **sudden loss of the pressor stimulus of a distended bladder**, typically when the patient rises at night with a vasodilated skin and having just assumed the upright posture — **contrary to common belief, straining is not an important factor**. In **cough syncope** raised intrathoracic pressure impedes venous return. In **reflex/orthostatic syncope** the baroreceptor reflexes that normally stabilise blood pressure on standing fail; **these responses become more sluggish with age, causing an increased tendency to postural syncope**, and this is exacerbated by **dehydration, drugs and central nervous system disease such as Parkinson's disease**. In **cardiac syncope** the fall in output is abrupt, which is why there is usually no warning.

4. **Drugs and the older patient.** A drug cause must be sought in every case. Implicated agents include **ganglion blockers, diuretics, phenothiazines, antihistamines, antidepressants, benzodiazepines, barbiturates and antiparkinsonian drugs**; among cardiovascular drugs, **nitrates, beta-blockers and nifedipine** are particularly important; and **donepezil may increase the risk of syncope**. **Orthostatic hypotension and syncope are common in the elderly following haemodialysis.** A postural fall of more than **20 mmHg is found in about 30%** and of more than **40 mmHg in 10%** of a large elderly population.
5. **Clinical features — and why the classical picture misleads.** The image of a patient falling, lying still for some seconds and then coming round **is actually unusual**. In a landmark study inducing vasovagal syncope in medical students, **only a minority lay still; 90% had asynchronous myoclonic jerks of the limbs**, in a few vigorous enough to be mistaken for a generalised clonic seizure. Other motor activity included **limb posturing, head turning, complex movements, eye deviation and eyelid flicker**, which may be misread as focal seizures, and **vocalisation was frequent**. The same high frequency of myoclonic movement has been documented in **cardiac syncope** (observed when arrhythmia is deliberately induced to test an implanted defibrillator) and in **tilt-induced syncope**, with **simultaneous EEG showing no epileptiform activity**. Anecdotally, a patient **propped up rather than allowed to fall flat is more likely to jerk**, presumably because the fall in blood pressure is more prolonged and severe; rarely the hypoxia may itself provoke a genuine epileptic seizure.
6. **Differential diagnosis of transient loss of consciousness. Epileptic seizure** — aura, prolonged tonic-clonic movement beginning at onset, lateral tongue biting, prolonged post-ictal confusion, cyanosis, incontinence (which occurs in syncope too and is not discriminating). **Psychogenic pseudosyncope and non-epileptic attacks** — prolonged apparent unconsciousness, eyes closed and resisting opening, high attack frequency, normal blood pressure and pulse during the event. **Falls without loss of consciousness** — mechanical, drop attacks, vestibular. **Metabolic** — hypoglycaemia, hypoxia, hyperventilation with hypocapnia (cerebral vasoconstriction), intoxication. **Cerebrovascular** — vertebrobasilar TIA, subclavian steal, subarachnoid haemorrhage. **Cataplexy. The single most valuable investigation is a careful eyewitness account, and short-term video-EEG is useful in the diagnosis of attack disorders where the nature of the event remains unclear.**
7. **Initial evaluation.** Take a **meticulous history of the circumstances, posture, prodrome, duration, colour, recovery and any injury**, plus a **witness account**. **Cough syncope should be considered in patients with COPD, emphysema and chronic cough — an adequate witness account is essential since some patients fail to recall the bout of coughing that triggers the attack.** Review all drugs. Examine for **cardiac murmurs (especially aortic stenosis), pulse, blood pressure supine and at 1 and 3 minutes standing, neurological signs and carotid bruits**. **Baseline tests: 12-lead ECG**, full blood count, urea, electrolytes, creatinine, glucose and calcium. **The presence of underlying cardiac disease increases the probability of a cardiac cause**, and **timely and effective treatment of cardiac syncope may be life-saving** — so risk-stratify at this first assessment.
8. **Further investigation. Routine 12-lead ECG will occasionally demonstrate an arrhythmia or predisposing abnormality. 24-hour ECG can be diagnostic if an attack is recorded, but the yield is very small unless attacks are very frequent, and interpretation of asymptomatic rhythm disturbance is difficult — in an older population minor asymptomatic dysrhythmias are common and may be incidental.** Therefore, for infrequent attacks use **prolonged external monitoring or an implantable loop recorder**, which has a good diagnostic yield and is safe in patients with and without structural heart disease. Add **echocardiography** if structural disease is suspected, **exercise testing** for exertional syncope, and **electrophysiological study** in selected cases. Use **head-up tilt testing** to support a suspected diagnosis of vasovagal syncope — it is safe in the elderly, and the yield increases with glyceryl trinitrate, but **positive tests occur in a significant minority of controls, which limits the positive predictive value**; provocation of the habitual attack is diagnostically very suggestive. Perform **carotid sinus massage**, since **carotid sinus syndrome is a common cause of syncope in the elderly and is not confined to those who faint on neck movement** — it is low-risk, but **not if there is a carotid bruit unless ultrasound has excluded severe stenosis**. **When cardiac syncope is suspected, specialist cardiac referral is usually indicated.**
9. **Management by cause. Micturition syncope can usually be avoided by micturating sitting down. Cough syncope** — direct treatment at the underlying chest condition. **Cardiac syncope requires appropriate specialist treatment with antiarrhythmic medication, pacing, or insertion of an implantable defibrillator as appropriate — timely and effective treatment may be life-saving. Aortic stenosis with syncope** is an indication for urgent valve intervention. **Carotid sinus syndrome with a cardioinhibitory response** — dual-chamber pacing reduces falls. **Vasovagal syncope** — education about prodromal recognition, trigger avoidance and **physical counter-pressure manoeuvres**.

10. **Management of reflex syncope with postural hypotension in the older patient.** This is a common and difficult management problem, and the approach is stepwise. **Attention should first be directed to factors causing or exacerbating the problem. Review the medication** — but recognise it is not always possible to discontinue relevant drugs; in Parkinson's disease the aggravating effect of dopaminergic drugs must be balanced against their therapeutic benefit. **Exclude treatable conditions such as Addison's disease.** Then: **avoid factors causing vasodilatation — high temperature, marked exertion and alcohol**; take **small frequent meals rather than large ones**; **avoid dehydration**; **change posture slowly and reverse the change temporarily if symptoms supervene**; use **leg crossing, calf tensing and abdominal compression** to ameliorate symptoms transiently. **Elastic compression stockings make a significant contribution** though patients may need help applying them and tolerance is poor in summer. **Sleeping with a slight head-up tilt** may help by expanding plasma volume, but more than about 10° is not tolerated. Patients should be encouraged to **remain as active as possible, since chronic exposure to low blood pressure seems to improve cerebral autoregulation**. If these measures fail, add drugs, recognising that **most are used outside their licence and benefits may be limited**: **fludrocortisone 100–200 µg at night is usually first**; **ephedrine 15 mg three times daily, increasing if necessary to 30 mg three times daily** (higher doses cause tremor, tachycardia and agitation); **midodrine, desmopressin and erythropoietin** have also been used. **Supine hypertension is a risk of drug treatment, and pacing does not help.** Finally, **because the risks of serious injury and complications from falls are very high in an elderly population, it is important that the patient recognises these risks and understands the need to take precautions to avoid syncopal attacks wherever possible**.

Latest Updates – 2 Points (2023–Present)

11. **Structured emergency department risk stratification and the syncope unit model.** The **Canadian Syncope Risk Score** — combining predisposition to vasovagal syncope, heart disease, systolic BP, troponin, QRS/QT abnormality, ECG diagnosis and ED diagnostic impression — has been externally validated across multiple countries and is now used to identify patients safely dischargeable from the emergency department, avoiding a hospital admission that in an older person carries its own risk of delirium, deconditioning and hospital-acquired infection. This is supported by randomised evidence that an **ED observation syncope protocol is equivalent to routine inpatient admission**, and by the **EHRA/HRS position statement on the syncope unit**, which recommends a **dedicated multidisciplinary service with standardised protocols** — an approach shown to increase diagnostic yield and reduce admissions and cost.
12. **Implantable loop recorders early rather than last, and reappraisal of pacing in reflex syncope.** Current practice, consistent with the **2018 ESC guideline and the 2020 Canadian Cardiovascular Society clinical practice update**, is that in recurrent unexplained syncope with suspected arrhythmia the **implantable loop recorder should be used early rather than after a long sequence of low-yield tests**, because it documents the mechanism of the **spontaneous** attack, which tilt testing cannot. For refractory reflex syncope, **closed-loop stimulation pacing (BIOSync CLS)** markedly reduced recurrence in patients over 40 with **tilt-documented asystolic vasovagal syncope**, and this has been carried into the **ESC pacing guideline** — but pacing remains ineffective for the **vasodepressor** form and for orthostatic hypotension, which is the crucial distinction to state.

Question 30. Drop attacks

6-Mark Answer | 5 textbook points + 1 latest update

Textbook Core – 5 Points

1. **Definition.** A drop attack is a **sudden, unexpected fall to the ground WITHOUT loss of consciousness and without warning (no prodrome), with preserved awareness throughout, occurring in the absence of dizziness or vertigo**. The patient characteristically says "my legs just gave way", and there is **no post-event confusion**. Recovery is usually immediate, although some patients are unable to rise for a period despite full alertness. It is **commonest in older women**, and is a **descriptive term rather than a diagnosis** — a syndrome requiring a cause to be sought.
2. **The central diagnostic problem. The most important point is that many "drop attacks" are in fact unrecognised syncope** — in the elderly, syncope frequently presents as a history of **falls or drop attacks with no recall of loss of consciousness**, because retrograde amnesia for the blackout is common. **Carotid sinus syndrome in particular is a modifiable risk factor for non-accidental falls in older adults (SAFE PACE)**, and is not confined to patients who describe fainting with neck movement. Therefore **every unexplained drop attack in an older person must be investigated as possible syncope** until proved otherwise.

3. **Causes.**

Group	Causes
Cardiovascular (must be excluded first)	**Unrecognised syncope**; **carotid sinus syndrome** (cardioinhibitory or vasodepressor); **orthostatic hypotension**; **bradyarrhythmia — sick sinus syndrome, complete heart block (Stokes–Adams)**; paroxysmal tachyarrhythmia; **aortic stenosis**
Neurological	**Vertebrobasilar ischaemia** (the classical but now over-diagnosed explanation — implausible unless there are other posterior circulation symptoms); **atonic (akinetic) seizures**; **third ventricle colloid cyst**; **normal pressure hydrocephalus**; **cervical myelopathy and cervical spondylosis**; Parkinson's disease and other parkinsonian syndromes with freezing; cataplexy; **Tumarkin otolithic crisis in Ménière's disease**
Musculoskeletal and other	**Sarcopenia and proximal muscle weakness**, knee osteoarthritis with buckling, vitamin D deficiency and osteomalacia, peripheral neuropathy; **drugs** — sedatives, antihypertensives, antidepressants, antiparkinsonian agents

4. **Evaluation.** Obtain a **meticulous history with an eyewitness account**, focusing on **posture and activity at onset, any prodrome, whether awareness was truly preserved, duration, ability to rise, injury pattern and drug history**. **Facial and knee injuries suggest a genuinely unprotected fall**, which supports syncope or an atonic mechanism rather than a mechanical trip. Examine the **cardiovascular system (murmurs, pulse, lying and standing blood pressure at 1 and 3 minutes)**, the **neurological system (gait, tone, power, cerebellar signs, cervical spine, cognition)** and perform a **gait and balance assessment**. Investigations: **12-lead ECG**, full blood count, urea, electrolytes, creatinine, glucose, calcium, vitamin D and B12, thyroid function; **echocardiography** if a murmur is present; **carotid sinus massage and head-up tilt testing**; **prolonged ambulatory monitoring or an implantable loop recorder** for infrequent attacks; and **MRI brain and cervical spine** if there are neurological features. **Assess bone health and fracture risk in all.**

5. **Management. Treat the identified cause — pacing for a documented cardioinhibitory carotid sinus syndrome (which reduces falls) or for high-grade heart block**, valve intervention for aortic stenosis, antiepileptic therapy for atonic seizures, shunting for normal pressure hydrocephalus, and surgery for cervical myelopathy. Where no cause is found — as is common — manage as an **unexplained fall** using a **multifactorial falls intervention**: **medication review with withdrawal of sedatives, antihypertensives and anticholinergics; strength and balance training; vitamin D and calcium; treatment of visual impairment; footwear and home hazard assessment; and osteoporosis treatment to reduce fracture risk**. Teach the patient and carer **how to get up from the floor and how to summon help**, and consider a **personal alarm or pendant**, since a long lie causes rhabdomyolysis, pressure injury, hypothermia and loss of confidence. Address the resulting **fear of falling**, which itself causes activity restriction and further deconditioning.

Latest Update – 1 Point (2023–Present)

6. The **2022 World Guidelines for Falls Prevention and Management in Older Adults** (a joint initiative of the geriatric societies, published in *Age and Ageing* and carried into current practice) formally abolish the artificial separation between "falls" and "syncope" clinics by recommending a **single stratified pathway**: every older person is assessed as **low, intermediate or high risk**, and anyone with an **unexplained fall, a fall with injury, or recurrent falls is placed in the high-risk group requiring a full multifactorial assessment that explicitly includes cardiovascular evaluation for syncope, orthostatic hypotension and carotid sinus syndrome**. The guideline also stresses **de-prescribing of fall-risk-increasing drugs (FRIDs)**, **exercise incorporating balance and strength training as the single most effective intervention**, and **concurrent assessment of bone health and fracture risk** — which together is the correct modern framework for the patient presenting with drop attacks.

G. Vascular Disease, Thrombosis and Prevention

Question 31. Ankle brachial index and toe pressures — indications, assessment and interpretation

7-Mark Answer | 6 textbook points + 1 latest update

Textbook Core – 6 Points

1. **Definition and principle.** The ankle brachial index (ABI) is the **ratio of the highest systolic pressure at the ankle to the highest systolic pressure in either arm**, measured with a sphygmomanometer and a hand-held Doppler probe. In health the ankle pressure equals or slightly exceeds the brachial pressure because of pulse-wave amplification; **a proximal arterial stenosis causes a pressure drop across the lesion, so the ABI falls**. It is a **simple, non-invasive, inexpensive and reproducible bedside test** and is the **first-line investigation for peripheral arterial disease (PAD)**.

2. **Indications. Diagnostic:** symptoms of PAD — **intermittent claudication, rest pain, non-healing foot ulcer or gangrene**; absent or diminished pulses or a femoral bruit. **Before compression therapy: mandatory before applying compression bandaging or stockings for a leg ulcer, because compression in the presence of significant arterial disease causes ischaemic necrosis**. **Screening in asymptomatic high-risk groups:** age ≥ 65 (or ≥ 50 with diabetes or smoking), diabetes with a foot at risk, chronic kidney disease, known atherosclerosis elsewhere (coronary, carotid, aortic aneurysm). **Prognostic:** as a marker of generalised atherosclerosis and cardiovascular risk. **Monitoring:** after revascularisation and in follow-up of known PAD.

3. **How to perform it. Rest the patient supine for 5–10 minutes** in a warm room. Use an appropriately sized cuff and an **8–10 MHz continuous-wave Doppler probe**. Measure the **brachial systolic pressure in both arms** and take the **higher**. At each ankle, measure the pressure over the **dorsalis pedis** and the **posterior tibial** arteries and take the **higher of the two**. **ABI for each leg = highest ankle pressure in that leg ÷ highest brachial pressure of the two arms.** Calculate each leg separately and report the lower of the two values as the patient's ABI. **Do not use a stethoscope** — Korotkoff sounds are unreliable at the ankle. If claudication is suspected but the resting ABI is normal, perform an **exercise ABI (treadmill or heel raises)**: a **post-exercise fall of more than 20%, or an absolute ankle pressure drop of more than 30 mmHg, is diagnostic**.

4. **Interpretation.**

ABI value	Interpretation
> 1.40	**Non-compressible, calcified vessels** — the result is uninterpretable; proceed to toe pressures
1.00 – 1.40	**Normal**
0.91 – 0.99	**Borderline** — perform an exercise ABI
≤ 0.90	**Diagnostic of peripheral arterial disease** (sensitivity ~75–90%, specificity ~95% against angiography)
0.40 – 0.90	Mild to moderate PAD — usually claudication
< 0.40	**Severe PAD / chronic limb-threatening ischaemia** — rest pain, ulceration, gangrene

   An ABI of **0.90 or less roughly doubles 10-year mortality and major coronary event rates**, so an abnormal ABI is an indication for full cardiovascular secondary prevention regardless of leg symptoms.

5. **Limitations — the crucial geriatric caveat. The ABI is falsely NORMAL or falsely HIGH when the tibial arteries are rigid and non-compressible due to medial (Mönckeberg) calcification** — classically in **long-standing diabetes, chronic kidney disease and dialysis, and advanced age**. This is precisely the population most likely to have severe disease, so a normal ABI in a diabetic patient with an ischaemic-looking foot must never be accepted at face value. Other limitations: it gives no anatomical localisation, it can miss isolated **iliac (proximal) stenosis** if collaterals are good, it is inaccurate with leg ulceration, oedema, pain or amputation, and it is operator-dependent.

6. **Toe pressures and the toe brachial index (TBI).** The **digital arteries are largely spared from medial calcification**, so toe pressures remain reliable when the ABI does not. Measure with a **small (2–2.5 cm) cuff on the great toe and photoplethysmography or laser Doppler**, after warming the foot. Normally the toe pressure is **about 30 mmHg lower than the ankle pressure**. **TBI = toe systolic pressure ÷ highest brachial pressure; a TBI below 0.70 is diagnostic of peripheral arterial disease.** Absolute toe pressures guide healing: **> 55 mmHg — wound healing likely; 30–55 mmHg — impaired healing; < 30 mmHg — chronic limb-threatening ischaemia with healing unlikely without revascularisation** (a threshold of < 50 mmHg is used in diabetes). Toe pressures are therefore **indicated whenever the ABI exceeds 1.40, in all patients with diabetes or renal failure, and to assess healing potential of a foot ulcer or the level of a planned amputation**. Complementary tests include **Doppler waveform analysis (triphasic → biphasic → monophasic → damped), segmental pressures with pulse volume recording, transcutaneous oxygen pressure ($TcPO_2$), duplex ultrasound, CT or MR angiography**.

Latest Update – 1 Point (2023–Present)

7. The **2024 ACC/AHA Guideline for the Management of Lower Extremity Peripheral Artery Disease** restructures PAD around **four clinical subsets — asymptomatic, chronic symptomatic, chronic limb-threatening ischaemia and acute limb ischaemia**. Key diagnostic recommendations: the **resting ABI remains the first-line test**, but **toe brachial index or other physiological testing is explicitly recommended when the ABI is greater than 1.40 or is normal despite a high clinical suspicion**, and a **wound, ischaemia and foot infection (WIfI) staging** should be applied to every threatened limb. For treatment, it emphasises **structured supervised exercise therapy as first-line for claudication**, **high-intensity statin plus antiplatelet therapy for all patients**, and — importantly — **low-dose rivaroxaban 2.5 mg twice daily plus aspirin (the COMPASS/VOYAGER-PAD regimen) to reduce major adverse limb and cardiovascular events**, weighed against bleeding risk in the older patient.

Question 32. Direct oral anticoagulants — choice, dosing and reversal

7-Mark Answer | 6 textbook points + 1 latest update

Textbook Core – 6 Points

1. **Drugs and mechanism. Dabigatran** is a **direct thrombin (factor IIa) inhibitor**. **Rivaroxaban, apixaban and edoxaban** are **direct factor Xa inhibitors**. Advantages over warfarin: **predictable pharmacokinetics, no routine monitoring, rapid onset and offset, fewer food and drug interactions**, and **lower intracranial haemorrhage** risk.
2. **Indications and contraindications.** Indicated for **stroke prevention in non-valvular atrial fibrillation**, **treatment and secondary prevention of DVT and pulmonary embolism**, and **VTE prophylaxis after hip or knee arthroplasty**. **Contraindicated in mechanical prosthetic heart valves and moderate-to-severe (rheumatic) mitral stenosis** — these require **warfarin**, an important point in Indian practice. Also avoid in severe hepatic impairment (Child–Pugh C) and active major bleeding.
3. **Standard doses in atrial fibrillation. Apixaban 5 mg twice daily**; **Rivaroxaban 20 mg once daily with food**; **Edoxaban 60 mg once daily**; **Dabigatran 150 mg twice daily** (110 mg twice daily if aged ≥ 80 or on verapamil).
4. **Dose reduction criteria (high-yield).**

Drug	Reduced dose	Criteria
Apixaban	2.5 mg BD	Any **2 of 3**: age ≥ 80 y, weight ≤ 60 kg, creatinine ≥ 1.5 mg/dL
Rivaroxaban	15 mg OD	CrCl 15–49 mL/min
Edoxaban	30 mg OD	CrCl 15–50 mL/min, weight ≤ 60 kg, or strong P-gp inhibitor
Dabigatran	110 mg BD	Age ≥ 80 y, verapamil, high bleeding risk; avoid if CrCl < 30

   Avoid all DOACs when **CrCl < 15 mL/min** (apixaban may be used with caution). **Use actual body weight and the Cockcroft–Gault formula, not eGFR, for dosing.**
5. **Choice in the older adult. Apixaban** has the most favourable geriatric profile — **lowest major and gastrointestinal bleeding** and the safest in chronic kidney disease. **Avoid dabigatran** if there is dyspepsia or gastrointestinal bleeding risk, or in significant renal impairment. Once-daily rivaroxaban or edoxaban may aid **adherence**. Avoid concurrent **NSAIDs and antiplatelets**; beware **P-gp and CYP3A4 interactions** (rifampicin, carbamazepine, phenytoin, azoles, verapamil, amiodarone). Check **renal function at least annually** — more often if CrCl < 60 (a practical rule is **CrCl ÷ 10 = review interval in months**).
6. **Reversal and bleeding management. Stop the drug**, note the time of the last dose, apply local haemostatic measures, restore volume, transfuse as needed and give **tranexamic acid** for mucosal bleeding. Specific antidotes: **Idarucizumab 5 g IV** — a monoclonal antibody fragment reversing **dabigatran**; **Andexanet alfa** — a modified factor Xa decoy reversing **apixaban and rivaroxaban**. Where antidotes are unavailable (common in India), give **4-factor prothrombin complex concentrate 50 units/kg**. **Activated charcoal** if ingestion was within 2–4 hours; **dabigatran is dialysable** (the factor Xa inhibitors are not, being highly protein-bound).

Latest Update – 1 Point (2023–Present)

7. **ANNEXA-I (NEJM 2024)** — in acute **intracranial haemorrhage** on a factor Xa inhibitor, **andexanet alfa** achieved significantly better **haemostatic efficacy** than usual care (largely PCC), but at the cost of **more thrombotic events, including ischaemic stroke**. Current guidance therefore recommends specific reversal agents as **first-line** for life-threatening or intracranial bleeding, with **4-factor PCC as the alternative**, and stresses **early restarting of anticoagulation** once haemostasis is secure.

Question 33. Deep vein thrombosis — prophylaxis and treatment

7-Mark Answer | 6 textbook points + 1 latest update

Textbook Core – 6 Points

1. **Risk factors (Virchow's triad). Stasis** — immobility, hospitalisation, **stroke with hemiplegia**, long travel, heart failure. **Endothelial injury** — surgery, especially **hip fracture and hip or knee arthroplasty**, trauma, central venous catheters. **Hypercoagulability** — **malignancy**, sepsis, dehydration, previous VTE, oestrogens, nephrotic syndrome, thrombophilia. **Advancing age is itself an independent risk factor**, with incidence rising sharply after 70.
2. **Clinical features and pitfalls.** Unilateral **calf or thigh pain, swelling, warmth, erythema, dilated superficial veins** and calf tenderness; Homans' sign is unreliable. **Clinical diagnosis alone is unreliable in the elderly.** Differential diagnosis: **cellulitis, ruptured Baker's cyst, dependent oedema, heart failure, lymphoedema, haematoma, superficial thrombophlebitis** and post-thrombotic syndrome.

3. **Diagnosis.** Apply the **Wells score** to stratify probability. **Low probability plus a negative D-dimer excludes DVT**, but D-dimer specificity falls markedly with age, so use the **age-adjusted cut-off (age × 10 µg/L for patients over 50)** to avoid unnecessary imaging. Confirm with **compression ultrasonography**; if the initial scan is negative but clinical probability is high, repeat at one week or proceed to whole-leg scanning.
4. **Prophylaxis. Early mobilisation and adequate hydration for all. Mechanical — intermittent pneumatic compression**; note that **graduated compression stockings are contraindicated after stroke** (CLOTS-1 showed no benefit and more skin breaks). **Pharmacological — LMWH (enoxaparin 40 mg subcutaneously daily; reduce to 20–30 mg if CrCl < 30 mL/min)** or fondaparinux, for the duration of the acute illness; **extended prophylaxis for 28–35 days after hip fracture surgery or hip arthroplasty**. Always **assess bleeding risk** before prescribing.
5. **Treatment. A DOAC is first-line — apixaban 10 mg twice daily for 7 days then 5 mg twice daily**, or **rivaroxaban 15 mg twice daily for 21 days then 20 mg daily**; **dabigatran and edoxaban require 5 days of parenteral LMWH lead-in**. Use **LMWH** in severe renal impairment and in pregnancy, and **warfarin (INR 2–3)** for mechanical valves or antiphospholipid syndrome. **Early ambulation with compression** relieves symptoms; an **IVC filter** is used only when anticoagulation is absolutely contraindicated, and should be retrievable. **Catheter-directed thrombolysis** is reserved for limb-threatening iliofemoral thrombosis (phlegmasia).
6. **Duration and complications. Provoked by a transient major risk factor → 3 months. Unprovoked, recurrent, or with a persistent risk factor such as active cancer → extended (indefinite) anticoagulation**, using reduced-dose **apixaban 2.5 mg twice daily or rivaroxaban 10 mg daily** after the first 6 months. Complications: **pulmonary embolism, post-thrombotic syndrome (up to 40%), recurrence and anticoagulant-related bleeding**. After unprovoked DVT, perform **age-appropriate cancer screening and a careful history and examination** — extensive occult-malignancy imaging is not indicated.

Latest Update – 1 Point (2023–Present)

7. **API-CAT trial (NEJM 2025)** — in patients with **active cancer** and proximal DVT or PE who had completed at least 6 months of anticoagulation, **extended reduced-dose apixaban 2.5 mg twice daily for 12 months was non-inferior to full-dose 5 mg twice daily** for recurrent VTE and caused **significantly less clinically relevant bleeding (12.1% versus 15.6%)**. This supports **dose de-escalation rather than discontinuation** for extended treatment — particularly valuable in frail older patients with cancer-associated thrombosis.

Question 34. Polypill

6-Mark Answer | 5 textbook points + 1 latest update

Textbook Core – 5 Points

1. **Concept and definition.** The polypill is a **fixed-dose combination tablet containing several cardiovascular drugs**, taken once daily, designed to deliver **comprehensive cardiovascular risk reduction in a single pill**. The concept was proposed by **Wald and Law (BMJ 2003)**, who calculated that a combination of a statin, three half-dose antihypertensives, aspirin and folic acid given to everyone over 55 could reduce ischaemic heart disease events by about 88% and stroke by about 80%. **The rationale is that most cardiovascular risk factors are continuous variables with no threshold, so a fixed low-dose combination benefits a broad population without individual measurement.**
2. **Typical composition.** A **statin** (atorvastatin or simvastatin), **aspirin 75–100 mg**, and **two or three antihypertensives at low dose** — commonly an **ACE inhibitor (ramipril or enalapril), a thiazide-type diuretic and/or a beta-blocker or calcium channel blocker**. Folic acid was in the original proposal but has been abandoned, since homocysteine lowering does not reduce events. **Secondary-prevention polypills** (for patients with established disease) always contain aspirin and a statin; **primary-prevention polypills** may omit aspirin because its net benefit in primary prevention is now considered unfavourable (ASPREE, ASCEND, ARRIVE).
3. **Advantages — particularly in geriatric and Indian practice. Improved adherence**, which is the dominant benefit — **reduced pill burden** is directly relevant to the polypharmacy of old age. **Lower cost**, which matters greatly in low- and middle-income settings. **Simplified prescribing and dispensing**, enabling task-shifting to nurses and community health workers. **Wider treatment coverage**, closing the large treatment gap in populations where risk factors are undiagnosed. **Fewer dispensing and administration errors.** India has been central to this field — the **TIPS (The Indian Polycap Study)** trials, conducted with the **Polycap** formulation, established the tolerability and risk-factor efficacy of the approach.

4. **Evidence. TIPS-1 and TIPS-2 (India)** showed that the Polycap produced the expected reductions in blood pressure, LDL-cholesterol and heart rate with acceptable tolerability. **UMPIRE, IMPACT and Kanyini-GAP** demonstrated substantially improved **adherence** compared with usual care. **TIPS-3 (NEJM 2021)** — a large primary-prevention trial in intermediate-risk people without cardiovascular disease — showed that a **polypill plus aspirin reduced major cardiovascular events**. **SECURE (NEJM 2022)** — the key secondary-prevention trial — randomised patients **over 65 who had had a recent myocardial infarction** to a polypill (aspirin, ramipril, atorvastatin) or usual care and showed a **significant reduction in cardiovascular death, myocardial infarction, stroke and urgent revascularisation, driven substantially by better adherence**. **PolyIran** confirmed benefit at a population level in a primary-care setting.
5. **Limitations and criticisms. Loss of individualisation** — doses cannot be titrated separately, so it is unsuitable where one component must be adjusted or where the patient is already well controlled on tailored therapy. **An adverse effect from one component requires stopping the whole tablet**, losing the benefit of the others. **Contraindications and interactions** — an ACE inhibitor in bilateral renal artery stenosis, a beta-blocker in asthma or severe bradycardia, a diuretic in gout or hyponatraemia, and aspirin where bleeding risk is high. **In the older adult the specific risks are hypotension, falls, hyperkalaemia, acute kidney injury, hyponatraemia and gastrointestinal bleeding — so it is generally more appropriate for stable, well-selected secondary prevention than for the frail or newly diagnosed patient.** Other issues are **medicalisation of healthy populations, uncertainty over the aspirin component in primary prevention, and regulatory and availability limitations** in many countries.

Latest Update – 1 Point (2023–Present)

6. In **July 2023 the World Health Organization added fixed-dose combination cardiovascular polypills to the WHO Model List of Essential Medicines**, for both **primary prevention in people at high cardiovascular risk and secondary prevention after an established event**. This was a landmark step, based on the SECURE, TIPS-3, PolyIran and UMPIRE evidence, and it **reclassifies the polypill from an experimental public-health idea into a recommended, essential therapeutic tool**. It is directly relevant to India, where the WHO **HEARTS** technical package and the national NCD programme promote standardised fixed-dose regimens to close the very large treatment gap in hypertension and secondary prevention. Practical caveat for the examination: current guidance regards the polypill as **most clearly indicated for secondary prevention and for high-risk primary prevention in resource-limited settings, not as a substitute for individualised titration in the frail older patient**.

PART II

RESPIRATORY MEDICINE AND SLEEP

Questions 35 – 54 · 20 questions · 7 sections

H. Respiratory Infection

Question 35. What is community-acquired pneumonia? Etiopathogenesis, diagnosis and management

15-Mark Essay | 10 textbook points + 2 latest updates

Textbook Core – 10 Points

1. **Definition.** Community-acquired pneumonia (CAP) is an **acute infection of the lung parenchyma** acquired **outside hospital**, or within **48 hours of admission**, characterised by **new symptoms and signs of lower respiratory tract infection together with a new radiographic infiltrate**. The presence of a **parenchymal focus on imaging distinguishes pneumonia from bronchitis**. **About two out of three hospitalisations for pneumonia are in patients over 70 years of age.**

2. **Aetiology by setting (high-yield).**

Setting	Predominant organisms
Older community patients	**S. pneumoniae, H. influenzae** and **respiratory viruses** are more frequent than in younger adults, whereas **Legionella, Mycoplasma and Chlamydophila are less frequent**
Nursing home residents	**Aspiration pneumonia**, **Gram-negative enteric bacilli** and **multiple anaerobes**
Viral (10–30% of CAP)	Influenza (including in vaccinated patients), **RSV**, parainfluenza, rhinovirus, adenovirus; emerging coronaviruses — **SARS, MERS, COVID-19**, whose incidence, severity and fatality all rise with age (>30% mortality over 75–80)
Others	**M. tuberculosis** (subacute/chronic — very relevant in India), **Aspergillus** in old TB cavities or lung abscess; fungal infection only with severe immunosuppression

   Multi-microbial pneumonia is common in the elderly.

3. **Pathogenesis.** Infection follows **micro-aspiration of oropharyngeal secretions** colonised by pathogens, and less often inhalation of aerosols or haematogenous spread. Ageing impairs every defence layer: **reduced cough and glottic reflexes with silent aspiration**, **impaired mucociliary clearance**, **reduced elastic recoil and lung "inflammageing"**, **immunosenescence with poor IgM response to pneumococcus**, malnutrition, sarcopenia and reduced ventilatory reserve. Structural changes — **bronchiectasis, tracheobronchomalacia, old TB scarring, sabre-sheath trachea** — impair mucus clearance and predispose to infection.

4. **Risk factors. Age itself**; **dysphagia and stroke**, **Parkinson's disease**, dementia, sedatives and antipsychotics, **GORD**, poor oral hygiene and **edentulism**, tube feeding, COPD, heart failure, diabetes, chronic kidney disease, malignancy, **inhaled corticosteroids**, proton pump inhibitors, alcohol, smoking, **institutionalisation**, and immobility.

5. **Clinical presentation — the geriatric pitfall. The classical picture of malaise, high fever, cough and pleuritic chest pain is rarely complete in the elderly. Fever and cough are absent in 30–40% of cases**, and onset is often gradual with less-marked signs, leading to **delayed diagnosis**. Presentation may be with **delirium, falls, functional decline, anorexia, incontinence or decompensation of a comorbidity**. Warning signs on examination: **tachypnoea > 30/min, tachycardia > 110/min, fever or hypothermia**, hypotension and hypoxaemia.

6. **Imaging.** A **chest radiograph (PA and lateral)** is recommended in every patient with suspected pneumonia, mainly to **distinguish pneumonia from bronchitis**. Look for alveolar consolidation, mixed alveolar–interstitial shadowing, atelectasis, and hidden **retrocardiac or perihilar** opacities. **The radiographic pattern is poorly related to the causative organism.** A **chest CT** is indicated when the film is normal but pneumonia is still suspected, in very early disease, or to detect pulmonary embolism. **Point-of-care lung ultrasound** shows B-lines with excellent sensitivity and specificity, and later "hepatisation" of consolidated lung — very useful in nursing homes and home care where radiography is difficult.

7. **Laboratory and microbiological tests.** Not required for patients managed at home. In hospitalised patients: **full blood count** (leucocytosis suggests bacterial infection but **may be absent in older patients**), **CRP**, **urea, electrolytes, creatinine and glucose** (dehydration, renal impairment and hyperglycaemia are common), **pulse oximetry**, and **arterial blood gas** if there is chronic respiratory failure, cyanosis, sweating or reduced consciousness. **Blood cultures and sputum examination in all patients admitted with pneumonia**; add **urinary pneumococcal and Legionella antigens**, respiratory viral PCR, and — in India — **sputum for AFB/CBNAAT** where the course is subacute.

8. **Severity assessment and the decision to admit.** Use **CURB-65** (Confusion, Urea > 7 mmol/L, Respiratory rate ≥ 30, Blood pressure < 90/60, age ≥ 65) or the **Pneumonia Severity Index**. Three reasons prompt hospital referral: (i) **the diagnosis is unclear and a serious alternative such as pulmonary embolism or heart failure is suspected**; (ii) **pneumonia is likely but the patient cannot be safely cared for at home** (help with ADLs, prevention of pressure sores and dehydration, follow-up); (iii) **severity signs are present**. **Remember that scores underestimate risk in the frail — always add a functional and social assessment.**
9. **Antibiotic therapy.** Initial therapy is **probabilistic** and must cover **S. pneumoniae and H. influenzae**. For outpatients **with comorbidities** (which applies to most older patients), a single oral agent may be used — **amoxicillin, amoxicillin-clavulanate, an advanced macrolide (azithromycin or clarithromycin), doxycycline, or a respiratory fluoroquinolone**. Per **ATS/IDSA 2019**: **a β-lactam plus a macrolide or doxycycline**, or a respiratory fluoroquinolone alone. In **nursing home patients the regimen must also cover anaerobes** because of aspiration. For **severe CAP** use a broad-spectrum combination, and cover **Pseudomonas** if there is bronchiectasis or recent β-lactam/fluoroquinolone use within three months. In older people **reduce doses for renal function**, and beware fluoroquinolone-associated **QT prolongation, tendinopathy, delirium, hypoglycaemia and C. difficile**.
10. **Supportive care, the 48-hour review and prevention.** Give **oxygen (target 88–92% if at risk of CO_2 retention)**, fluids, **early mobilisation, chest physiotherapy, VTE prophylaxis, nutritional support, delirium prevention and swallow assessment**. **Assessment at 48 hours is the key step** — defervescence and clinical stability indicate a favourable response, allowing **IV-to-oral switch**. If the response is unfavourable, consider **resistant organism, tuberculosis or virus, empyema or encysted effusion, non-infectious lung disease, drug adverse effect, or worsening comorbidity**, and repeat imaging including CT. **Persistence of the radiological shadow at 12 weeks, marked weight loss or heavy tobacco/asbestos exposure must raise suspicion of lung cancer** and prompt bronchoscopy or CT. Prevention: **influenza, pneumococcal and COVID-19 vaccination, smoking cessation, oral hygiene, dysphagia management, and review of sedatives and PPIs**.

Latest Updates – 2 Points (2023–Present)

11. **Corticosteroids in severe CAP.** The **CAPE COD trial (NEJM 2023)** showed that **intravenous hydrocortisone 200 mg/day started within 24 hours reduced 28-day mortality** in patients admitted to intensive care with severe CAP. Current international guidance therefore **recommends adjunctive corticosteroids in severe CAP** (but **not** in influenza or suspected aspergillosis), with careful monitoring for **hyperglycaemia and delirium** in older patients.
12. **2024 ACIP pneumococcal vaccination update.** The routine age for pneumococcal vaccination in the United States was **lowered from 65 to 50 years**, and a **single dose of a higher-valency conjugate vaccine (PCV20 or PCV21) is now recommended** for pneumococcal-naïve adults, simplifying the older PCV13-then-PPSV23 sequence. This reflects the substantial residual burden of pneumococcal disease from age 50 onward.

Current Practice Update

Textbook	Antibiotics plus supportive care; steroids not mentioned. Pneumococcal vaccination framed around PCV13/PPSV23 from age 65.
Current recommendation	Adjunctive hydrocortisone in severe CAP (CAPE COD 2023); single-dose PCV20/PCV21 from age 50 (ACIP 2024).
Exam approach	Finish the management section with: "CAPE COD (NEJM 2023) — hydrocortisone reduces mortality in severe CAP; ACIP 2024 has lowered the pneumococcal vaccination age to 50."

Question 36. Pneumonia in the elderly

7-Mark Answer | 6 textbook points + 1 latest update

Textbook Core – 6 Points

1. **Burden.** Pneumonia is a major cause of hospitalisation and death in old age — **about two out of three pneumonia admissions are patients over 70**. It is often the **terminal event** in frailty and dementia ("the old man's friend"), and carries much higher mortality than in younger adults.
2. **Predisposing factors specific to ageing. Immunosenescence** with a poor IgM response to pneumococcal polysaccharide, **impaired cough and swallowing reflexes causing silent aspiration**, reduced mucociliary clearance, loss of lung elastic recoil and **"inflammageing"**, reduced chest wall compliance, **poor oral hygiene and edentulism**, malnutrition, sarcopenia, and structural airway change (bronchiectasis, old TB scarring, tracheobronchomalacia).

3. **Aetiology. S. pneumoniae, H. influenzae and respiratory viruses** predominate, while **Legionella, Mycoplasma and Chlamydophila are less common than in younger adults**. In **nursing home residents**, **aspiration pneumonia, Gram-negative enteric bacilli and anaerobes** are frequent. **Multi-microbial infection is common.** In India also consider **tuberculosis** and **aspergillosis in post-tubercular cavities**.
4. **Atypical presentation. Fever and cough are absent in 30–40%.** The classical lobar picture is rarely complete. Common presentations are **delirium, falls, sudden functional decline, anorexia, incontinence, tachypnoea alone**, or decompensated heart failure or diabetes. Signs to look for: **respiratory rate > 30/min, heart rate > 110/min, fever or hypothermia**. **Tachypnoea is the most sensitive early sign.**
5. **Diagnostic approach. Chest radiograph** to separate pneumonia from bronchitis; **CT** if the film is normal but suspicion is high; **point-of-care lung ultrasound** is very useful in nursing homes and home care. **Leucocytosis may be absent**; check CRP, renal function and glucose, and **oxygen saturation**. In hospitalised patients take **blood cultures and sputum**. Assess severity with **CURB-65**, remembering that **age ≥ 65 already scores one point**.
6. **Management and prevention.** Decide on **site of care** using severity, frailty and the ability to be cared for at home. Give **empirical antibiotics covering pneumococcus and H. influenzae**, with **anaerobic cover in nursing home/aspiration pneumonia**, renally adjusted. Add **oxygen, hydration, early mobilisation, VTE prophylaxis, nutrition, swallow assessment and delirium prevention**. **Review at 48 hours**; failure to improve suggests resistance, tuberculosis, empyema, an alternative diagnosis or underlying **lung cancer** (especially if shadowing persists at 12 weeks). Prevent with **influenza, pneumococcal and COVID-19 vaccines, oral hygiene, smoking cessation, and review of sedatives, antipsychotics and PPIs**.

Latest Update – 1 Point (2023–Present)

7. **Respiratory syncytial virus (RSV) is now a vaccine-preventable cause of pneumonia in the elderly.** Following the **2023 licensure of adjuvanted and mRNA RSV vaccines**, the **2024 ACIP recommendation** is a **single lifetime dose of RSV vaccine for all adults aged 75 and above, and for those aged 50–74 with risk factors** (chronic lung or heart disease, diabetes, immunocompromise, frailty, nursing home residence). RSV causes severe lower respiratory infection and exacerbations in older adults comparable to influenza, and vaccination is now part of the standard geriatric immunisation package alongside influenza, pneumococcal and COVID-19 vaccines.

Question 37. Hospital-acquired pneumonia

7-Mark Answer | 6 textbook points + 1 latest update

Textbook Core – 6 Points

1. **Definition and types.** Hospital-acquired (nosocomial) pneumonia is pneumonia developing **48 hours or more after hospital admission and not incubating at the time of admission**. **Ventilator-associated pneumonia (VAP)** arises more than 48 hours after endotracheal intubation. **Early-onset** (within 4 days) tends to involve community-type organisms; **late-onset** (day 5 onward) involves **multidrug-resistant** organisms.
2. **Why it is severe in older patients.** Nosocomial respiratory infections are **often severe because of resistance of the causative bacteria to antibiotics and because of the vulnerability of patients already weakened by the illness that brought them to hospital**. Additional geriatric contributors: **immobility and supine posture, dysphagia and silent aspiration, sedatives, nasogastric tubes, delirium, malnutrition and poor oral hygiene**.
3. **Organisms. S. aureus (including MRSA), Pseudomonas aeruginosa and enteric Gram-negative bacteria** predominate, frequently with **antibiotic resistance**. Pneumonia in patients recently treated with antibiotics is often due to **complex, resistant or polymicrobial** flora. In Indian hospitals, **Acinetobacter baumannii, Klebsiella pneumoniae with carbapenem resistance** and ESBL producers are major concerns.
4. **Clinical features and diagnosis.** New or worsening **infiltrate on chest radiograph** plus at least two of — **fever or hypothermia, leucocytosis or leucopenia, purulent secretions, worsening oxygenation**. In the old, **delirium or unexplained deterioration may be the only clue**. Obtain **lower respiratory tract samples (deep tracheal aspirate, or bronchoalveolar lavage in the ventilated) before starting antibiotics**, plus **blood cultures**. Differentiate from **atelectasis, pulmonary embolism, pulmonary oedema, ARDS and drug-induced lung injury**.
5. **Treatment.** Start **prompt empirical broad-spectrum therapy** guided by **local antibiogram, prior antibiotic exposure and risk factors for MDR organisms** (recent IV antibiotics within 90 days, prolonged stay, septic shock, ARDS, renal replacement therapy). Typical empirical cover: an **antipseudomonal β-lactam (piperacillin-tazobactam, cefepime or meropenem) ± MRSA cover (vancomycin or linezolid)** ± a second antipseudomonal agent in septic shock. **De-escalate at 48–72 hours on culture results and treat for 7 days** in most patients. Adjust doses for renal function and monitor for **nephrotoxicity, C. difficile and delirium**.

6. **Prevention (highly examinable). Hand hygiene**; **semi-recumbent positioning at 30–45°**; **daily sedation interruption and assessment of readiness to extubate**; **subglottic secretion drainage**; **oral care with chlorhexidine and regular dental hygiene**; **early mobilisation and chest physiotherapy**; **avoid unnecessary nasogastric tubes, sedatives and acid suppression**; **swallow screening after stroke**; and **antimicrobial stewardship with isolation of colonised patients**.

Latest Update – 1 Point (2023–Present)

7. **IDSA guidance on antimicrobial-resistant Gram-negative infections (updated 2023–2024)** now defines preferred agents for resistant hospital-acquired and ventilator-associated pneumonia: **ceftazidime-avibactam, ceftolozane-tazobactam or imipenem-relebactam** for difficult-to-treat **Pseudomonas**; **ceftazidime-avibactam or meropenem-vaborbactam** for KPC-producing carbapenem-resistant Enterobacterales; **ceftazidime-avibactam plus aztreonam** for metallo-β-lactamase producers; and **cefiderocol or sulbactam-durlobactam** for carbapenem-resistant **Acinetobacter**. The parallel message is **shorter courses — 7 days is sufficient for most HAP/VAP**, with de-escalation guided by culture and clinical response.

Question 38. Case — 67-year-old man with cough, purulent expectoration, fever, tachypnoea, tachycardia and hypotension

7-Mark Answer | 6 textbook points + 1 latest update

Recognise the diagnosis in the first line: this is **severe community-acquired pneumonia with sepsis and septic shock**. Marks are earned for immediate resuscitation running in parallel with investigation, not sequentially.

Textbook Core – 6 Points

1. **Immediate recognition and triage.** Cough with purulent sputum and fever indicate **pneumonia**; **tachypnoea, tachycardia and hypotension indicate sepsis with shock**. Apply **ABCDE**, secure two large-bore cannulae, attach monitoring and calculate **CURB-65** — this patient already scores for **age ≥ 65, respiratory rate ≥ 30 and systolic BP < 90**, giving a high score that mandates **admission to a high-dependency or intensive care area**.
2. **The first hour — resuscitation bundle. Oxygen** to a target of **94–98% (88–92% if there is known chronic CO_2 retention)**; **blood cultures before antibiotics**; **measure serum lactate**; **intravenous broad-spectrum antibiotics within one hour**; **balanced crystalloid 30 mL/kg** given in aliquots with reassessment; and **early noradrenaline** through a peripheral or central line if the mean arterial pressure remains below 65 mmHg despite fluids. **In the older patient give fluid in 250–500 mL boluses with repeated reassessment, because rapid loading precipitates pulmonary oedema in unrecognised diastolic dysfunction.** Insert a urinary catheter for hourly output.
3. **Investigations. Chest radiograph** (consolidation; look for effusion or cavitation); **arterial blood gas with lactate**; **full blood count, CRP or procalcitonin, urea, electrolytes, creatinine, liver function, glucose, coagulation profile**; **blood cultures × 2 and sputum Gram stain and culture**; **urinary pneumococcal and Legionella antigen**; **respiratory viral PCR including influenza and COVID-19**; **sputum for AFB/CBNAAT** in the Indian setting; **ECG and troponin** (type 2 myocardial infarction and new atrial fibrillation are common); **bedside echocardiography and lung ultrasound**. Add **CT chest ± CT pulmonary angiogram** if the radiograph is normal or pulmonary embolism is possible.
4. **Antibiotic therapy.** Empirical cover for **severe CAP**: **intravenous ceftriaxone or amoxicillin-clavulanate (or piperacillin-tazobactam) PLUS a macrolide (azithromycin/clarithromycin) or a respiratory fluoroquinolone (levofloxacin)**. Add **antipseudomonal cover** if there is bronchiectasis, structural lung disease or recent β-lactam/fluoroquinolone use within three months; add **anaerobic cover** if aspiration is suspected; add **oseltamivir** during influenza season. Adjust doses for renal function, then **de-escalate and switch to oral therapy at 48–72 hours** once stable.
5. **Ongoing organ support and monitoring.** Target **MAP ≥ 65 mmHg**, adequate urine output and falling lactate. Escalate respiratory support stepwise — **controlled oxygen → high-flow nasal oxygen → non-invasive ventilation (pH < 7.35) → intubation**. Add **vasopressin** if noradrenaline requirement is high; give **hydrocortisone in refractory shock**. Watch for **acute kidney injury, delirium, hyperglycaemia, gastrointestinal bleeding and pressure sores**; provide **VTE prophylaxis, early enteral nutrition, glycaemic control and early mobilisation**. Reassess formally at **48 hours**.
6. **Complications and goals of care.** Look for **parapneumonic effusion and empyema, lung abscess, ARDS, acute kidney injury, atrial fibrillation, myocardial infarction and multi-organ failure**. If the patient does not improve, consider **resistant organism, tuberculosis, empyema requiring drainage, pulmonary embolism or an underlying lung cancer**. In parallel, undertake **early discussion of goals of care, ceilings of treatment and resuscitation status** with the patient and family, and involve palliative care if the trajectory is poor. Plan **vaccination and smoking cessation** before discharge.

Latest Update – 1 Point (2023–Present)

7. **CAPE COD trial (NEJM 2023)** — in adults admitted to intensive care with **severe community-acquired pneumonia**, **intravenous hydrocortisone 200 mg per day started within 24 hours reduced 28-day mortality**. **Adjunctive corticosteroids are therefore now recommended in severe CAP** (excluding influenza and suspected aspergillosis). Contemporary sepsis practice has also shifted to **balanced crystalloids rather than saline**, **restrictive fluid strategy with early vasopressors** (including peripheral noradrenaline), and **dynamic assessment of fluid responsiveness** rather than fixed large-volume loading — all particularly important in the frail older patient.

I. Obstructive Airway Disease and Inhaled Therapy

Question 39. Management of chronic obstructive airway disease

7-Mark Answer | 6 textbook points + 1 latest update

Textbook Core – 6 Points

1. **Confirm the diagnosis and assess.** COPD requires **post-bronchodilator spirometry showing a persistent airflow limitation**. **In older adults use the lower limit of normal rather than the fixed $FEV_1/FVC < 0.70$ ratio, because ageing alone produces a "pseudo-obstructive" pattern and the fixed ratio over-diagnoses COPD in the old.** Assess with the **mMRC dyspnoea scale or CAT score**, exacerbation history, and **GOLD ABE grouping**. Careful history and spirometry usually distinguish COPD from asthma and exclude comorbid conditions.
2. **Non-pharmacological measures — the foundation. Smoking cessation is the single most effective intervention and works at any age**. **Pulmonary rehabilitation** — a comprehensive, tailored programme of exercise training, education and behaviour change — improves symptoms, exercise capacity and quality of life. Add **vaccination (influenza, pneumococcal, COVID-19, RSV, pertussis)**, **nutritional support** (low BMI predicts mortality), reduction of **biomass fuel and indoor air pollution exposure** — a major issue in Indian households — and treatment of anxiety and depression.
3. **Inhaled pharmacotherapy. Long-acting bronchodilators are the mainstay**: **LAMA** may be added to a **LABA** when breathlessness predominates. In patients who remain breathless or exacerbate frequently, **regardless of FEV_1**, a **LAMA can be added to an inhaled LABA-corticosteroid combination or vice versa (triple therapy)**. **Inhaled corticosteroids** reduce exacerbations, particularly with **blood eosinophils ≥ 300 cells/µL** or a history of asthma, but cause a **small but significant increase in non-fatal pneumonia** — a risk that does not outweigh their benefit in appropriate patients. Re-evaluate after starting therapy, judging success by **symptoms, exercise tolerance and ability to perform activities of daily living rather than by lung function alone**. **Always check inhaler technique and prefer fixed-dose combinations to reduce device burden.**
4. **Additional oral therapy.** If symptoms persist or the patient cannot use inhalers, **slow-release theophylline** may be tried at **low dose in older people**, watching for gastrointestinal effects and interactions with antibiotics during exacerbations. **Roflumilast (a PDE-4 inhibitor)** is an option in frequent exacerbators with chronic bronchitis and copious sputum — monitor for **weight loss, diarrhoea and mood disturbance**, and note the limited safety evidence in the elderly. A trial of **mucolytics or high-dose N-acetylcysteine** may help viscid sputum. **Nebulised bronchodilators** help very advanced disease or hopeless inhaler technique. **Maintenance oral corticosteroids should generally be avoided — the risks outweigh the benefits.**
5. **Management of exacerbations.** Exacerbations account for **up to 10% of medical admissions**; **one-third are readmitted within 30 days and 15% die within three months**. Common organisms: **H. influenzae, S. pneumoniae and M. catarrhalis**. Give **bronchodilators, controlled oxygen to a target saturation of 88–92%**, and **oral corticosteroids for 5–7 (up to 14) days**, which shorten stay and reduce treatment failure. **Antibiotics when there is increased cough with purulent sputum** — beta-lactam ± inhibitor, doxycycline or a fluoroquinolone; add antipseudomonal cover if bronchiectasis coexists; **avoid antibiotics if the sputum is not purulent**. **Non-invasive ventilation if pH < 7.35** despite maximal therapy — this reduces mortality, intubation and length of stay; **high-flow nasal oxygen** is an alternative, especially with do-not-intubate orders. Screen for **osteoporosis** in anyone receiving repeated steroid courses.

6. **Long-term support, comorbidity and end-of-life care.** Assess for **long-term oxygen therapy** in severe disease or cor pulmonale, and for **home NIV** in persistent hypercapnia. Manage comorbidities actively — **ischaemic heart disease and heart failure (cardioselective beta-blockers are safe and should be used when indicated), osteoporosis, obstructive sleep apnoea, anxiety, depression, sarcopenia and falls**. Consider **early-discharge and admission-avoidance teams** for suitable non-acidotic patients with good social support. **Patients judged to be in the last year of life should be placed on an end-of-life register and offered palliation including opioids and benzodiazepines for refractory breathlessness** — the move from active to palliative care all too often comes too late.

Latest Update – 1 Point (2023–Present)

7. **Biologic therapy has entered COPD.** The **BOREAS (NEJM 2023)** and **NOTUS (NEJM 2024)** trials showed that **dupilumab** (an anti-IL-4Rα monoclonal antibody) **significantly reduced exacerbations and improved lung function and quality of life in COPD with a type-2 inflammatory phenotype (blood eosinophils ≥ 300 cells/µL) already on triple inhaled therapy**, leading to regulatory approval in 2024–25 and its incorporation into the **GOLD 2025/2026** reports as an add-on for this phenotype. GOLD also continues to emphasise **single-inhaler triple therapy for exacerbators**, the **eosinophil count as the key biomarker for ICS use**, and **checking inhaler technique before any escalation**.

Question 40. Asthma management based on GINA guidelines

7-Mark Answer | 6 textbook points + 1 latest update

Textbook Core – 6 Points

1. **Diagnosis and the geriatric difficulty.** Asthma is **chronic airway inflammation with bronchial hyper-responsiveness and variable airflow obstruction**, causing episodic wheeze, cough, chest tightness and dyspnoea, which is **fully or partly reversible**. It is **under-diagnosed and misdiagnosed in older people**, who have **reduced awareness of bronchoconstriction**, an **impaired bronchodilator response** from reduced beta-2 receptor density, and coexisting COPD. Confirm with **spirometry showing reversibility** or peak flow variability, and exclude heart failure, COPD, GORD, vocal cord dysfunction and drug-induced cough.
2. **Assessment of control (GINA framework).** Over the last 4 weeks ask about **daytime symptoms more than twice a week, any night waking due to asthma, reliever use more than twice a week, and activity limitation** — none = well controlled, 1–2 = partly controlled, 3–4 = uncontrolled. Separately assess **risk factors for exacerbation** (poor adherence, incorrect inhaler technique, high SABA use, low FEV_1, comorbidity) and **treatment side effects**. Management follows a **cycle of assess → adjust treatment → review response**.
3. **The central GINA principle. Short-acting beta-2 agonist (SABA)-only treatment is no longer recommended at any step** — it increases exacerbations and asthma death. **Every adult with asthma should receive inhaled-corticosteroid-containing therapy**, either as regular treatment or, at minimum, as ICS-formoterol taken whenever a reliever is needed.
4. **GINA Track 1 (preferred) — ICS-formoterol reliever.**

Step	Treatment
Steps 1–2	**As-needed low-dose ICS-formoterol** (anti-inflammatory reliever, "AIR")
Step 3	**Low-dose maintenance ICS-formoterol plus as-needed ICS-formoterol (MART)**
Step 4	**Medium-dose maintenance ICS-formoterol (MART)**
Step 5	Add **LAMA**; refer for phenotyping and **biologic therapy**; consider high-dose ICS-formoterol

   Track 2 is used when Track 1 is not possible or the patient is already stable on another regimen: **daily ICS (step 2) → ICS-LABA (steps 3–4) → add-on therapy (step 5)**, with a **SABA reliever**.
5. **Add-on and add-back therapy in older adults. Adding a LABA gives better control than increasing the ICS dose**, but **a LABA must never be used as monotherapy** because it does not treat inflammation. **LAMAs** (tiotropium, glycopyrronium, aclidinium) are of limited benefit alone but are useful added to ICS-LABA in the old, where reduced beta-2 receptor density relatively increases anticholinergic benefit — but they are **contraindicated in urinary retention and glaucoma**. **Leukotriene modifiers** are simple oral agents with proven efficacy in older adults; **slow-release theophylline** has a narrow therapeutic index. **Biologics** for severe disease target **IgE (omalizumab), IL-5 (mepolizumab, reslizumab, benralizumab)** and **IL-4/IL-13 (dupilumab)**, but **efficacy and safety data in patients over 65 remain sparse**, so their use should be restricted to carefully selected cases.

6. **Practical points and drugs to avoid.** Always use an **MDI with a spacer** to reduce oropharyngeal candidiasis and dysphonia and to overcome coordination problems; **tobacco smoking limits the efficacy of inhaled corticosteroids**. Monitor control by **symptoms, night-time relief, peak flow and where available exhaled nitric oxide**. **Beta-blockers (including topical glaucoma drops), verapamil, aspirin and NSAIDs can worsen asthma**. Watch for ICS-related **osteoporosis, cataract, glaucoma, skin thinning and dysphonia** in long-term use, and give **a written asthma action plan** with carer involvement where cognition is impaired.

Latest Update – 1 Point (2023–Present)

7. **GINA 2024–2025 strategy reports** have consolidated the **two-track approach with anti-inflammatory reliever therapy as the preferred option at every step**, and now explicitly extend **anti-inflammatory reliever (AIR) therapy to mild asthma**, so that **no patient is left on SABA alone**. They also formalise **anti-inflammatory reliever combinations of ICS with a short-acting beta-2 agonist (ICS-SABA)** as an alternative where ICS-formoterol is unavailable, emphasise **biomarker-based phenotyping (blood eosinophils, FeNO, IgE) before escalating to a biologic**, and highlight **treatable traits and comorbidity management (obesity, GORD, rhinosinusitis, OSA, anxiety)** — an approach that fits geriatric multimorbidity particularly well.

Current Practice Update

Textbook	Stepwise therapy with a SABA reliever; add LABA before increasing ICS; theophylline and leukotriene modifiers as add-ons.
Current recommendation	GINA — SABA-only treatment abandoned; as-needed or maintenance-and-reliever ICS-formoterol (AIR/MART) is the preferred track at all steps.
Exam approach	Open the management section with "GINA no longer recommends SABA-alone therapy — every adult receives ICS-containing treatment," then draw the two-track step table.

Question 41. Senile emphysema

7-Mark Answer | 6 textbook points + 1 latest update | ***Asked ×2***

Textbook Core – 6 Points

1. **Definition and why the name is a misnomer.** "Senile emphysema" (better called the **ageing or senile lung**) is the **physiological enlargement of alveolar ducts and alveoli that occurs with normal ageing**. **Crucially, it occurs WITHOUT destruction of alveolar walls and without inflammation** — the features that define true emphysema. It is therefore a **normal age-related change, not a disease**, and the term "emphysema" is strictly incorrect.
2. **Mechanism.** There is an **alteration in the collagen and elastic fibre network** of the lung, leading to **dilated alveolar ducts and alveolar enlargement** and a **loss of elastic recoil**. Loss of recoil reduces the tethering (radial traction) of small airways, so they **close at higher lung volumes**, producing **premature airway closure, air trapping and hyperinflation**. Alveolar surface area for gas exchange falls (roughly 4% per decade after 30).
3. **Associated extrapulmonary changes.** The chest wall stiffens: **rib cage stiffening, degenerative spinal change with kyphosis, reduced thickness of parietal muscles and reduced respiratory muscle mass**, giving **decreased chest wall compliance, increased anteroposterior diameter and a flattened diaphragm with decreased curvature**. The result is **increased work of breathing, a higher respiratory rate with lower tidal volume, and reduced exercise tolerance**.
4. **Physiological consequences (very examinable).**

Parameter	Change with age
Residual volume (RV), functional residual capacity (FRC)	**Increased** (air trapping)
Total lung capacity (TLC)	**Essentially unchanged** or slightly reduced by chest-wall restriction
Vital capacity (FVC)	**Decreased**
FEV_1 and FEV_1/FVC ratio	**Decreased** — a disproportionate fall in FEV_1 gives a **"pseudo-obstructive" spirometric pattern**
DLCO	Mildly reduced (loss of surface area)
PaO_2	Falls gradually with age (ventilation–perfusion mismatch from small airway closure); $PaCO_2$ unchanged
Ventilatory response	**Markedly impaired response to hypoxia and hypercapnia** → lower threshold for ventilatory decompensation

5. **Clinical relevance.** The senile lung is **asymptomatic at rest** — there is no cough, sputum or wheeze — but there is **reduced ventilatory reserve**, so exercise tolerance falls and the older person **decompensates rapidly during pneumonia, heart failure, surgery or anaesthesia**. Physical signs may mimic emphysema (**barrel chest, reduced expansion, hyper-resonance, quiet breath sounds**), and this must not be mistaken for COPD.
6. **Distinguishing senile lung from true emphysema (the crux of the answer).**

Feature	Senile lung	True emphysema
Alveolar wall destruction	**Absent**	**Present**
Inflammation	Absent	Present
Airspace enlargement	Uniform, ductal	Non-uniform (centriacinar/panacinar), with bullae
Symptoms	None at rest	Chronic breathlessness, cough, sputum
Bronchodilator reversibility / progression	Stable, non-progressive	Progressive decline
Cause	Normal ageing	Smoking, biomass smoke, α_1-antitrypsin deficiency

The key practical implication: use the lower limit of normal (LLN) for the FEV_1/FVC ratio rather than the fixed 0.70 cut-off, because the fixed ratio misclassifies the normal ageing lung as airflow obstruction and over-diagnoses COPD in older people. There is **no specific treatment** — management is prevention: **smoking avoidance, exercise and pulmonary reserve maintenance, vaccination, and avoidance of biomass smoke and air pollution**.

Latest Update – 1 Point (2023–Present)

7. **GOLD 2023–2026 reports formally recognise the diagnostic problem posed by the ageing lung** by introducing the categories of **"Pre-COPD"** (respiratory symptoms and/or structural or functional abnormality with **normal spirometry**) and **"PRISm" — Preserved Ratio Impaired Spirometry** (**$FEV_1/FVC \geq 0.70$ but $FEV_1 < 80\%$ predicted**), both of which carry increased risk of progression and mortality. GOLD also continues to caution that the **fixed FEV_1/FVC ratio of 0.70 over-diagnoses airflow obstruction in older adults**, and to recommend confirming abnormality against age-appropriate reference equations — the modern, guideline-level restatement of exactly what "senile emphysema" describes.

Question 42. Metered dose inhalers and inhaler technique in the older adult

7-Mark Answer | 6 textbook points + 1 latest update

Textbook Core – 6 Points

1. **Why it matters.** Inhaled therapy is first-line in **COPD and asthma**, but the drug only reaches the airway if the technique is correct. **Using inhaler devices is a demanding task for older patients** because it needs **coordination and respiratory muscle strength**. Poor technique is a major cause of apparent **"treatment failure"** and leads to unnecessary escalation of therapy — **always check the technique before stepping up treatment**.
2. **Correct MDI technique (stepwise).** (1) Remove the cap and **shake well**; (2) sit or stand upright and **breathe out fully, away from the device**; (3) seal the lips around the mouthpiece; (4) **begin a slow deep inhalation and actuate the canister at the same moment**; (5) continue inhaling steadily over **4–5 seconds**; (6) **hold the breath for 10 seconds**; (7) wait **30 seconds** before a second puff; (8) **rinse the mouth** after an inhaled corticosteroid.
3. **Common errors.** Failure to shake the canister; not exhaling first; **poor hand-breath coordination** — actuating before or after inhalation; inhaling too fast; **no breath-hold**; firing into the mouth while breathing out; using an empty canister; and not rinsing, leading to **oropharyngeal candidiasis and dysphonia**. Multiple puffs fired in quick succession.
4. **Age-related barriers. Most cognitively intact older patients without physical limitation can use an inhaler correctly, but learning and coordination difficulties are present in about 20%. Cognitive impairment has a dramatic impact**: **an MMSE score below 24 is associated with a very high risk of failure**, and the **abnormal pentagon-copying test alone identifies patients unable to use inhaler devices**. Other barriers: **poor manual dexterity, weak hand grip, arthritis, impaired vision and impaired executive function** — the last is especially common in hospitalised patients.

5. **Device selection.** **The perfect device does not exist** — the choice must be individualised to the patient's cognition, dexterity and inspiratory flow. An **MDI with a spacer (valved holding chamber)** removes the need for hand–breath coordination and **reduces oropharyngeal deposition, candidiasis and dysphonia**; it is the preferred combination in most older adults. **Dry powder inhalers** need adequate inspiratory flow, often lacking in severe COPD and frailty, but need no coordination. **Breath-actuated MDIs** help coordination problems. **Nebulisers** are reserved for very advanced disease or when the inhaler technique is so poor that no other relief is possible; **adapters fitted to the inhaler ends** can help weak grip.
6. **Improving technique and adherence.** Use **fixed-dose combination inhalers** to reduce the number of devices, since **device quantity impacts adherence**; keep the patient on **one device type** wherever possible. Teach by **physical demonstration with teach-back**, not verbal instruction alone. Ask the patient to **bring all inhalers to every clinic visit**, and **re-train the patient and/or the caregiver**. **Screen cognition (MMSE or a secondary memory test)** to identify those at risk of poor compliance, and involve the carer or care-home staff for supervised administration.

Latest Update – 1 Point (2023–Present)

7. **GOLD 2025 / 2026** makes checking inhaler technique a **mandatory step at every visit and a required check before any escalation of therapy**, and emphasises **choosing the device before choosing the drug**. GOLD now also asks clinicians to consider the **environmental impact of inhalers** — conventional pressurised MDIs use high global-warming-potential HFA-134a/227ea propellants, and the shift is towards **low-GWP propellants (HFA-152a, HFO-1234ze)** and greater use of **dry powder inhalers where clinically appropriate** — though in older adults **clinical suitability and inspiratory flow must always outweigh environmental considerations**.

J. Parenchymal and Pleural Disease

Question 43. Diffuse parenchymal lung disease in older adults

15-Mark Essay | 10 textbook points + 2 latest updates | ***Asked ×3***

Source note: Diffuse parenchymal lung disease is **not covered adequately as a standalone topic in the uploaded textbook** — Pathy's refers to it mainly in the context of ageing lung changes, drug-related pneumonitis and the differential diagnosis of B-lines on ultrasound. The answer below is built on current standard respiratory guidance and is labelled accordingly.

Textbook Core – 10 Points

1. **Definition.** Diffuse parenchymal lung disease (DPLD), or **interstitial lung disease**, is a heterogeneous group of disorders characterised by **inflammation and/or fibrosis of the alveolar interstitium, alveoli and small airways**, producing a **restrictive ventilatory defect with impaired gas transfer** — reduced **TLC, FVC and DLCO with a preserved or raised FEV_1/FVC ratio**.
2. **Epidemiology in older adults.** DPLD is **predominantly a disease of later life**. **Idiopathic pulmonary fibrosis (IPF) has a median age at diagnosis of about 65–70**, a male predominance and rising incidence with each decade; it is rare below 50. **Interstitial lung abnormalities** are found incidentally on CT in up to **10% of older people** and a proportion progress. In India, **hypersensitivity pneumonitis and connective tissue disease-associated ILD** form a larger share than in Western registries, and **post-tubercular fibrosis** is an important mimic.
3. **Pathogenesis.** Modern understanding of IPF is of **repetitive micro-injury to a genetically susceptible, aged alveolar epithelium** rather than primary inflammation. Ageing mechanisms are central — **telomere shortening, cellular senescence, mitochondrial dysfunction, impaired autophagy and defective epithelial repair**. Injured type II pneumocytes drive **aberrant fibroblast activation and myofibroblast proliferation with excess extracellular matrix deposition** and formation of **fibroblastic foci**. Risk factors: **smoking, gastro-oesophageal reflux with micro-aspiration, dusts and metals, viral infection**, and gene variants (**MUC5B promoter polymorphism, telomerase TERT/TERC mutations, surfactant protein genes**). In hypersensitivity pneumonitis the mechanism is an immune response to inhaled organic antigen.

4. **Classification.**

Group	Examples
Known cause	**Connective tissue disease-ILD** (rheumatoid arthritis, systemic sclerosis, myositis, Sjögren), **hypersensitivity pneumonitis** (birds, mould, biomass), **pneumoconioses** (asbestosis, silicosis), **drug-induced** — **amiodarone, methotrexate, nitrofurantoin, bleomycin, immune checkpoint inhibitors**, radiation
Idiopathic interstitial pneumonias	**IPF (UIP pattern)**, non-specific interstitial pneumonia (NSIP), cryptogenic organising pneumonia (COP), acute interstitial pneumonia, desquamative interstitial pneumonia and respiratory bronchiolitis-ILD (smoking-related)
Granulomatous	**Sarcoidosis**
Rare / distinct	Lymphangioleiomyomatosis, Langerhans cell histiocytosis, pulmonary alveolar proteinosis, eosinophilic pneumonia

   A crucial modern concept is the **progressive pulmonary fibrosis (PPF)** phenotype — any non-IPF fibrotic ILD that shows worsening symptoms, physiology or radiology despite standard treatment.

5. **Clinical features. Insidious exertional breathlessness** and a **persistent dry cough**. **In older adults symptoms are frequently attributed to ageing, deconditioning, heart failure or COPD, causing years of diagnostic delay.** Signs: **fine bibasal end-inspiratory "Velcro" crackles** (an early and important sign in IPF), **finger clubbing (about 50% of IPF)**, and later **cyanosis, cor pulmonale and right heart failure**. Look for extrapulmonary clues to CTD — **arthritis, Raynaud phenomenon, sclerodactyly, mechanic's hands, rash, dry eyes and mouth**. Take a meticulous **occupational, environmental, avian, mould and drug history**.

6. **Investigations — imaging is central. Chest radiograph** shows reduced lung volumes with reticulonodular shadowing but is insensitive. **High-resolution CT is the key investigation.** The **usual interstitial pneumonia (UIP) pattern** — **subpleural, basal-predominant reticulation with honeycombing and traction bronchiectasis, and no significant ground-glass** — is diagnostic of IPF in the right clinical setting and **removes the need for biopsy**. Other patterns: **ground-glass with subpleural sparing (NSIP)**; **mosaic attenuation with air trapping and centrilobular nodules (hypersensitivity pneumonitis)**; **peripheral consolidation (organising pneumonia)**; **upper-zone perilymphatic nodules and hilar nodes (sarcoidosis)**.

7. **Other investigations. Full pulmonary function tests** — restrictive pattern with **reduced FVC, TLC and DLCO**; DLCO is the most sensitive early marker and the best correlate of prognosis. **Six-minute walk test with oximetry** to document **exertional desaturation**. **Serology** — ANA, rheumatoid factor, anti-CCP, ENA, myositis panel, ANCA; **serum precipitins** for hypersensitivity pneumonitis; serum ACE and calcium for sarcoidosis. **Bronchoalveolar lavage** for lymphocytosis (hypersensitivity pneumonitis, sarcoidosis) and to exclude infection. **Echocardiography** for pulmonary hypertension. **Surgical lung biopsy is high-risk in the elderly and is largely avoided; transbronchial lung cryobiopsy is a lower-risk alternative in selected patients.** The diagnosis should be made by a **multidisciplinary discussion of clinician, radiologist and pathologist** — this is the accepted gold standard.

8. **Management — general and non-pharmacological. Remove the cause** — antigen avoidance in hypersensitivity pneumonitis, stopping the culprit drug, smoking cessation. **Pulmonary rehabilitation** improves exercise capacity and quality of life. **Ambulatory and long-term oxygen** for resting or exertional hypoxaemia. **Vaccination** (influenza, pneumococcal, COVID-19, RSV). **Treat comorbidities** that materially affect outcome — **gastro-oesophageal reflux, obstructive sleep apnoea, pulmonary hypertension, ischaemic heart disease, depression, deconditioning and osteoporosis**. Manage **cough** and breathlessness symptomatically, with **low-dose opioids** where refractory.

9. **Management — pharmacological. IPF: antifibrotic therapy with nintedanib (a triple tyrosine kinase inhibitor; adverse effects diarrhoea, deranged LFTs, bleeding risk with anticoagulants) or pirfenidone (nausea, photosensitivity, weight loss)** — both slow the rate of FVC decline. **Corticosteroids and immunosuppression are harmful in IPF** (the PANTHER-IPF trial showed increased mortality with prednisolone + azathioprine + N-acetylcysteine) and must not be used. **Non-IPF ILD: corticosteroids ± mycophenolate, azathioprine or rituximab** for CTD-ILD and hypersensitivity pneumonitis; **nintedanib is licensed for the progressive pulmonary fibrosis phenotype** (INBUILD trial) and for systemic sclerosis-ILD (SENSCIS). **Lung transplantation** is the only curative option but **age over 65–70 with frailty and comorbidity usually precludes it**.

10. **Prognosis and natural history. IPF has the worst prognosis — median survival about 3–5 years from diagnosis**, comparable to many cancers. The course is variable: **slow decline, rapid decline, or stability punctuated by acute exacerbations**. **Acute exacerbation of IPF** — new ground-glass on a background of UIP without infection, heart failure or PE — carries an **in-hospital mortality above 50%**. Prognostic tools: the **GAP index (Gender, Age, Physiology — FVC and DLCO)**, and a **decline in FVC of ≥ 10% or DLCO of ≥ 15% over 6–12 months**. NSIP, COP and sarcoidosis carry a much better prognosis than IPF. Because trajectory is poor, **early advance care planning, symptom control and palliative care must run in parallel with disease-modifying therapy in older patients**.

> **Latest Updates – 2 Points (2023–Present)**

11. **Progressive pulmonary fibrosis is now a treatable entity in its own right.** The **2022 ATS/ERS/JRS/ALAT guideline** formally defined **PPF** as at least two of — worsening respiratory symptoms, physiological progression (**absolute FVC decline ≥ 5% or DLCO decline ≥ 10% within one year**), or radiological progression — occurring in a patient with non-IPF fibrotic ILD. On the basis of the **INBUILD** trial, **nintedanib is recommended for PPF regardless of the underlying ILD**, so the modern question at the bedside is no longer only "which ILD?" but **"is this fibrosis progressive?"**
12. **Nerandomilast — the first new antifibrotic class in a decade.** The phase III **FIBRONEER-IPF** and **FIBRONEER-ILD** trials (**NEJM 2025**) showed that **nerandomilast**, an oral **preferential phosphodiesterase-4B (PDE4B) inhibitor**, **significantly reduced the decline in FVC at 52 weeks in both IPF and progressive pulmonary fibrosis**, at both 9 mg and 18 mg twice daily, and **whether given alone or added to background nintedanib or pirfenidone**. Longer follow-up of FIBRONEER-ILD also showed reduced risk of death. It is under regulatory review and represents the first successful new mechanism in pulmonary fibrosis since the antifibrotics.

Current Practice Update

Textbook	ILD discussed only as a radiological differential; corticosteroids implied as generic therapy for interstitial disease.
Current recommendation	Antifibrotics (nintedanib, pirfenidone) are standard in IPF and **steroids are contraindicated** (PANTHER-IPF); nintedanib is licensed for the progressive pulmonary fibrosis phenotype; nerandomilast is emerging.
Exam approach	Write one line — "steroids are harmful in IPF (PANTHER-IPF); antifibrotics are the standard of care, and nintedanib now also treats progressive pulmonary fibrosis of any cause (INBUILD)."

Question 44. Pleural effusion in a 70-year-old — etiopathogenesis, features, differential diagnosis, approach and management

15-Mark Essay | 10 textbook points + 2 latest updates | ***Asked ×2***

Textbook Core – 10 Points

1. **Definition and normal physiology.** Pleural effusion is **abnormal accumulation of fluid in the pleural space**. Normally only **10–20 mL** of fluid is present, produced by the parietal pleural capillaries and absorbed by **parietal pleural lymphatics**. Effusion results when **formation exceeds absorption**. About **250–500 mL** is needed for blunting of the costophrenic angle on an erect chest radiograph, but as little as **50 mL** is detectable on ultrasound or lateral decubitus film.
2. **Pathogenetic mechanisms.** (i) **Increased hydrostatic pressure** — heart failure; (ii) **reduced oncotic pressure** — hypoalbuminaemia, nephrotic syndrome, cirrhosis; (iii) **increased capillary permeability** — infection, malignancy, inflammation; (iv) **impaired lymphatic drainage** — malignant mediastinal node involvement; (v) **movement of fluid from the peritoneum** through diaphragmatic defects — hepatic hydrothorax, Meigs syndrome, pancreatitis; (vi) **thoracic duct rupture** — chylothorax; (vii) **bleeding** — haemothorax.
3. **Causes in a 70-year-old (frequency order matters).**

Transudate	Exudate
Congestive heart failure — the commonest cause overall in the elderly; hypoalbuminaemia; nephrotic syndrome; cirrhosis with hepatic hydrothorax; peritoneal dialysis; constrictive pericarditis; hypothyroidism (may be either); **pulmonary embolism (usually exudate, occasionally transudate)**	**Malignancy** — lung, breast, lymphoma, mesothelioma, ovary, unknown primary; **parapneumonic effusion and empyema**; **tuberculosis — very important in India and often the commonest exudate**; pulmonary embolism/infarction; connective tissue disease (rheumatoid, SLE); pancreatitis; subphrenic or hepatic abscess; drugs (amiodarone, methotrexate, nitrofurantoin); post-cardiac injury (Dressler); asbestos-related benign effusion; chylothorax

In older patients effusions are frequently multifactorial — heart failure plus hypoalbuminaemia plus infection may coexist.

4. **Clinical features. Breathlessness** proportional to size and to underlying lung reserve, **pleuritic chest pain** (parietal pleural involvement, suggesting an exudate), and a **dry cough**. In the frail older patient the presentation may again be **non-specific — fatigue, delirium, anorexia or reduced mobility**. Signs on the affected side: **reduced chest expansion, stony dull percussion note, absent or diminished breath sounds, reduced vocal resonance and tactile fremitus**, with a **bronchial breath sound and aegophony at the upper level**; large effusions cause **tracheal and mediastinal shift away** from the effusion. Always seek clues to the cause — **raised JVP and oedema (heart failure), clubbing and weight loss (malignancy), fever and purulent sputum (empyema), lymphadenopathy, joint disease, and evening rise of temperature with weight loss (tuberculosis)**.

5. **Initial imaging. Erect PA chest radiograph** — homogeneous basal opacity with a **meniscus sign**; a lateral film detects smaller effusions; look for **mediastinal shift, a mass, cardiomegaly or a "white-out"**. **Thoracic ultrasound is now mandatory before any pleural procedure** — it detects small effusions, distinguishes fluid from consolidation and pleural thickening, identifies **septations suggesting empyema or malignancy**, and reduces the risk of pneumothorax and organ injury. **Contrast-enhanced CT thorax** (ideally before complete drainage) is required for suspected malignancy or empyema and shows **nodular, circumferential or mediastinal pleural thickening > 1 cm**, the **"split pleura" sign** of empyema, and any underlying lung lesion.
6. **Diagnostic thoracentesis and Light's criteria. Aspirate all undiagnosed effusions** under ultrasound guidance; a bilateral effusion with clear-cut heart failure may be treated first and tapped only if atypical or non-resolving. Apply **Light's criteria — the fluid is an EXUDATE if any one is met:** (i) **pleural fluid/serum protein ratio > 0.5**; (ii) **pleural fluid/serum LDH ratio > 0.6**; (iii) **pleural fluid LDH > two-thirds of the upper limit of normal serum LDH**. **Light's criteria misclassify about 25% of transudates as exudates in patients already on diuretics** — in that situation use a **serum-to-pleural-fluid albumin gradient > 1.2 g/dL** or a **protein gradient > 3.1 g/dL**, which indicates a transudate.
7. **Pleural fluid analysis — what to send.**

Test	Interpretation
Appearance	Straw (transudate), turbid/purulent (empyema), blood-stained (**malignancy, PE/infarct, trauma, TB**), milky (chylothorax)
Protein, LDH, albumin	Light's criteria and gradients
pH and glucose	**pH < 7.20 or glucose < 60 mg/dL → complicated parapneumonic effusion requiring drainage**; also low in TB, malignancy, rheumatoid and oesophageal rupture
Cell count and differential	**Neutrophils** — parapneumonic, PE, pancreatitis; **lymphocytes** — **tuberculosis, malignancy, lymphoma, chronic effusion**; **eosinophils > 10%** — air or blood in the pleural space, drugs, parasites, asbestos
Adenosine deaminase (ADA)	**> 40 U/L with a lymphocyte-predominant exudate strongly suggests tuberculosis** — the single most useful test in India
Cytology	Positive in about **60%** of malignant effusions on first sample; a second sample raises the yield — send large volume (≥ 50 mL)
Microbiology	Gram stain, culture, **AFB smear, culture and CBNAAT/GeneXpert**; inoculate fluid into blood culture bottles
Others	Amylase (pancreatitis, oesophageal rupture, malignancy), triglycerides > 110 mg/dL (chylothorax), haematocrit > 50% of blood (haemothorax), rheumatoid factor, NT-proBNP

8. **When the tap is non-diagnostic.** About **20–25% of effusions remain undiagnosed after two thoracenteses**. Next steps: **CT-guided cutting-needle pleural biopsy** of thickened pleura (high yield in malignancy and tuberculosis), or **medical (local anaesthetic) thoracoscopy — the investigation of choice, with a diagnostic yield above 90% for malignancy and tuberculosis, and it allows talc poudrage in the same sitting**. Blind Abrams needle biopsy retains a role for suspected tuberculosis where thoracoscopy is unavailable. Consider **bronchoscopy** only if there is haemoptysis or a parenchymal lesion. If all are negative and there is asbestos exposure, keep **mesothelioma** in mind and follow up.
9. **Management by cause. Transudate — treat the underlying disease** (diuretics and guideline-directed therapy for heart failure, albumin/diuretics for cirrhosis); repeated taps are rarely needed. **Parapneumonic effusion** — antibiotics alone if simple; **intercostal chest drain if the fluid is purulent, pH < 7.20, glucose < 60 mg/dL, loculated, or organisms are seen**. **Empyema** — small-bore chest drain plus prolonged antibiotics; if drainage fails, **intrapleural fibrinolytic therapy with alteplase plus DNase (MIST-2 regimen)** improves drainage and reduces surgical referral, with **VATS decortication** for failure. **Tuberculous effusion** — standard **ATT for 6 months**; therapeutic aspiration for symptoms; steroids are not routinely indicated. **Therapeutic aspiration should be limited to about 1.5 litres at one sitting**, stopping earlier for cough or chest discomfort, to avoid **re-expansion pulmonary oedema**.
10. **Malignant pleural effusion and the geriatric approach.** A malignant effusion signifies **advanced disease with a median survival of about 3–12 months** (worst in lung cancer, best in breast cancer and mesothelioma of epithelioid type); prognosis can be estimated by the **LENT score**. Management is **palliative and symptom-driven**: observe if asymptomatic; **therapeutic aspiration** for a frail patient with limited life expectancy; **indwelling pleural catheter (IPC)** — preferred where the lung fails to re-expand (**trapped lung**) or where hospital stay must be minimised; and **chest drain with talc pleurodesis** where the lung re-expands and durable control is wanted. **In the older patient the choice is driven by performance status, life expectancy, home support and patient preference, not by the size of the effusion.** Treat the underlying malignancy where appropriate and involve **palliative care early**.

Latest Updates – 2 Points (2023–Present)

11. **BTS Pleural Disease Guideline 2023 (Thorax).** Key changes relevant to older patients: **thoracic ultrasound must be used for all pleural interventions**; a **contrast CT should be obtained before complete drainage** where malignancy is suspected; **medical thoracoscopy is the preferred investigation for an undiagnosed exudate**; **indwelling pleural catheters and talc pleurodesis are equally acceptable first-line options for malignant effusion**, with the decision made by shared decision-making; and the **RAPID score** (Renal, Age, Purulence, Infection source, Dietary/albumin) is endorsed for risk-stratifying pleural infection, in which **age over 70 is an adverse prognostic factor**.
12. **Intrapleural therapy and IPC strategy have matured.** Following **MIST-2**, combined **intrapleural alteplase plus DNase** is established for pleural infection when drainage fails, and is increasingly used in frail patients who are unfit for surgery. For malignant effusion, the **AMPLE and IPC-PLUS** trials support **indwelling pleural catheters with daily drainage, and talc administered through the catheter** to achieve pleurodesis — an approach that **reduces total hospital days**, which is the outcome that matters most to an older patient with limited life expectancy.

K. Respiratory Failure and Critical Care

Question 45. Respiratory failure, including acute type 2 respiratory failure

*7-Mark Answer | 6 textbook points + 1 latest update | **Asked ×2***

Textbook Core – 6 Points

1. **Definition and classification.** Respiratory failure is failure of the lungs to maintain adequate gas exchange, defined on arterial blood gas as **PaO_2 < 60 mmHg (8 kPa)** breathing room air, with or without **$PaCO_2$ > 50 mmHg (6.7 kPa)**.

	Type 1 (hypoxaemic)	Type 2 (hypercapnic / ventilatory)
Gases	PaO_2 ↓, $PaCO_2$ normal or ↓	PaO_2 ↓, **$PaCO_2$ ↑**
Mechanism	**V/Q mismatch, shunt, diffusion defect**	**Alveolar hypoventilation** (pump failure)
A–a gradient	**Widened**	Normal in pure hypoventilation; widened if lung disease coexists
Examples	Pneumonia, pulmonary oedema, PE, ARDS, ILD	COPD exacerbation, opioid/sedative overdose, neuromuscular disease, kyphoscoliosis, obesity hypoventilation, severe asthma (late)

2. **Acute versus chronic — read the bicarbonate. In acute type 2 failure the pH is low with a normal bicarbonate; in chronic failure the bicarbonate is raised with a near-normal pH (renal compensation); in acute-on-chronic failure the bicarbonate is raised AND the pH is low.** This single distinction determines urgency and is a guaranteed examination mark. Also calculate the **alveolar–arterial oxygen gradient**, which rises normally with age (approximately **age/4 + 4 mmHg**).
3. **Why older people are predisposed.** Ageing produces **loss of elastic recoil with small airway closure and V/Q mismatch, reduced chest wall compliance from kyphosis and rib cage stiffening, reduced respiratory muscle mass and strength, and a lower PaO_2**. Most importantly there is a **marked decline in efferent neural output and a blunted ventilatory response to hypoxia and hypercapnia**, giving a **lower threshold for ventilatory decompensation**. Add **sarcopenia, malnutrition, sedatives and opioids, aspiration and immobility**, and small insults precipitate failure.
4. **Clinical features. Hypoxaemia** — dyspnoea, tachypnoea, restlessness, confusion, cyanosis (a late and unreliable sign), tachycardia, arrhythmia; chronically, **polycythaemia, pulmonary hypertension and cor pulmonale**. **Hypercapnia — headache (especially morning), warm peripheries with a bounding pulse, flushing, drowsiness progressing to coma, flapping tremor (asterixis) and papilloedema. In older adults the earliest and often the only sign is unexplained delirium or drowsiness — always check an arterial blood gas.** Pulse oximetry cannot detect hypercapnia and is misleading on supplemental oxygen.
5. **Investigations. Arterial blood gas is the diagnostic test** (record the FiO_2). Add **chest radiograph, ECG, full blood count, urea and electrolytes, glucose**, and where relevant **CT chest or CTPA, echocardiography, sputum culture, toxicology, thyroid function, creatine kinase and nerve conduction studies** for neuromuscular causes. In chronic disease do **spirometry, overnight oximetry and transcutaneous CO_2 monitoring**. Look actively for **reversible precipitants — infection, pneumothorax, pulmonary embolism, heart failure, sedative drugs, electrolyte disturbance and pain-related hypoventilation**.

6. **Management of acute type 2 respiratory failure.** (i) **Treat the precipitating cause** — bronchodilators, corticosteroids, antibiotics, drainage of pneumothorax, reversal of opioids with naloxone. (ii) **Controlled oxygen therapy targeting SpO_2 88–92%** using a 24–28% Venturi mask — **excessive oxygen worsens hypercapnia through loss of hypoxic drive, the Haldane effect and increased V/Q mismatch, and increases coronary and cerebral vascular resistance**. (iii) **Non-invasive ventilation (BiPAP) when pH < 7.35 with $PaCO_2$ > 6 kPa despite maximal medical therapy** — it **reduces mortality, intubation rate and hospital stay**. (iv) **Repeat the ABG at 1 hour**; failure to improve, pH < 7.25, reduced consciousness or intolerance of the mask indicates **intubation and invasive ventilation** — or, in a frail patient, that **NIV is the agreed ceiling of care**. (v) **Respiratory stimulants (doxapram) are now rarely used.** (vi) Document a **ceiling-of-treatment and resuscitation decision early**, and provide **palliation of breathlessness with opioids and benzodiazepines** where treatment is not succeeding.

Latest Update – 1 Point (2023–Present)

7. **High-flow nasal oxygen (HFNO) has become an established third option between conventional oxygen and NIV.** Delivering **heated, humidified gas at 30–60 L/min with a precise FiO_2**, it generates modest PEEP and washes out anatomical dead space. **ERS/ATS guidance and the GOLD reports** support HFNO in **acute hypoxaemic respiratory failure** (where it reduces intubation compared with conventional oxygen), and it is **safe and effective in exacerbated COPD with both normocapnic and hypercapnic failure**. It is especially valuable in older patients as a **comfort-preserving option where intubation is not appropriate (do-not-intubate orders) or to provide intermediate care outside the intensive care unit** — although **NIV remains first-line for acidotic hypercapnic failure**.

Question 46. Management of ARDS

7-Mark Answer | 6 textbook points + 1 latest update

Textbook Core – 6 Points

1. **Definition and diagnosis.** By the **Berlin definition**, ARDS requires all four of: **onset within one week of a known insult**; **bilateral opacities on chest imaging not fully explained by effusion, collapse or nodules**; **respiratory failure not fully explained by cardiac failure or fluid overload** (objectively assessed, e.g. by echocardiography); and **hypoxaemia graded on PaO_2/FiO_2 with PEEP ≥ 5 cmH_2O — mild 200–300, moderate 100–200, severe ≤ 100**. Pathologically there is **diffuse alveolar damage with non-cardiogenic pulmonary oedema, surfactant loss and hyaline membranes**.
2. **Treat the cause and give supportive care.** Identify and treat the precipitant — **pneumonia and sepsis (commonest), aspiration, pancreatitis, trauma, transfusion (TRALI), drugs, burns**. Give **source control and appropriate antimicrobials**, plus general intensive care: **VTE prophylaxis, stress-ulcer prophylaxis, early enteral nutrition, glycaemic control, sedation minimisation, delirium prevention and early mobilisation**.
3. **Lung-protective ventilation — the single most important intervention. Tidal volume 4–6 mL/kg of PREDICTED body weight, plateau pressure < 30 cmH_2O and driving pressure < 15 cmH_2O** (ARMA/ARDSNet — an absolute mortality reduction of about 9%). Accept **permissive hypercapnia** with pH above 7.20. Use **higher PEEP in moderate–severe ARDS** guided by a PEEP–FiO_2 table, targeting **SpO_2 88–95% or PaO_2 55–80 mmHg**. **Routine recruitment manoeuvres with aggressive PEEP titration are not recommended** (ART trial showed harm).
4. **Prone positioning and neuromuscular blockade. Prone position for at least 16 hours per day** in **moderate-to-severe ARDS with PaO_2/FiO_2 < 150** — the **PROSEVA** trial showed a large mortality reduction. **Neuromuscular blockade (cisatracurium) for 48 hours** may be used in early severe ARDS with ventilator dyssynchrony, but is **not routine** (ROSE trial was neutral) and risks **ICU-acquired weakness, which is particularly disabling in the old**. In older patients, **pressure-area care, eye and airway protection and careful positioning during proning** need explicit attention.
5. **Fluids, pharmacotherapy and rescue. Conservative fluid strategy** once shock has resolved (FACTT) — this shortens ventilator days. **Corticosteroids**: dexamethasone improved outcomes in DEXA-ARDS and are standard in **COVID-19-related ARDS**; use is reasonable in early moderate–severe ARDS but not universal. **No benefit from inhaled nitric oxide (oxygenation improves but mortality does not), surfactant, statins or beta-agonists. Veno-venous ECMO** for refractory severe ARDS in expert centres (EOLIA) — **age above 65–70 with frailty and comorbidity is generally a contraindication**.

6. **Geriatric considerations and outcome.** Older age is an **independent predictor of ARDS mortality**, and survivors face **ICU-acquired weakness, sarcopenia, prolonged weaning, delirium, post-intensive-care cognitive impairment and loss of independence**. Therefore: **establish goals of care and a ceiling of treatment early** with the patient and family; consider whether **NIV or HFNO is the appropriate ceiling**; involve **physiotherapy, nutrition, speech and swallow and geriatric medicine from day one**; plan **early tracheostomy** where prolonged ventilation is expected; and provide **rehabilitation and palliative care alongside organ support**.

Latest Update – 1 Point (2023–Present)

7. **The 2023 New Global Definition of ARDS** (a global consensus conference endorsed by ESICM/ATS) broadened the Berlin definition to make ARDS diagnosable outside well-resourced intensive care units. It **adds a "non-intubated ARDS" category for patients on high-flow nasal oxygen at ≥ 30 L/min or non-invasive ventilation with CPAP ≥ 5 cmH_2O**; permits **SpO_2/FiO_2 ratio ≤ 315 (with SpO_2 ≤ 97%) as an alternative to PaO_2/FiO_2** where blood gases are unavailable; and accepts **lung ultrasound as well as chest radiograph or CT** for the bilateral-opacity criterion. This makes the diagnosis workable in resource-limited settings, including much of India, and in older patients managed outside the ICU.

Question 47. Long-term oxygen therapy and home non-invasive ventilation

7-Mark Answer | 6 textbook points + 1 latest update

Textbook Core – 6 Points

1. **Definition and principle of LTOT.** Long-term oxygen therapy is supplemental oxygen given for **at least 15 hours per day**, which was shown in the **NOTT** and **MRC** trials to **improve survival** in severe COPD with persistent hypoxaemia. Along with **smoking cessation**, it is one of only two interventions proven to prolong life in COPD. It must be prescribed **during a period of clinical stability** and to **current non-smokers**.
2. **Criteria for LTOT.** Assess by arterial blood gas on two occasions at least 3 weeks apart when stable:

Criterion	Threshold
Absolute	**PaO_2 < 7.3 kPa (< 55 mmHg)**
With additional features	**PaO_2 7.3–8.0 kPa (55–60 mmHg)** plus **secondary polycythaemia, pulmonary hypertension, peripheral oedema (cor pulmonale) or nocturnal hypoxaemia**
Screening for referral	SpO_2 **< 93%**, or **< 95%** with possible cor pulmonale

Short-burst oxygen may relieve breathlessness if exercise desaturation is documented.

3. **Benefits and limitations.** LTOT improves **survival** (about **50% of patients survive a further 3 years**), **polycythaemia** and **neuropsychiatric function**; cognitive gains are small and mostly in the oldest groups; **quality-of-life benefit is modest and inconsistent**. **There are no randomised data on LTOT in advanced old age, or in isolated nocturnal or exertional hypoxaemia.** Low BMI and comorbidity predict earlier death; disability, low BMI, lung function and LTOT use are the main mortality predictors in older COPD patients.
4. **Practical and geriatric issues. Fire risk** if the patient or household continues to smoke. The concentrator and long tubing are a **trip and falls hazard** — engineers should fit room-switching mechanisms. **Cognitive impairment and poor manual dexterity** threaten safe use. **Care home logistics** matter: staff training, and portable cylinders or conservers if the day room is far from the bedroom. **Target saturation 88–92% in CO_2 retainers** — excessive oxygen depresses hypoxic drive, increases coronary and cerebral arterial resistance and worsens acidosis.
5. **Acute non-invasive ventilation.** Indicated in **acute hypercapnic respiratory failure with pH < 7.35 despite maximal medical therapy**. Compared with standard treatment, NIV **reduces mortality, the need for intubation and length of hospital stay**. **Bilevel positive airway pressure (BiPAP)** is used. NIV is an appropriate **ceiling of treatment** in frail older patients with a do-not-intubate decision. **High-flow nasal oxygen** is safe and effective in exacerbated COPD with normocapnic or hypercapnic failure and is useful for do-not-intubate patients or intermediate care outside the ICU.
6. **Home (domiciliary) NIV.** Indicated for **chronic hypercapnic respiratory failure** — most importantly **persistent hypercapnia ($PaCO_2$ > 7 kPa) reassessed 2–4 weeks after an exacerbation**, and also in **obesity hypoventilation syndrome, neuromuscular disease and chest-wall deformity (kyphoscoliosis)**. **CPAP — not NIV — is the treatment for obstructive sleep apnoea.** Success requires adequate **cognition, mask tolerance, carer support** and structured follow-up; palliative NIV may relieve symptoms at the end of life.

Latest Update – 1 Point (2023–Present)

7. **GOLD 2025 / 2026 reports** confirm that **LTOT is NOT indicated for isolated moderate resting desaturation or exercise-induced desaturation alone** (based on the **LOTT trial**, which found no benefit in survival or hospitalisation) — oxygen should only be prescribed for **severe resting hypoxaemia**. Conversely, **long-term home NIV should be considered in patients with severe chronic hypercapnia and a history of hospitalisation for acute respiratory failure**, following the **HOT-HMV** trial, which showed that home NIV added to oxygen prolonged the time to readmission or death.

Current Practice Update

Textbook	Short-burst oxygen may help breathlessness with exercise desaturation; home NIV not discussed in detail.
Current recommendation	GOLD — no LTOT for moderate or exercise-only desaturation (LOTT); home NIV recommended for persistent hypercapnia after an exacerbation (HOT-HMV).
Exam approach	Quote the classical PaO_2 thresholds, then add "LOTT: no benefit for moderate desaturation; HOT-HMV: home NIV for persistent hypercapnia."

L. Pulmonary Vascular Disease

Question 48. Pulmonary embolism

7-Mark Answer | 6 textbook points + 1 latest update

Textbook Core – 6 Points

1. **Definition and risk factors.** Pulmonary embolism (PE) is **obstruction of the pulmonary arterial tree, usually by thrombus embolising from a deep vein of the leg or pelvis**. Risk factors follow **Virchow's triad**: **immobility, hospitalisation, stroke with hemiplegia, hip fracture and arthroplasty, malignancy, previous VTE, heart failure, dehydration, sepsis, oestrogens and thrombophilia**. **Advancing age is an independent risk factor**, and PE is both commoner and more often fatal in the old.
2. **Clinical features and the diagnostic trap. Sudden breathlessness, pleuritic chest pain, haemoptysis, tachypnoea, tachycardia, syncope and hypotension.** PE **shares fever, cough, chest pain and dyspnoea with pneumonia**; the clues that favour PE are **clear lung fields on the chest radiograph, a context favouring venous thromboembolism, or clinical signs of deep vein thrombosis**. Conversely, a **pulmonary infarct produces signs shared with pneumonia and is very often secondarily infected**. In older patients PE presents atypically with **unexplained breathlessness, delirium, falls, syncope or new atrial fibrillation**, and is a common missed diagnosis at autopsy.
3. **Risk-stratified diagnostic approach.** First ask whether the patient is **haemodynamically unstable**. If **stable**, apply a validated pre-test probability score (**Wells or revised Geneva**) and, if low/intermediate, measure **D-dimer** — a negative result excludes PE. **D-dimer specificity falls with age, so use the age-adjusted cut-off (age × 10 µg/L in patients over 50)**, which substantially reduces unnecessary imaging in the elderly. If D-dimer is raised or probability is high, perform **CT pulmonary angiography**. Use a **V/Q or V/Q-SPECT scan** when there is renal impairment or contrast allergy. If **unstable**, perform **bedside echocardiography** for right ventricular dilatation and strain and treat immediately.
4. **Supporting investigations. ECG** — sinus tachycardia is commonest; **S1 Q3 T3** is classic but uncommon; also right axis deviation, RBBB and **anterior T-wave inversion (V1–V4)**. **Chest radiograph** usually normal or shows **oligaemia (Westermark), a wedge-shaped peripheral opacity (Hampton hump), raised hemidiaphragm or a small effusion**. **ABG** — hypoxaemia with hypocapnia and a widened A–a gradient. **Troponin and BNP** for right ventricular injury and prognosis. **Compression ultrasound of the legs** supports the diagnosis when imaging is not possible.
5. **Severity stratification. High-risk (massive)** — **persistent hypotension or shock**; mortality > 15%. **Intermediate-risk (submassive)** — normotensive but with **right ventricular dysfunction on imaging and/or raised troponin**; subdivided into intermediate-high (both positive) and intermediate-low. **Low-risk** — **sPESI = 0** (the simplified PESI counts age > 80, cancer, chronic cardiopulmonary disease, heart rate ≥ 110, systolic BP < 100, SpO_2 < 90%), permitting **early discharge and outpatient treatment**.

6. **Treatment.** Start **therapeutic anticoagulation immediately** once PE is suspected with high probability, before confirmation, unless bleeding risk is prohibitive. **DOACs are first-line — apixaban 10 mg twice daily for 7 days then 5 mg twice daily**, or **rivaroxaban 15 mg twice daily for 21 days then 20 mg daily**; dabigatran and edoxaban need **5 days of LMWH lead-in**. Use **LMWH** in severe renal impairment and pregnancy and **warfarin** for antiphospholipid syndrome. For **high-risk PE give systemic thrombolysis (alteplase 100 mg over 2 hours)**; if thrombolysis is contraindicated or fails, use **catheter-directed therapy or surgical embolectomy**. **Routine thrombolysis is not given in intermediate-risk PE** (PEITHO showed reduced decompensation but excess major and intracranial bleeding, especially over 75). **Duration:** provoked by a transient factor **3 months**; unprovoked or persistent risk factor **extended/indefinite**, with reduced-dose apixaban 2.5 mg twice daily or rivaroxaban 10 mg daily thereafter. Reserve an **IVC filter** for absolute contraindication to anticoagulation.

Latest Update – 1 Point (2023–Present)

7. **Catheter-based reperfusion is now supported by randomised evidence.** The **PEERLESS trial (Circulation 2025)** randomised 550 patients with intermediate-risk PE to **large-bore mechanical thrombectomy versus catheter-directed thrombolysis** and found **a significantly better composite outcome with thrombectomy (win ratio 5.01), driven by less clinical deterioration or need for bailout therapy and less post-procedural intensive care use**, with shorter hospital stay and fewer 30-day readmissions. **STORM-PE (2025)** then showed mechanical thrombectomy improved right-ventricular recovery compared with anticoagulation alone. These approaches are attractive in older patients because they **avoid systemic thrombolysis and its intracranial bleeding risk**, but the decision must still be made by a **Pulmonary Embolism Response Team (PERT)**.

Question 49. Chronic pulmonary thromboembolism (CTEPD / CTEPH)

7-Mark Answer | 6 textbook points + 1 latest update

Textbook Core – 6 Points

1. **Definition. Chronic thromboembolic pulmonary disease (CTEPD)** is the persistence of **organised, fibrotic thromboembolic material obstructing the pulmonary arteries with mismatched perfusion defects, persisting after at least 3 months of therapeutic anticoagulation**. When this is accompanied by pulmonary hypertension — **mean pulmonary artery pressure > 20 mmHg with pulmonary vascular resistance > 2 Wood units and a pulmonary artery wedge pressure ≤ 15 mmHg (2022 ESC/ERS haemodynamic definition)** — it is termed **CTEPH**, which is **Group 4 pulmonary hypertension**. CTEPD without PH is now a recognised entity in its own right.
2. **Epidemiology and pathogenesis.** CTEPH develops in about **2–4% of survivors of acute PE**, typically within two years, and **up to 25% have no documented previous PE**. Pathogenesis is **failure of thrombus resolution** with organisation into fibrous webs, bands and stenoses, compounded by a **secondary small-vessel microvasculopathy** in non-obstructed territories that resembles pulmonary arterial hypertension and explains why haemodynamics may not improve after surgery. Risk factors: **large or recurrent PE, unprovoked PE, splenectomy, ventriculo-atrial shunts, infected pacemaker leads, inflammatory bowel disease, chronic osteomyelitis, malignancy, thyroid replacement therapy, non-O blood group and antiphospholipid syndrome**.
3. **Clinical features. Progressive exertional breathlessness and reduced exercise tolerance**, developing after an apparent "honeymoon period" following the acute PE. Later come **fatigue, exertional chest pain, presyncope or syncope and haemoptysis**. Signs of pulmonary hypertension and right heart failure: **loud pulmonary component of the second heart sound, right ventricular heave, raised JVP with prominent v waves, tricuspid regurgitation murmur, hepatomegaly, ascites and peripheral oedema**. A **pulmonary flow murmur over the lung fields is relatively specific**. **In older adults the breathlessness is commonly misattributed to COPD, heart failure, obesity or deconditioning, and diagnosis is typically delayed by more than a year.**
4. **Diagnosis — the single most examinable point.** Screen any patient with **persistent breathlessness ≥ 3 months after PE**. Start with **echocardiography** (raised tricuspid regurgitant velocity, right ventricular dilatation and dysfunction, flattened septum). **The ventilation–perfusion (V/Q) scan is the screening investigation of CHOICE — a normal V/Q scan effectively excludes CTEPH, and CTPA may miss chronic organised disease.** Confirmatory imaging is by **CT pulmonary angiography (webs, bands, eccentric wall-adherent thrombus, abrupt vessel cut-off, calcified thrombus, mosaic perfusion, bronchial collaterals) and digital subtraction pulmonary angiography**. **Right heart catheterisation is mandatory** to confirm haemodynamics and operability. Add **lung function tests, ABG, six-minute walk test, NT-proBNP and a thrombophilia/antiphospholipid screen**.

5. **Management — pulmonary endarterectomy is the treatment of choice.** Every patient must be **referred to an expert CTEPH centre** for multidisciplinary assessment of operability. **Pulmonary endarterectomy (PEA)** under deep hypothermic circulatory arrest is **potentially curative** for proximal, surgically accessible disease and is the first-line treatment; in experienced centres mortality is under 5%, and **age alone is not a contraindication** though frailty and comorbidity raise operative risk. All patients need **lifelong therapeutic anticoagulation** — **vitamin K antagonists remain preferred, and DOACs are used with caution and are contraindicated in antiphospholipid syndrome**.
6. **Inoperable and residual disease.** For patients who are **technically inoperable (distal disease), unfit for surgery, or who have persistent/recurrent pulmonary hypertension after PEA** — a group heavily weighted towards older patients — the options are: **balloon pulmonary angioplasty (BPA)**, now an established treatment that improves haemodynamics, right ventricular function, exercise capacity and quality of life; and **medical therapy with riociguat**, a **soluble guanylate cyclase stimulator** and the **only licensed drug for CTEPH** (CHEST-1), with **subcutaneous treprostinil** for severe disease. Add **diuretics for right heart failure, supplemental oxygen, supervised rehabilitation, vaccination and pregnancy avoidance**; consider **lung transplantation** in the young with refractory disease. **Calcium channel blockers and vasodilator testing have no role in CTEPH.**

Latest Update – 1 Point (2023–Present)

7. **A multimodal, sequenced strategy is now the standard of care.** The **2023 ESC Working Group clinical consensus statement on balloon pulmonary angioplasty** establishes BPA as a **complementary — not competing — treatment alongside PEA and riociguat**, recommends **medical pre-treatment with riociguat before BPA** (the **RACE** trial showed fewer procedure-related complications with riociguat pre-treatment), and endorses BPA in **CTEPD both with and without pulmonary hypertension**. Importantly, the phase III **MACiTEPH trial (2025) of macitentan was stopped for futility** with no improvement in six-minute walk distance, so **riociguat remains the only drug with proven benefit** — a useful negative result to quote.

M. Thoracic Oncology

Question 50. Bronchogenic carcinoma — types and management

7-Mark Answer | 6 textbook points + 1 latest update

Source note: Bronchogenic carcinoma is **not covered adequately as a standalone topic in the uploaded textbook** — Pathy's refers to lung cancer chiefly in the context of paraneoplastic syndromes, therapy-related myelodysplasia and geriatric oncology principles. The answer below is built on current standard oncology guidance and is labelled accordingly.

Textbook Core – 6 Points

1. **Definition and epidemiology.** Primary malignancy arising from the **bronchial epithelium**; the **commonest cause of cancer death worldwide**. The **median age at diagnosis is about 70**, making it essentially a disease of older adults. **Tobacco smoking** is the dominant risk factor; in India also important are **biomass fuel smoke and indoor air pollution** (notably in women who have never smoked), **asbestos, radon, arsenic, chromium** and **scarring from prior pulmonary tuberculosis**.
2. **Classification.**

Type	Share	Key features
Adenocarcinoma	~40%	**Commonest overall and in never-smokers and women**; **peripheral**; carries the actionable driver mutations; hypertrophic pulmonary osteoarthropathy
Squamous cell	~25–30%	**Central**, **cavitation**, **hypercalcaemia via PTHrP**, strongly smoking-related
Large cell	~10%	Undifferentiated, poor prognosis
Small cell (SCLC)	~15%	**Central**, rapidly growing, **early widespread metastasis**, almost always smokers; **SIADH**, **ectopic ACTH/Cushing**, **Lambert–Eaton myasthenic syndrome**

   The first three are grouped as **non-small cell lung cancer (NSCLC), about 85%**.
3. **Clinical features. Cough, haemoptysis, breathlessness, chest pain, weight loss** and recurrent or non-resolving pneumonia. **Older adults present late and atypically** — with fatigue, anorexia, delirium, falls or functional decline rather than respiratory symptoms, and smoker's cough is often dismissed. **Local invasion:** hoarseness (recurrent laryngeal nerve), **superior vena cava obstruction**, **Pancoast tumour with Horner's syndrome** and T1 root pain, phrenic nerve palsy, dysphagia, pericardial effusion.

4. **Diagnosis and staging. Chest radiograph → contrast CT chest and upper abdomen → tissue diagnosis**. Central lesions: **bronchoscopy with EBUS-guided transbronchial needle aspiration**; peripheral lesions: **CT-guided percutaneous biopsy**. Stage with **PET-CT** and **MRI brain**, using the **TNM 9th edition (2024)**. In NSCLC always test for **EGFR, ALK, ROS1, BRAF, KRAS G12C, RET, MET** alterations and **PD-L1 expression**, since these determine treatment. Assess **lung function and performance status** before any radical treatment.
5. **Management of NSCLC. Stage I–II: surgical resection (lobectomy with lymph node dissection)**; **sublobar resection or stereotactic body radiotherapy (SBRT)** for the frail or those with poor lung function — SBRT is a major advance for older patients. Adjuvant chemotherapy for stage II–III; adjuvant osimertinib for resected EGFR-mutant disease. **Stage III (unresectable): concurrent chemoradiotherapy followed by consolidation durvalumab**. **Stage IV: targeted therapy if a driver mutation is present** (osimertinib for EGFR, alectinib for ALK), otherwise **immunotherapy (pembrolizumab) alone if PD-L1 ≥ 50%, or combined with a platinum doublet**.
6. **Management of SCLC and geriatric principles. Limited stage:** platinum plus etoposide with **concurrent thoracic radiotherapy**, and **prophylactic cranial irradiation** in responders. **Extensive stage:** platinum–etoposide plus **atezolizumab or durvalumab**. **Treatment decisions must be based on comprehensive geriatric assessment and performance status, not chronological age** — but frailty, renal function, hearing (cisplatin ototoxicity), neuropathy and social support guide dose reduction, use of **carboplatin instead of cisplatin**, or single-agent therapy. **Early integrated palliative care improves quality of life and survival.** Symptom control: **SVC stenting**, palliative radiotherapy for haemoptysis and bone pain, opioids for breathlessness, and **smoking cessation at any age**.

Latest Update – 1 Point (2023–Present)

7. Two 2024 practice-changing trials: **ADRIATIC (NEJM 2024) — consolidation durvalumab after concurrent chemoradiotherapy significantly improved overall survival in limited-stage small cell lung cancer**, the first major advance in this setting for decades; and **LAURA (NEJM 2024) — osimertinib after chemoradiotherapy markedly prolonged progression-free survival in unresectable stage III EGFR-mutant NSCLC**. In relapsed SCLC, **tarlatamab**, a DLL3-targeted bispecific T-cell engager (DeLLphi-301/304), has been approved and improved survival over chemotherapy in the second line.

Question 51. Case — solitary pulmonary nodule on chest X-ray in a 30 pack-year smoker with cough

7-Mark Answer | 6 textbook points + 1 latest update

Textbook Core – 6 Points

1. **Definition and first principle.** A solitary pulmonary nodule (SPN) is a **single, rounded opacity up to 3 cm in diameter, completely surrounded by aerated lung, with no associated atelectasis, hilar enlargement or pleural effusion**. **A lesion larger than 3 cm is a mass and should be regarded as malignant until proved otherwise.** In a 67-year-old-type patient with a **30 pack-year smoking history**, the pre-test probability of malignancy is high — **this nodule must be actively worked up, not simply observed**.
2. **The immediate next steps.** (i) **Obtain and compare all previous chest radiographs or CT scans — documented stability of a solid nodule for 2 years or more effectively excludes malignancy**, and this simple step can end the investigation. (ii) The **next investigative procedure of choice is a contrast-enhanced thin-section (1 mm) CT of the chest and upper abdomen**, which characterises the nodule, detects additional nodules, and stages the mediastinum, adrenals and liver. (iii) Take a full history for **weight loss, haemoptysis, prior malignancy, tuberculosis, occupational asbestos and biomass exposure, and immune status**, and examine for clubbing, lymphadenopathy and extrathoracic disease.

3. **Reading the CT — features favouring benign versus malignant.**

Feature	Benign	Malignant
Size	< 8 mm	**> 8 mm, and risk rises steeply above 20 mm**
Margin	Smooth, well defined	**Spiculated, lobulated, "corona radiata"**
Calcification	**Central, diffuse, laminated or "popcorn" (hamartoma)**	Eccentric or stippled
Fat within the lesion	**Diagnostic of hamartoma**	Absent
Growth	**Stable ≥ 2 years (solid nodules)**	Volume doubling time 20–400 days
Site and type	Lower zone, solid	**Upper lobe; part-solid nodules carry the highest malignant risk**

Formal risk can be estimated with the **Brock (PanCan) or Mayo Clinic model**, which combine **age, smoking, prior cancer, nodule size, upper-lobe location, spiculation and nodule count**. Common benign causes: **tuberculoma and other granulomas (very common in India), hamartoma, resolving pneumonia, round atelectasis, arteriovenous malformation, rheumatoid nodule, hydatid cyst.**

4. **Stratified management by size and risk (Fleischner-type approach).**

Solid nodule < 6 mm, low risk → no routine follow-up
Solid nodule < 6 mm, high risk (smoker) → optional CT at 12 months
Solid nodule 6–8 mm → CT at 6–12 months, then 18–24 months
Solid nodule > 8 mm → **PET-CT and/or tissue diagnosis**
Part-solid nodule with solid component ≥ 6 mm → **treat as likely malignant → PET-CT / biopsy**
Pure ground-glass nodule → longer-interval surveillance (indolent adenocarcinoma spectrum)

PET-CT is most useful for solid nodules above 8–10 mm; note **false positives in tuberculosis, sarcoidosis and other granulomatous infection — a major limitation in India — and false negatives in lesions under 8 mm, carcinoid and ground-glass adenocarcinoma**.

5. **Obtaining tissue.** Choose the route by nodule location: **CT-guided percutaneous transthoracic needle biopsy** for peripheral nodules (high yield; risk of pneumothorax around 20–25%, higher with emphysema); **bronchoscopy with EBUS-guided or radial-probe/navigational sampling** for central or bronchus-sign-positive lesions, which also allows **EBUS-TBNA mediastinal staging**; and **VATS wedge excision**, which is both diagnostic and therapeutic when the probability of malignancy is high. **In a high-risk nodule, a negative needle biopsy does not exclude cancer — proceed to surgical excision or close surveillance.** Send tissue for **histology, AFB smear/culture and CBNAAT** in the Indian setting.

6. **Definitive management and follow-up.** If **benign** (fat or benign calcification, or 2-year stability) — reassure, treat any infective cause, and continue smoking cessation. If **malignant or highly suspicious** — complete **staging with PET-CT and MRI brain**, assess **fitness with pulmonary function tests, performance status and comprehensive geriatric assessment**, and discuss at a **multidisciplinary tumour board**. **Lobectomy with lymph node dissection** is standard for a fit patient with early-stage disease; **sublobar resection or stereotactic body radiotherapy (SBRT)** is used for those with poor lung function or frailty, and gives excellent local control with low morbidity in older adults. Test for **EGFR, ALK, ROS1 and PD-L1** if advanced. In all cases: **smoking cessation at any age, vaccination, and enrolment in a lung cancer screening programme if eligible**.

Latest Update – 1 Point (2023–Present)

7. **Lung cancer screening eligibility has widened and nodule management has been standardised.** The **2023 American Cancer Society guideline** recommends **annual low-dose CT for adults aged 50–80 with a ≥ 20 pack-year smoking history, and — importantly — has removed the "years since quitting" restriction**, so former smokers remain eligible indefinitely; this patient's 30 pack-year history makes him screening-eligible. Nodules detected in a screening programme are now categorised by **Lung-RADS version 2022**, which standardises follow-up intervals and formally incorporates **atypical pulmonary cysts and juxtapleural nodules**. **Semi-automated volumetry with volume-doubling time, increasingly AI-assisted**, is replacing two-dimensional diameter measurement for detecting growth.

N. Sleep Medicine

Question 52. Obstructive sleep apnoea — features, comorbidities, tests, management and prognosis

7-Mark Answer | 6 textbook points + 1 latest update

Textbook Core – 6 Points

1. **Definition and epidemiology.** Obstructive sleep apnoea (OSA) is the **most frequent respiratory sleep disorder** and involves **repeated episodes of complete (apnoea) or partial (hypopnoea) upper airway obstruction during sleep**. It is defined by an **apnoea–hypopnoea index (AHI) > 5 per hour of sleep together with excessive daytime sleepiness or neurocognitive/behavioural disturbance not attributable to another cause**. Prevalence is **1–4% of general adults but far higher in the elderly — about 50% of older people have an AHI > 5 and 20% have an AHI > 15**, compared with 10% of younger people.
2. **Pathogenesis in the older patient.** The dominant mechanism is **increased upper airway collapsibility with age**, due to **hormonal change (after the menopause the prevalence in women equalises with men)**, **reduced pharyngeal dilator muscle tone and altered pharyngeal reflexes with increased airway resistance during sleep**, and anatomical factors including **edentulism and increased fat deposition in the pharyngeal wall**. Comorbidities that accumulate with age — **stroke, heart failure, hypothyroidism, obesity** — further promote sleep-disordered breathing.
3. **Clinical features and the age-related shift.** The classical triad is **snoring, witnessed apnoeas and excessive daytime sleepiness**. **However, the proportion of OSA patients who do NOT snore increases after the age of 50**, so the classical triad is unreliable in the old. Instead suspect OSA from **unrefreshing sleep, nocturia, morning headache, fatigue rather than frank sleepiness, poor concentration, memory impairment, irritability or depression, falls and road traffic accidents**. Examination: **obesity and increased neck circumference, crowded oropharynx (high Mallampati), retrognathia, nasal obstruction, hypertension**.
4. **Associated comorbidities. Cardiovascular — resistant hypertension (especially non-dipping and nocturnal), atrial fibrillation and other arrhythmias, ischaemic heart disease, heart failure, pulmonary hypertension** and **stroke**. **Metabolic** — obesity, insulin resistance and type 2 diabetes, metabolic syndrome. **Neurocognitive — cognitive impairment and dementia, depression, delirium**; nocturnal hypoxia carries risks of arrhythmia and of myocardial and cerebral infarction. **Other** — nocturia, glaucoma, reduced quality of life, and increased perioperative and sedative-related risk.
5. **Investigations.** Begin with **history from the patient and the bed partner**, the **Epworth Sleepiness Scale** and a screening tool such as **STOP-BANG**. Confirm with **polysomnography**, which remains the gold standard; a **home sleep apnoea test** is acceptable in high-probability uncomplicated patients. Severity by AHI: **mild 5–15, moderate 15–30, severe > 30**. AASM scoring should **include arousal-based scoring of hypopnoeas**. Add **overnight oximetry, thyroid function, ECG, echocardiography and ABG** where obesity hypoventilation or cor pulmonale is suspected.
6. **Management and prognosis. Diet and sleep-hygiene measures**, **weight reduction**, avoidance of **alcohol, sedatives and opioids**, **positional therapy**, treatment of nasal obstruction, and **optimisation of dentition**. **CPAP is the treatment of choice** and improves sleepiness, quality of life, blood pressure and cognition; adherence in older adults is comparable to younger patients when support is given. Alternatives: **mandibular advancement devices** for mild–moderate disease or CPAP intolerance, and **surgery (uvulopalatopharyngoplasty, hypoglossal nerve stimulation)** in selected cases. Prognosis: untreated moderate-to-severe OSA increases **cardiovascular events, stroke, cognitive decline, accidents and mortality**; treatment reliably improves symptoms, though large trials (SAVE) did not show a reduction in cardiovascular events, largely because of poor CPAP adherence.

Latest Update – 1 Point (2023–Present)

7. **SURMOUNT-OSA (NEJM 2024)** — in adults with **obesity and moderate-to-severe OSA**, **tirzepatide** (a dual GIP/GLP-1 receptor agonist) produced a **large reduction in the apnoea–hypopnoea index (roughly 25–29 fewer events per hour versus placebo) with improvements in oxygen saturation, blood pressure and sleep-related quality of life**, both with and without background CPAP. This led to the **first regulatory approval of a drug for obstructive sleep apnoea (December 2024)** and establishes **pharmacological weight loss as a disease-modifying treatment for OSA** rather than merely an adjunct — though in older adults the risks of **sarcopenia, weight loss and gastrointestinal effects** require careful weighing.

Question 53. Sleep problems in the elderly, and the approach to insomnia in a 65-year-old recently retired professional

7-Mark Answer | 6 textbook points + 1 latest update | ***Asked ×2***

Textbook Core – 6 Points

1. **Burden and normal ageing changes. More than half of people over 65 report a sleep-related problem.** Normal ageing produces a **shorter, more superficial and more fragmented total sleep time**, with **increased sleep latency, reduced slow-wave (N3) and REM sleep, and more awakenings**; **50–80% of older adults nap in the daytime**, which itself indicates that night sleep is unrefreshing. **Insomnia is the commonest sleep disorder in older people, with a prevalence of 40–50% over the age of 65.**
2. **The spectrum of sleep disorders in old age.**

Disorder	Key feature and treatment
Insomnia	Difficulty initiating or maintaining sleep — **CBT-I first; medication only for acute insomnia**
Obstructive sleep apnoea	Snoring, witnessed apnoeas, daytime sleepiness — **polysomnography, CPAP**; affects ~20% of older adults
Restless legs syndrome	Motor restlessness, night-time pacing; **commoner with iron deficiency and CKD** — check ferritin
Periodic limb movement disorder	Leg kicking with arousals — diagnosed on polysomnography
REM sleep behaviour disorder	Dream enactment; associated with **Parkinson's disease and dementia**
Advanced sleep phase	Falling asleep and waking too early — **common in institutionalised elderly**; bright light therapy

3. **Secondary causes — always look for these first. Medical** — pain from arthritis, **orthopnoea and paroxysmal nocturnal dyspnoea**, cough, **nocturia and prostatism**, **gastro-oesophageal reflux** (worsened by recumbency), heart failure, COPD, Parkinson's disease, dementia. **Psychiatric — depression (insomnia is a cardinal symptom), anxiety, bereavement**. **Drugs** — diuretics, corticosteroids, certain antidepressants, theophylline, beta-blockers, **caffeine and late alcohol (which fragments sleep)**, and **hypnotic withdrawal**. **Behavioural — daytime naps over 30 minutes, an earlier bedtime, and increased time in bed not sleeping**.
4. **Assessment. For most of these disorders a detailed history is sufficient.** Characterise the complaint precisely — **delayed sleep onset, frequent awakenings or early-morning waking** — and take a **collateral history from the bed partner** (snoring, apnoeas, kicking, dream enactment). Use a **2-week sleep diary, with actigraphy where available**. Screen for **depression, cognition, alcohol and the full drug chart**, and examine for causes. Reserve **polysomnography** for suspected OSA, periodic limb movement disorder or REM sleep behaviour disorder — it is not needed for uncomplicated insomnia. Address **unrealistic sleep expectations** (such as the belief that 8 unbroken hours are essential).
5. **The retired 65-year-old — the specific approach.** Retirement from a busy professional career removes the **external time cues (zeitgebers) that anchored the circadian rhythm** — fixed waking time, morning light exposure, daytime activity and social contact. The typical sequence is **later and irregular rising, long daytime naps, reduced physical activity and light exposure, an earlier bedtime, excessive time in bed, loss of role and purpose with low mood, and increased evening alcohol**. Management is therefore **behavioural, not pharmacological**: (i) **fixed wake-up time seven days a week**; (ii) **bright morning light exposure and outdoor activity**; (iii) **regular daytime exercise, avoided within 3 hours of bedtime**; (iv) **abolish or limit naps to under 30 minutes before 3 pm**; (v) **restrict time in bed to actual sleep time (sleep restriction) and use the bed only for sleep and sex (stimulus control)**; (vi) **build a new structure of daily purpose and social engagement**; (vii) **limit caffeine after midday and evening alcohol and fluids**; (viii) **screen for and treat depression**.
6. **Treatment principles and drug safety. Sleep hygiene measures and cognitive behavioural therapy for insomnia (CBT-I) are first-line and should be tried BEFORE any pharmacological treatment**; brief cognitive therapy is effective and avoids the long-term side effects of drugs. If a drug is unavoidable, use it for **acute insomnia only, at the lowest dose and for the shortest time, with a planned stop date**. **Benzodiazepines and Z-drugs (zolpidem, zopiclone) should be avoided in older adults** — they are on the **AGS Beers list** and cause **falls, hip fracture, cognitive impairment, delirium, daytime sedation, road accidents, tolerance and dependence**. Also **avoid sedating antihistamines and tricyclics because of anticholinergic burden**. Safer options where indicated: **low-dose melatonin or prolonged-release melatonin**, **very low-dose doxepin**, and treatment of the underlying cause. Always **deprescribe long-term hypnotics gradually with behavioural support**.

Latest Update – 1 Point (2023–Present)

7. **Dual orexin receptor antagonists (DORAs) have changed the pharmacological hierarchy. Lemborexant, daridorexant and suvorexant** promote sleep by **blocking wake-promoting orexin signalling rather than by non-selective GABA-A sedation**, and randomised data in adults aged 65 and over show **improved sleep onset and maintenance with improved daytime functioning and, importantly, no worsening of postural stability or next-morning falls risk compared with Z-drugs**. Current guidance (AASM and the **AGS Beers Criteria 2023**, which continue to advise against benzodiazepines and Z-drugs in older adults) positions DORAs as the **preferred pharmacological option when CBT-I has failed** — although CBT-I remains first-line and drug treatment should still be short-term.

Current Practice Update

Textbook	Sleep hygiene and CBT first; medication for acute insomnia only, with hypnotics implied as the drug class.
Current recommendation	Benzodiazepines and Z-drugs are explicitly avoided (Beers 2023); dual orexin receptor antagonists or low-dose melatonin/doxepin are preferred when a drug is needed.
Exam approach	Write "CBT-I is first-line; if a drug is required, avoid benzodiazepines and Z-drugs (Beers 2023) and prefer a DORA or low-dose melatonin."

Question 54. REM sleep behaviour disorder

7-Mark Answer | 6 textbook points + 1 latest update

Textbook Core – 6 Points

1. **Definition.** REM sleep behaviour disorder (RBD) is a **parasomnia characterised by the apparent acting out of dreams**, caused by **loss of the normal muscle atonia of REM sleep**. Normal REM sleep has both **tonic phenomena (muscle atonia) and phasic phenomena (brief muscle twitches)**; in RBD the brainstem circuits that generate atonia (the sublaterodorsal nucleus and its projections to the ventral medulla) fail, so **dream content is enacted**.
2. **Clinical features.** Vivid, often **frightening or violent dreams** of being attacked or chased, enacted as **shouting, swearing, punching, kicking, leaping from bed**. Episodes occur in the **second half of the night when REM is most abundant**. The patient **wakes rapidly, is fully oriented and can recall the dream** — which distinguishes RBD from confusional arousals and night terrors. There is a **male predominance and a typical onset after 50**. **Injury to the patient or to the bed partner is common** and is often the reason for presentation; the collateral history from the bed partner is essential.
3. **Causes and associations. RBD is mostly idiopathic, but has a strong association with Parkinson's disease and dementia** — it is now regarded as a **prodromal α-synucleinopathy**. It is seen in **Parkinson's disease, dementia with Lewy bodies (where it is a core clinical feature) and multiple system atrophy**. Secondary causes: **antidepressants — SSRIs, SNRIs, tricyclics and mirtazapine (a very common and reversible cause)**, beta-blockers, alcohol withdrawal, **narcolepsy**, and brainstem lesions from stroke, tumour or demyelination.
4. **Prognostic significance (the most examinable point).** Prospective cohorts show that **idiopathic RBD converts to an overt neurodegenerative synucleinopathy at a rate of roughly 6% per year**, so that **more than 70–80% phenoconvert to Parkinson's disease, dementia with Lewy bodies or multiple system atrophy within 12–15 years**. Post-mortem studies of idiopathic RBD confirm underlying synuclein pathology. Markers of earlier conversion include **abnormal olfaction, colour vision impairment, subtle motor signs, mild cognitive impairment, constipation, orthostatic hypotension and abnormal dopamine transporter imaging**. **These patients should therefore be followed longitudinally with counselling appropriate to their wishes.**
5. **Diagnosis.** Requires **history plus polysomnography**. Video-polysomnography demonstrates **REM sleep without atonia (RSWA) — excessive tonic or phasic chin/limb EMG activity during REM** — with recorded dream-enacting behaviour, and is the diagnostic gold standard; screening questionnaires (RBD-SQ, RBD1Q) support but do not replace it. Polysomnography also **excludes the important mimics**: **obstructive sleep apnoea with pseudo-RBD, nocturnal frontal lobe epilepsy, periodic limb movement disorder, non-REM parasomnias (sleepwalking, night terrors), nocturnal delirium and severe restless legs**. Review the drug chart for antidepressants, and image the brainstem if there are focal neurological signs.
6. **Management. Environmental safety comes FIRST and is the single most important intervention — remove sharp objects, firearms and bedside furniture, pad or lower the bed or place the mattress on the floor, cushion the floor, use bed rails cautiously, place a barrier or move the bed partner to a separate room**. Then **withdraw or substitute the culprit antidepressant** where possible, and treat coexisting OSA. Pharmacologically, the textbook agent is **clonazepam 0.25–1 mg at night**, which is highly effective, but in older people it causes **sedation, falls, cognitive impairment and worsening of sleep apnoea**; **melatonin 3–12 mg at night is therefore preferred first-line in the elderly**, being safer and comparably effective. **Rivastigmine** may help when RBD occurs with dementia with Lewy bodies. Counsel and support the **bed partner**, and explain the neurodegenerative association sensitively and only as much as the patient wishes to know.

Latest Update – 1 Point (2023–Present)

7. **RBD is now formally embedded in the biological staging of Parkinson's disease.** The **2024 Neuronal Synuclein Disease Integrated Staging System (NSD-ISS)** and the parallel **SynNeurGe** framework redefine synucleinopathy biologically rather than clinically, using **seed amplification assays (α-synuclein SAA) on CSF or skin, which are positive in over 90% of patients with isolated RBD**. Isolated RBD with a positive assay is classified as **prodromal neuronal synuclein disease (Stage 2)** — i.e. **biologically established Parkinson's/Lewy body disease before motor or cognitive signs appear**. Consequently, **isolated RBD cohorts are now the principal recruitment population for disease-modifying neuroprotection trials**, which makes accurate diagnosis and referral of these patients increasingly important.

Appendix — Rapid Revision Grid

Consolidated examinable numbers, named trials and Indian context lines from all 54 questions

A. Numbers worth memorising

Topic	Number
Heart failure	HFrEF **≤ 40%**, HFmrEF **41–49%**, HFpEF **≥ 50%**; **> 75%** of overt HF in the old is due to hypertension or coronary disease; spironolactone **12.5–50 mg** reduced mortality by **30%** in NYHA III–IV; digoxin over 70 — **0.125 mg/day**, level **0.5–0.9 ng/mL**
Refractory heart failure	Stage D one-year mortality approaches **50%**; acceptable creatinine rise during decongestion **up to 30%**
Beta blockers	Only **4** agents proven: **bisoprolol, carvedilol, metoprolol succinate, nebivolol**; double dose no sooner than every **2 weeks**; SENIORS studied age **≥ 70**
Cardiogenic shock	Systolic BP **< 90 mmHg for > 30 min** with hypoperfusion; MAP target **≥ 65 mmHg**; fluid in **250 mL** aliquots
ACS / unstable angina	ECG within **10 min**; troponin at **0 and 1–3 h**; immediate invasive **< 2 h**, high risk **< 24 h**, GRACE **> 140**; prasugrel contraindicated **> 75 y, < 60 kg**; DAPT **12 months** (1–3 if HBR)
STEMI ECG criteria	**≥ 1 mm** in 2 contiguous leads; **≥ 2 mm** in V2–V3 men ≥ 40, **≥ 2.5 mm** men < 40, **≥ 1.5 mm** women; PCI within **120 min** of diagnosis
Secondary prevention targets	LDL **< 55 mg/dL** with **≥ 50%** reduction; BP **< 130/80**; HbA1c **~7%**; weight loss **5–10%**; exercise **150 min/week**
JNC 8	Age **≥ 60** — treat at **≥ 150/90**, goal **< 150/90**; < 60, diabetes and CKD — **< 140/90**; beta-blockers **not** first-line
Hypertension classification	Stage 1 **140–159/90–99**; Stage 2 **≥ 160/100**; ISH **≥ 140 systolic with < 90 diastolic**; 2025 AHA/ACC target **< 130/80**
Accelerated hypertension	Usually diastolic **> 120–130**; grade **III** retinopathy (accelerated) vs grade **IV** papilloedema (malignant); reduce MAP by **≤ 20–25%** in the first hour; dissection — systolic **< 120** and HR **< 60** within 20 min
Postural hypotension	Fall of **≥ 20 mmHg systolic or ≥ 10 mmHg diastolic within 3 min**; present in **~30%** of elderly (> 40 mmHg in **10%**); fludrocortisone **100–200 µg**; ephedrine **15–30 mg tds**; head-up tilt of bed **~10°**
Aortic stenosis	Prevalence **2.8%** at ≥ 75; sclerosis in **26–29%** over 65; symptomatic AS untreated — **20% three-year survival**; severe = area **< 1.0 cm²**, velocity **≥ 4 m/s**, mean gradient **≥ 40 mmHg**; octogenarian SAVR five-year survival **~65%**; valvuloplasty — mortality **3–10%**, stroke **10–25%**, restenosis **66%** at 6 months
Mitral disease	Mucoid degeneration in **~1%** of autopsies > 65; mitral annular calcification in **9%** of autopsies > 50, and **44%** of women > 90; **two-thirds** of MAC present with an apical systolic murmur; surgery when EF **≤ 60%** or ESD **≥ 40 mm**
ABI and toe pressures	ABI **≤ 0.90** = PAD; **< 0.40** = severe; **> 1.40** = non-compressible; exercise fall **> 20%**; **TBI < 0.70** = PAD; toe pressure **> 55** heals, **< 30 mmHg** = CLTI
Atrial fibrillation	**43 million** worldwide; lifetime risk at 55 **~35%**; **1.5×** commoner in men; **up to 30%** of ischaemic strokes; **20–30%** develop heart failure; **up to 60%** relative increase in cognitive decline; **3.5-fold** increase in mortality; each **10 mmHg** systolic rise increases AF risk **11–16%**; rate target **< 110** lenient
Tachyarrhythmias	Rate **> 100**/min; QRS **≥ 120 ms** = broad; ectopy in **up to 100%** of older people; ventricular arrhythmia in **70–80%** over 60; adenosine **6 then 12 mg**; amiodarone **300 mg** then **900 mg/24 h**; magnesium **2 g**; post-MI ectopy **> 10/hour** is adverse
Tilt test	Tilt to **60–70°** for **20–45 min**; GTN **400 µg**; cardioinhibitory type 2B = **asystole > 3 s**; sensitivity **~50–70%**, specificity **~80–90%**
Polypill	Wald & Law 2003 — **88%** reduction in IHD, **80%** in stroke (theoretical); aspirin **75–100 mg**
Cardiorenal syndrome	**5 Ronco types**; IV diuretic at **1–2.5×** the home oral dose; creatinine rise **up to 30%** acceptable; acetazolamide **500 mg** IV daily
Infective endocarditis in the elderly	Incidence **4×**, mortality **2×** that of younger patients; antibiotics **4–6 weeks**; **3** sets of blood cultures
Complete heart block	Ventricular escape **30–40 bpm**; PR > 200 ms = first degree; PR > 240 ms + LBBB with lamin A/C = pace
Pacemaker complications	Pneumothorax **1–2%** (subclavian) vs **<0.1%** (cephalic/axillary); tamponade **<0.2%**; infection **<1%**; early complications **~7%** at 2 weeks; CRT/His if EF < 50% and pacing > 40%
CHA_2DS_2-VASc	Max **9**; anticoagulate men ≥ 2, women ≥ 3; **CHA_2DS_2-VA max 7**, threshold ≥ 2 (2024 ESC)
HAS-BLED	Max **9**; **≥ 3 = high risk** (modify, do not withhold); falls — need **~295 falls/year** to outweigh benefit
Apixaban dose reduction	**2 of 3**: age ≥ 80, weight ≤ 60 kg, creatinine ≥ 1.5 mg/dL → 2.5 mg BD
DOAC reversal	Idarucizumab **5 g IV**; 4F-PCC **50 U/kg**; charcoal if < 2–4 h; dabigatran **dialysable**
Stents	BMS restenosis **20–30%**; DES TLR **< 5%**; ISR = **≥ 50%** renarrowing; DAPT **6 months** elective, **12 months** post-ACS,

	1–3 months if HBR
Statins in the old	Time to benefit **~2.5 years**; NLA/AGS 2025 LDL-C **70–189 mg/dL**; prasugrel contraindicated **> 75 y, < 60 kg**
DVT	D-dimer age-adjusted cut-off = **age × 10 µg/L** if > 50; enoxaparin **40 mg OD**; extended prophylaxis **28–35 days** post hip surgery; provoked = **3 months**; PTS up to **40%**
LTOT	**≥ 15 h/day**; PaO_2 **< 7.3 kPa (55 mmHg)**, or **7.3–8.0 kPa** with cor pulmonale/polycythaemia; refer if SpO_2 **< 93%**; target SpO_2 **88–92%**; **~50% survive 3 years**
NIV	Acute NIV if **pH < 7.35**; home NIV if **$PaCO_2$ > 7 kPa** at 2–4 weeks post-exacerbation
Lung cancer	NSCLC **~85%**, SCLC **~15%**; median age at diagnosis **~70**; TNM **9th edition (2024)**
Inhalers	Difficulty in **~20%**; **MMSE < 24** predicts failure; breath-hold **10 s**; inhale over **4–5 s**; wait **30 s** between puffs
Pneumonia in the elderly	**2 of 3** pneumonia admissions are aged > 70; **fever and cough absent in 30–40%**; viruses cause **10–30%** of CAP; RR > **30**/min, HR > **110**/min as severity signs; review at **48 hours**; persistent shadow at **12 weeks** → suspect cancer
CURB-65	Confusion, Urea > 7 mmol/L, RR ≥ 30, BP < 90/60, **age ≥ 65**
HAP	Onset **≥ 48 h** after admission; VAP **> 48 h** after intubation; late-onset from **day 5**; treat **7 days**; head-up **30–45°**
DPLD / IPF	IPF median age **65–70**, median survival **3–5 years**; acute exacerbation mortality **> 50%**; progression = **FVC ↓ ≥ 5%** or **DLCO ↓ ≥ 10%** in 1 year; **GAP index**
Pleural effusion	Normal fluid **10–20 mL**; **250–500 mL** to blunt costophrenic angle; **50 mL** detectable on USG; Light's — protein ratio **> 0.5**, LDH ratio **> 0.6**, LDH **> ⅔ ULN**; albumin gradient **> 1.2 g/dL** = transudate; drain if **pH < 7.20** or **glucose < 60 mg/dL**; **ADA > 40 U/L** = TB; aspirate max **1.5 L** at a sitting
COPD	Exacerbations = **10%** of medical admissions; **⅓ readmitted at 30 days, 15% die at 3 months**; ICS if eosinophils **≥ 300/µL**; steroids **5–7 days**; oxygen target **88–92%**
Senile lung	**RV and FRC ↑, FVC ↓, FEV_1/FVC ↓ ("pseudo-obstructive"), TLC ~unchanged**; use **LLN not 0.70**; PRISm = ratio ≥ 0.70 with FEV_1 < 80%
Respiratory failure	PaO_2 **< 60 mmHg (8 kPa)**; $PaCO_2$ **> 50 mmHg (6.7 kPa)**; NIV if **pH < 7.35**; intubate if **pH < 7.25**; normal A–a gradient ≈ **age/4 + 4**
ARDS	Tidal volume **4–6 mL/kg PBW**, plateau **< 30**, driving pressure **< 15** cmH_2O; prone if P/F **< 150** for **≥ 16 h/day**; Berlin severity **≤ 100 / 100–200 / 200–300**; new global definition **SpO_2/FiO_2 ≤ 315**, HFNO **≥ 30 L/min**
Pulmonary embolism	Age-adjusted D-dimer = **age × 10 µg/L** if > 50; high-risk mortality **> 15%**; alteplase **100 mg over 2 h**; sPESI includes **age > 80**; provoked **3 months**
CTEPH	Occurs in **2–4%** after PE; **up to 25%** have no prior PE; mPAP **> 20 mmHg** with PVR **> 2 WU**; screen after **3 months** anticoagulation; **V/Q scan is the screening test of choice**
OSA	AHI **> 5** + symptoms = OSA; elderly — **50% have AHI > 5, 20% have AHI > 15** (vs 10% young); severity **5–15 / 15–30 / > 30**; non-snorers increase after age **50**
Sleep / insomnia	**> 50%** of over-65s have a sleep problem; insomnia prevalence **40–50%**; **50–80%** nap in the day; RLS in **20%** of over-80s; limit naps to **< 30 min**
RBD	Phenoconversion **~6% per year**; **> 70–80% at 12–15 years**; melatonin **3–12 mg**; clonazepam **0.25–1 mg**; α-synuclein SAA positive in **> 90%** of isolated RBD
Solitary pulmonary nodule	Nodule **≤ 3 cm** (> 3 cm = mass); stability **2 years** excludes malignancy (solid); PET useful **> 8 mm**; pneumothorax after CT biopsy **20–25%**; screening **age 50–80, ≥ 20 pack-years**

B. Trials and guidelines to name

Name	One-line message
RALES / EMPHASIS-HF	Spironolactone reduces mortality 30% in NYHA III–IV; eplerenone benefits mild symptoms — equal benefit at all ages
PARADIGM-HF / DIG / SHIFT	Sacubitril–valsartan beats enalapril; digoxin reduces hospitalisation but not mortality; ivabradine for HR ≥ 70
EMPEROR-Preserved / DELIVER	SGLT2 inhibitors reduce events across the whole ejection fraction spectrum, including HFpEF
FINEARTS-HF (NEJM 2024)	Finerenone reduces worsening HF and CV death in HFmrEF and HFpEF
STEP-HFpEF (NEJM 2023)	Semaglutide improves symptoms and exercise capacity in obesity-related HFpEF
STRONG-HF	Rapid uptitration to full dose within 2 weeks of discharge reduces death and readmission
ADVOR / CLOROTIC	Acetazolamide and thiazide added to loop diuretics improve decongestion
CIBIS-II / MERIT-HF / COPERNICUS / SENIORS	The four evidence-based beta-blockers; SENIORS specifically in age ≥ 70
REDUCE-AMI / ABYSS (2024)	No benefit of long-term beta-blockade after MI with preserved EF — a deprescribing opportunity
DanGer Shock (NEJM 2024)	Microaxial flow pump reduces mortality in STEMI-related cardiogenic shock, at the cost of more

	complications
CULPRIT-SHOCK / IABP-SHOCK II	Culprit-lesion-only PCI is superior in shock; balloon pump is neutral
SENIOR-RITA (NEJM 2024)	Invasive strategy in NSTE-ACS over 75 reduces recurrent MI but not death — individualise by frailty
2023 ESC ACS guideline	No routine $P2Y_{12}$ pre-treatment in NSTE-ACS; 24-h invasive timing downgraded to IIa; abbreviated DAPT if HBR
SELECT (NEJM 2023)	Semaglutide reduces MACE by 20% in obesity with established CVD without diabetes
SPRINT / STEP / ESPRIT / BPROAD / HYVET	Intensive systolic lowering benefits older adults; HYVET showed mortality benefit even over 80
2025 AHA/ACC hypertension guideline	Retires 2017 guideline; universal target < 130/80 with encouragement toward < 120 systolic; PREVENT calculator; single-pill combinations; renal denervation IIb
2024 ESC hypertension guideline	New "Elevated BP" category 120–139/70–89; systolic target 120–129 where tolerated; mandatory orthostatic screening
JNC 8 (JAMA 2014)	Age ≥ 60 — treat at ≥ 150/90 to a goal of < 150/90; now superseded
SHEP / Syst-Eur	Treating isolated systolic hypertension reduces stroke by 36–42%; Syst-Eur also reduced dementia
PATHWAY-2	Spironolactone is the drug of choice for resistant hypertension
EARLY TAVR (NEJM 2024)	Early TAVI beats surveillance in asymptomatic severe aortic stenosis
COAPT / RESHAPE-HF2 / MATTERHORN (2024)	Transcatheter edge-to-edge repair reduces HF hospitalisation in secondary MR; non-inferior to surgery with fewer adverse events
SAFE PACE	Carotid sinus syndrome is a modifiable cause of non-accidental falls; pacing reduces falls
BIOSync CLS	Closed-loop stimulation pacing reduces recurrence in tilt-documented asystolic vasovagal syncope
2024 ESC AF guideline	CHA_2DS_2-VA replaces CHA_2DS_2-VASc; AF-CARE replaces ABC; early rhythm control favoured
EAST-AFNET 4 / CASTLE-AF	Early rhythm control improves outcomes; ablation benefits AF with heart failure
NOAH-AFNET 6 / ARTESiA (NEJM 2023)	Device-detected subclinical AF — anticoagulation gives little net benefit; do not treat it as clinical AF
2024 ACC/AHA PAD guideline	Toe brachial index when ABI > 1.40; WIfI staging; supervised exercise first-line; rivaroxaban 2.5 mg bd plus aspirin
SECURE (NEJM 2022) / TIPS-3 / PolyIran	Polypill improves adherence and reduces events; WHO added it to the Essential Medicines List in 2023
FIDELIO-DKD / FIGARO-DKD	Finerenone protects kidney and heart in CKD with type 2 diabetes, with less hyperkalaemia
2022 World Falls Guidelines	Stratified falls pathway; unexplained falls require cardiovascular assessment for syncope
2023 ESC IE guideline / Duke-ISCVID	Cardiac CT and FDG-PET/CT as major criteria; partial oral therapy accepted
POET	Partial oral antibiotics non-inferior to full IV course in stabilised left-sided endocarditis
2023 ESC Cardiomyopathy guideline	First standalone guideline; phenotype-based approach
ATTR-ACT / ATTRibute-CM / HELIOS-B	Tafamidis, acoramidis, vutrisiran — disease-modifying therapy in ATTR cardiac amyloidosis
EXPLORER-HCM	Mavacamten (cardiac myosin inhibitor) for symptomatic obstructive HCM
2023 HRS/APHRS/LAHRS physiologic pacing	Conduction system pacing (LBBAP) when high ventricular pacing burden expected; minimise RV pacing in sinus node disease
ANNEXA-I (2024)	Andexanet alfa better haemostasis than usual care in Xa-inhibitor ICH, but more thrombotic events
AGENT IDE (2024)	Paclitaxel-coated balloon superior to plain balloon for coronary in-stent restenosis; first US coronary DCB approval
NLA/AGS consensus (JAGS 2025)	Statin reasonable > 75 y for primary prevention if LDL-C 70–189 mg/dL and no life-limiting illness; CAC to refine; deprescribe when life-limited
STAREE	Atorvastatin 40 mg vs placebo, age ≥ 70, disability-free survival — **results awaited**
PROSPER / ALLHAT-LLT	Older evidence: modest or no benefit of statins for primary prevention in the old
API-CAT (NEJM 2025)	Extended apixaban 2.5 mg BD non-inferior to 5 mg BD in cancer-associated VTE with less bleeding
CLOTS-1	Graduated compression stockings of no benefit and harmful after stroke
NOTT / MRC	Established the survival benefit of LTOT ≥ 15 hours per day
LOTT	No benefit of oxygen for moderate resting or exercise-induced desaturation
HOT-HMV	Home NIV plus oxygen prolongs time to readmission or death in persistently hypercapnic COPD
ADRIATIC (2024)	Consolidation durvalumab improves overall survival in limited-stage SCLC

LAURA (2024)	Osimertinib after chemoradiotherapy in unresectable stage III EGFR-mutant NSCLC
DeLLphi-301 / 304	Tarlatamab (DLL3 bispecific) in relapsed small cell lung cancer
GOLD 2025/2026	Check inhaler technique at every visit before escalating; consider low-GWP propellants
CAPE COD (NEJM 2023)	Hydrocortisone reduces 28-day mortality in severe CAP
ACIP 2024 (pneumococcal)	Routine vaccination age lowered to 50; single dose PCV20 or PCV21
ACIP 2024 (RSV)	Single lifetime RSV vaccine dose for all ≥ 75, and 50–74 with risk factors
ATS/IDSA 2019 CAP	β-lactam + macrolide/doxycycline, or respiratory fluoroquinolone alone
IDSA AMR guidance 2023–24	Ceftazidime-avibactam, cefiderocol, sulbactam-durlobactam for resistant HAP/VAP; 7-day courses
2022 ATS/ERS/JRS/ALAT	Defined progressive pulmonary fibrosis (PPF); nintedanib recommended
INBUILD	Nintedanib slows FVC decline in progressive fibrosing ILD of any cause
PANTHER-IPF	Prednisolone + azathioprine + NAC **increases mortality** in IPF — steroids are harmful
FIBRONEER-IPF / -ILD (NEJM 2025)	Nerandomilast (oral PDE4B inhibitor) reduces FVC decline in IPF and PPF, alone or added to antifibrotics
BTS Pleural Disease 2023	Ultrasound for all procedures; thoracoscopy for undiagnosed exudate; IPC or talc equally acceptable; RAPID score
MIST-2	Intrapleural alteplase + DNase improves drainage in pleural infection
AMPLE / IPC-PLUS	Indwelling pleural catheter reduces hospital days; talc via IPC achieves pleurodesis
BOREAS / NOTUS (NEJM 2023, 2024)	Dupilumab reduces exacerbations in eosinophilic COPD on triple therapy
GINA 2024–2025	No SABA-alone at any step; ICS-formoterol (AIR/MART) is the preferred track
GOLD 2023–2026	Pre-COPD and PRISm defined; LLN preferred over fixed 0.70 ratio; check inhaler technique before escalating
ARMA / ARDSNet	Low tidal volume 4–6 mL/kg PBW reduces mortality in ARDS
PROSEVA	Prone positioning ≥ 16 h/day reduces mortality in severe ARDS
ROSE / ART / FACTT / EOLIA	Routine paralysis neutral; aggressive recruitment harmful; conservative fluids beneficial; ECMO for refractory ARDS
New Global Definition of ARDS (2023)	Adds non-intubated ARDS on HFNO ≥ 30 L/min, $SpO_2/FiO_2 \leq 315$, lung ultrasound
PEITHO	Thrombolysis in intermediate-risk PE prevents decompensation but causes excess bleeding, especially > 75 years
PEERLESS (Circulation 2025) / STORM-PE	Large-bore mechanical thrombectomy better than catheter-directed thrombolysis, and better than anticoagulation alone for RV recovery
CHEST-1 / RACE / MACiTEPH (2025)	Riociguat is the only licensed CTEPH drug; riociguat pre-treatment makes BPA safer; macitentan trial stopped for futility
SURMOUNT-OSA (NEJM 2024)	Tirzepatide markedly reduces AHI — first drug approved for OSA
SAVE	CPAP improves symptoms but did not reduce cardiovascular events (adherence-limited)
AGS Beers 2023	Avoid benzodiazepines and Z-drugs in older adults; DORAs are the preferred hypnotic if one is needed
NSD-ISS 2024 / SynNeurGe	Isolated RBD with a positive α-synuclein seed amplification assay = biologically defined prodromal synuclein disease
ACS 2023 / Lung-RADS 2022	LDCT screening 50–80 with ≥ 20 pack-years, no quit-time limit; standardised nodule categories

C. Indian context lines worth adding

Topic	Line to write
Heart failure and valve disease	**Rheumatic valvular heart disease** remains an important cause of heart failure and of mitral and aortic disease in Indian older adults
Anticoagulation in AF	**Rheumatic mitral stenosis and mechanical valves require warfarin, not a DOAC** — a common examination trap in the Indian setting
Hypertension	High dietary salt with low potassium intake, a large treatment gap, and **API and Lipid Association of India** consensus favouring early combination therapy and intensive LDL targets
Atherosclerosis	Indians develop ASCVD **a decade earlier**, with more small dense LDL, higher Lp(a) and a higher prevalence of diabetes at lower BMI
Polypill	India led this field through the **TIPS/Polycap** programme; WHO **HEARTS** and the national NCD programme promote standardised fixed-dose regimens
Cardiorenal syndrome	Consider **NSAID and traditional/herbal remedy nephrotoxicity** and late presentation with advanced CKD as common contributors
Infective endocarditis	Rheumatic valvular disease remains an important substrate in India, with viridans streptococci still prominent
Cardiomyopathy	**Endomyocardial fibrosis** is a regionally important cause of restrictive cardiomyopathy in India
DOACs	Rheumatic mitral stenosis is common in India — these patients need **warfarin, not a DOAC**; idarucizumab and andexanet have limited availability, so **4F-PCC** is the practical reversal option
Lung cancer	Biomass fuel smoke and indoor air pollution are major risks in never-smoking Indian women; post-tubercular scarring is a recognised substrate
Statins / prevention	Indians develop ASCVD at a younger age with a higher burden of small dense LDL; API and Lipid Association of India consensus recommend lower LDL-C targets in high-risk groups
Pneumonia	Always consider **tuberculosis** in subacute pneumonia and send sputum for AFB/CBNAAT; aspergillosis complicates old TB cavities
HAP	**Carbapenem-resistant Klebsiella and Acinetobacter** dominate Indian ICUs — empirical therapy must follow the local antibiogram
DPLD	**Hypersensitivity pneumonitis (birds, mould, biomass)** and **CTD-ILD** form a larger share than in Western registries; exclude post-tubercular fibrosis
Pleural effusion	**Tuberculosis is often the commonest exudative effusion**; **ADA > 40 U/L with lymphocytic predominance** is the key test; ATT for 6 months
COPD / senile lung	**Biomass fuel smoke and indoor air pollution** are major non-smoking causes of airflow obstruction, especially in rural women
Solitary nodule	**Tuberculoma is the commonest benign SPN**, and granulomatous disease causes **false-positive PET-CT** — always obtain tissue before declaring malignancy

MD GERIATRIC MEDICINE

Consolidated Examination Answer Bank — NEUROLOGY

31 Questions • Organised by Subject • Mark-Wise Format • Updated to 2026

Scope. This volume consolidates the complete neurology answer bank into a single continuously numbered document, organised into six subject sections. Duplicated and overlapping questions have been merged, and the two Rapid Revision Grids have been combined into one appendix.

Primary source: Pathy's Principles and Practice of Geriatric Medicine, 6th edition (2022); Essentials of Clinical Geriatrics, 7th edition.

Latest updates: Guidelines, randomised trials and regulatory decisions, 2023–2026 (AHA/ASA, ERC/ESICM, ILAE, AASM, AAN, EAN, NICE, WHO, VA/DoD, AGS Beers, FDA/EMA, NEJM, JAMA, Lancet).

Mark-wise point counts used throughout:

- **20 marks** = 14 textbook points + 3 latest updates = **17 points**
- **15 marks** = 10 textbook points + 2 latest updates = **12 points**
- **7 marks** = 6 textbook points + 1 latest update = **7 points**
- **6 marks** = 5 textbook points + 1 latest update = **6 points**

Panels used in this document. Purple = *Latest Updates*. Blue table = *Current Practice Update*, present only where the textbook meaningfully differs from present-day recommendations. Orange = *Examiner's Tip* with verbatim mark-earning lines. Green = *Extra Points to Remember* or guidance for converting an answer between mark weights, used for repeated questions.

Compiled 27 August 2026 • Each question begins on a fresh page for clean printing and rapid lookup.

Table of Contents

SECTION A — EPILEPSY AND SEIZURE DISORDERS		
1.	Epilepsy in Elderly People and its Management	20 MARKS
2.	Complex Partial Seizure	7 MARKS
3.	Newer Anticonvulsants	7 MARKS
4.	Case — 80-Year-Old Hypertensive, Diabetic Woman with CAD and Atrial Fibrillation, Admitted with Altered Sensorium Following GTCS	15 MARKS
SECTION B — CEREBROVASCULAR DISEASE		
5.	Transient Ischaemic Attack	7 MARKS · ASKED ×2
6.	Lacunar Stroke	7 MARKS
7.	Thrombolysis and Mechanical Thrombectomy in Acute Stroke	7 MARKS
8.	Wallenberg Syndrome — Cause, Clinical Features and Treatment	7 MARKS
9.	Subarachnoid Haemorrhage	7 MARKS
10.	Treatment of Symptomatic and Asymptomatic Carotid Disease in the Elderly	7 MARKS
11.	Subdural Haematoma in the Elderly	7 MARKS
12.	Post-Stroke Depression and Post-Stroke Pain	7 MARKS
SECTION C — NEURODEGENERATIVE, MOVEMENT AND BALANCE DISORDERS		
13.	Normal Pressure Hydrocephalus	7 MARKS
14.	Progressive Supranuclear Palsy and Corticobasal Degeneration	7 MARKS
15.	Motor Neuron Disease — Presentation, Evaluation and Treatment	7 MARKS
16.	Restless Legs Syndrome	7 MARKS
17.	Benign Paroxysmal Positional Vertigo — Diagnosis and Epley Manoeuvre	7 MARKS
SECTION D — NEUROMUSCULAR, SPINAL CORD AND PERIPHERAL NERVE DISORDERS		
18.	Myasthenia Gravis in the Older Adult	7 MARKS
19.	Subacute Combined Degeneration of the Cord	7 MARKS
20.	Peripheral Neuropathy in Older Adults — Aetiology, Clinical Features, Investigations and Management	15 MARKS · ASKED ×2
SECTION E — HEADACHE AND FACIAL PAIN		
21.	Temporal Arteritis and its Neurological Complications	7 MARKS
22.	Trigeminal Neuralgia	7 MARKS
23.	Cluster Headache	7 MARKS · ASKED ×2
24.	Tension Headache	6 MARKS
SECTION F — NEUROLOGICAL EMERGENCIES, ENCEPHALOPATHIES AND TOXIDROMES		
25.	Hypoxic-Ischaemic Encephalopathy — Causes, Clinical Manifestations, Diagnosis and Management	15 MARKS
26.	Post-Acute Management of Traumatic Brain Injury	7 MARKS
27.	Wernicke Encephalopathy	7 MARKS
28.	CSF Findings in Pyogenic, Tuberculous, Fungal and Carcinomatous Meningitis	7 MARKS
29.	Serotonin Syndrome	7 MARKS
30.	Neuroleptic Malignant Syndrome	7 MARKS
31.	Rhabdomyolysis	7 MARKS
APPENDIX		
	Rapid Revision Grid — examinable numbers, named trials and guidelines, drugs of choice	

SECTION A
Epilepsy and Seizure Disorders

Question 1. Epilepsy in Elderly People and its Management 20 MARKS

Answer

Textbook Core – 14 Points

1. **Definition and burden.** The **elderly have the highest incidence of new-onset epilepsy of any age group** — over 100 per 1,00,000 per year after 65. ILAE definition: two unprovoked seizures more than 24 hours apart, **or one unprovoked seizure with a recurrence risk above 60%**, or an epilepsy syndrome. Because recurrence after a first unprovoked seizure with a structural lesion is **60–90%** in old age, **treatment is usually started after the first seizure**.
2. **Causes. Cerebrovascular disease is the commonest cause (30–50%)** — both acute stroke and chronic small-vessel disease. Others: **neurodegenerative dementia** (Alzheimer's carries up to a 10-fold risk), **brain tumour** (primary and metastatic), head trauma and **chronic subdural haematoma**, CNS infection (in India, **neurocysticercosis and tuberculoma**), metabolic derangement, and alcohol or drug withdrawal. Around **25–50% remain unexplained** (late-onset unexplained epilepsy).
3. **Seizure types. Focal (partial) seizures predominate**, most often **focal seizures with impaired awareness** of temporal or frontal origin, with or without secondary generalisation. Primary generalised epilepsy beginning in old age is rare, so a generalised seizure in an older person should prompt a search for a focal structural or metabolic cause.
4. **Atypical presentation — the core geriatric point.** The aura is often **absent or not reported**. Seizures present as **brief staring and behavioural arrest, unexplained confusion, memory lapses, wandering, falls or syncope-like collapse**. Automatisms may be subtle. **Post-ictal confusion is prolonged — often hours to days, occasionally weeks — and Todd's paresis lasts longer**, so epilepsy is frequently misdiagnosed as TIA, syncope, delirium or dementia.
5. **Acute symptomatic (provoked) seizures must be separated from epilepsy.** Seizures occurring **within 7 days of a stroke, head injury or CNS infection**, or from hypoglycaemia, hyponatraemia, uraemia, hepatic failure, hypoxia, sepsis, drugs or alcohol withdrawal, are **provoked** — they do not by themselves define epilepsy and often do not require long-term antiseizure medication once the cause is corrected.
6. **Differential diagnosis. Syncope** (cardiac arrhythmia, orthostatic hypotension, carotid sinus hypersensitivity — the commonest misdiagnosis), **TIA** (negative rather than positive phenomena, no post-ictal confusion), transient global amnesia, hypoglycaemia, delirium, sleep disorders including **REM sleep behaviour disorder**, drop attacks, and psychogenic non-epileptic attacks.
7. **History is the single most valuable investigation.** Obtain a **detailed eyewitness account** — posture and circumstances at onset, warning, colour, duration, movements, tongue biting, incontinence, and the nature and duration of recovery. Review the drug chart for **pro-convulsant drugs**: tramadol, quinolones, bupropion, theophylline, antipsychotics, high-dose beta-lactams, and abrupt benzodiazepine withdrawal.
8. **Investigations. ECG with 24-hour Holter or tilt testing** to exclude cardiac syncope. Blood tests: sodium, calcium, magnesium, glucose, urea, creatinine, LFT, and drug levels. **EEG has low sensitivity in the elderly (about 26–38%)** and interictal abnormalities are less common, so a normal EEG never excludes epilepsy; use sleep-deprived, prolonged or video-EEG where doubt persists. **MRI brain is the imaging investigation of choice** — superior to CT for hippocampal sclerosis, small infarcts and tumour.
9. **Non-convulsive status epilepticus (NCSE).** An under-recognised and treatable cause of **unexplained prolonged confusion, stupor or coma in the elderly**, often after a generalised seizure or in a septic/metabolically deranged patient. **EEG is mandatory in any older patient whose consciousness does not recover within 30–60 minutes of a seizure.**
10. **Principles of drug therapy.** Use **monotherapy**; **start low, go slow, and aim for a lower maintenance dose than in younger adults**. Age alters pharmacokinetics: reduced serum albumin raises the **free (active) fraction of phenytoin and valproate**, reduced hepatic and renal clearance prolongs half-lives, and reduced lean body mass lowers the volume of distribution. **Measure free rather than total phenytoin** where available.
11. **Drug of choice. Levetiracetam and lamotrigine are first line for focal epilepsy in older adults**, with lacosamide as an alternative; levetiracetam has the highest chance of long-term seizure freedom and lamotrigine the best tolerability. **Avoid enzyme-inducing drugs** — carbamazepine, phenytoin and phenobarbitone — because they interact with warfarin, DOACs, statins, antihypertensives, antiarrhythmics and chemotherapy, and accelerate **vitamin D metabolism and bone loss**. Gabapentin is a last resort.
12. **Adverse effects that matter in old age. Hyponatraemia** (carbamazepine, oxcarbazepine, eslicarbazepine); **ataxia, dizziness and falls** with almost all agents; sedation and cognitive slowing (topiramate, phenobarbitone, benzodiazepines); **osteoporosis and fracture** with enzyme inducers; **behavioural change, irritability and depression with levetiracetam**; rash and Stevens–Johnson syndrome with lamotrigine if titrated fast; oedema and cognitive change with gabapentin and pregabalin; and phenytoin's zero-order kinetics, gum hypertrophy and cerebellar atrophy.

13. **Status epilepticus.** ABC, oxygen, IV access, **capillary glucose, and IV thiamine before glucose**. First line: **IV lorazepam 0.1 mg/kg** (or IM midazolam 10 mg if no access). Second line: **IV levetiracetam 60 mg/kg, fosphenytoin 20 PE/kg, or valproate 40 mg/kg** — all equally effective. Third line: anaesthesia with midazolam or propofol infusion and continuous EEG. In the elderly, **reduce doses, infuse slowly and monitor blood pressure and respiration closely**.

14. **Holistic and non-drug management, and prognosis.** Injury and **falls prevention**, bathing and cooking safety, driving restriction, treatment of vascular risk factors, deprescribing pro-convulsant and anticholinergic drugs, **bone health (calcium, vitamin D, DEXA)**, screening for depression and cognitive impairment, carer education in first aid, and counselling about SUDEP. Prognosis is good — **about 80% become seizure-free on low-dose monotherapy** — but mortality, fracture and dementia risk remain elevated.

Latest Updates – 3 Points

15. **ILAE Task Force on Epilepsy in the Elderly — critical review (*Epilepsia*, 2023).** An **individualised approach is essential** because older adults are a highly heterogeneous group. Across comparative studies, **levetiracetam has the highest probability of long-term seizure freedom**, while **carbamazepine users are almost twice as likely to discontinue treatment for adverse effects as lamotrigine users**. Current practice therefore places **lamotrigine, levetiracetam and lacosamide first line, with gabapentin as a last resort**. The Task Force also highlighted that quality of life is a missing outcome in most trials, and that prognosis must move beyond seizure control to patient-centred outcomes.

16. **Late-onset unexplained epilepsy (LOUE) is a marker of vascular brain disease and future dementia.** Recent cohort work and the prospective multicentre **ELUCID study (2025)** show that in LOUE the excess morbidity and mortality relate **more to incident dementia and stroke than to the seizures themselves**, and that seizures are usually easily controlled. Practical implication: a first seizure after the age of 55 should trigger **aggressive vascular risk-factor management and structured cognitive follow-up**, not merely a prescription for an antiseizure drug.

17. **Antiseizure medication–anticoagulant interactions (2024–2025).** Emerging evidence, including a systematic review of levetiracetam and DOACs (2024) and outcome data in older Asian patients with atrial fibrillation (2025), suggests that **even "non-interacting" agents such as levetiracetam may lower DOAC levels through P-glycoprotein induction**. About **one in six older patients with epilepsy is at risk of a clinically significant drug interaction**. Non-enzyme-inducing agents remain preferred, but a formal interaction check — not an assumption of safety — is now the standard.

Current Practice Update

Item	Position
Textbook	Phenytoin and carbamazepine remain widely used; newer agents are reserved as add-on therapy. A first unprovoked seizure may be observed without treatment.
Current recommendation	**Lamotrigine, levetiracetam and lacosamide are first line**; enzyme inducers are avoided because of interactions and bone loss. Treat after the **first** seizure when a structural cause is present. Late-onset epilepsy is managed as a **vascular and cognitive risk state**, not just a seizure disorder.
Exam approach	Write the classical framework, then state: "The ILAE Task Force (2023) recommends lamotrigine, levetiracetam or lacosamide first line in older adults, with gabapentin as a last resort."

Examiner's Tip — Verbatim Mark-Earning Lines

- "The elderly have the **highest incidence of new-onset epilepsy of any age group**, and cerebrovascular disease is the commonest cause."
- "**A normal EEG never excludes epilepsy in an older person** — sensitivity is only 26–38%."
- "Any older patient with **prolonged unexplained confusion needs an EEG to exclude non-convulsive status epilepticus**."
- "**Start low, go slow, aim low** — older patients respond to lower doses and tolerate fewer adverse effects."
- **Skeleton:** Incidence/definition → Causes → Seizure types → Atypical presentation → Provoked vs unprovoked → Differentials → History → Investigations → NCSE → Prescribing principles → Drug of choice → Adverse effects → Status epilepticus → Holistic care/prognosis → 3 updates.

Question 2. Complex Partial Seizure 7 MARKS

Answer

Textbook Core – 6 Points

1. **Definition and current terminology.** A seizure arising in **one hemisphere with impairment of awareness or consciousness**. The **ILAE 2017 classification renamed "complex partial seizure" as focal seizure with impaired awareness** (focal impaired-awareness seizure) — both terms should be written in the answer. It is the **commonest seizure type in older adults**.
2. **Site of origin and causes.** Most arise from the **temporal lobe (mesial temporal structures — hippocampus and amygdala)**, fewer from the frontal lobe. Causes in the elderly: stroke and small-vessel disease, tumour, hippocampal sclerosis, head trauma and subdural haematoma, Alzheimer's disease, and in India **neurocysticercosis and tuberculoma**.
3. **Clinical sequence (temporal lobe type). Aura** — **rising epigastric sensation, déjà vu or jamais vu, sudden fear, olfactory or gustatory hallucination** → **motionless stare and behavioural arrest** → **automatisms**: oro-alimentary (lip smacking, chewing, swallowing) and manual (picking, fumbling) → **post-ictal confusion and amnesia**. Attacks last **30 seconds to 2–3 minutes**, with post-ictal confusion of 5–30 minutes, often much longer in the elderly.
4. **Frontal lobe variant.** Brief, **frequently nocturnal, occurring in clusters**, with bizarre hypermotor, bicycling or thrashing movements, vocalisation and **minimal or absent post-ictal confusion** — commonly mislabelled as psychogenic non-epileptic attacks or a sleep disorder.
5. **Differential diagnosis. Absence seizure** (no aura, seconds only, **no post-ictal confusion**, 3 Hz generalised spike-and-wave, a disorder of childhood), TIA, transient global amnesia, syncope, hypoglycaemia, delirium and psychogenic non-epileptic seizures. In older adults the **prolonged post-ictal state readily mimics delirium or dementia**, and repeated brief attacks may be dismissed as "absent-mindedness".
6. **Investigations and treatment. EEG** — anterior temporal spikes or sharp waves; yield improves with sleep-deprived or prolonged recording. **MRI brain** for hippocampal sclerosis, tumour or infarct. Treatment: focal-onset agents — **levetiracetam or lamotrigine are preferred in older adults**; lacosamide, oxcarbazepine and carbamazepine are alternatives. In drug-resistant cases (failure of two appropriate drugs), refer for pre-surgical evaluation — **anterior temporal lobectomy** gives seizure freedom in about two-thirds of selected patients.

Latest Update – 1 Point

7. **Focal impaired-awareness seizures in Alzheimer's disease.** Work published from 2023 onward has established that **subclinical epileptiform activity and unrecognised focal impaired-awareness seizures are common in Alzheimer's disease** and are associated with earlier onset and faster cognitive decline. The practical implication is that an older patient with dementia and **fluctuating cognition, unexplained confusional episodes, staring spells or amnestic gaps** should have a **prolonged or sleep EEG**, since these episodes are treatable — a diagnosis routinely missed when they are attributed to the dementia itself.

Examiner's Tip — Verbatim Mark-Earning Lines

- "**Aura → blank stare → automatisms → post-ictal confusion** — the four-step signature of a focal impaired-awareness seizure."
- "**Post-ictal confusion distinguishes a complex partial seizure from an absence seizure.**"
- "Always write both terms: **complex partial seizure = focal seizure with impaired awareness (ILAE 2017)**."
- **Skeleton:** Definition + ILAE 2017 term → Temporal origin/causes → Clinical sequence → Frontal variant → Differentials → EEG/MRI/drugs/surgery → Alzheimer's overlap.

Question 3. Newer Anticonvulsants 7 MARKS

Answer

Textbook Core – 6 Points

1. **Definition and general advantages.** The **second- and third-generation antiseizure medications** — lamotrigine, levetiracetam, topiramate, gabapentin, pregabalin, oxcarbazepine, zonisamide, lacosamide, brivaracetam, perampanel, eslicarbazepine and cenobamate. Their advantages over phenytoin, carbamazepine, phenobarbitone and valproate are **fewer cytochrome P450 interactions, linear (first-order) kinetics, low protein binding, a wider therapeutic index, and no need for routine level monitoring** — all particularly valuable in polypharmacy.
2. **Mechanisms of action. Sodium-channel blockade** — lamotrigine, oxcarbazepine, eslicarbazepine; **slow inactivation of sodium channels** — lacosamide; **SV2A synaptic vesicle protein binding** — levetiracetam and brivaracetam; **α2δ calcium-channel subunit binding** — gabapentin and pregabalin; **AMPA receptor antagonism** — perampanel; **multiple mechanisms** — topiramate and zonisamide.
3. **Key drugs and their place. Levetiracetam** — broad spectrum, intravenous form available, no significant interactions, renal dose adjustment; the workhorse drug in geriatrics. **Lamotrigine** — broad spectrum, mood-stabilising, best tolerability, but needs slow titration. **Lacosamide** — focal seizures, IV available. **Oxcarbazepine** — focal seizures, better tolerated than carbamazepine. **Topiramate** — broad spectrum and also effective in migraine. **Gabapentin and pregabalin** — weak antiseizure agents, mainly used for neuropathic pain.
4. **Adverse effects to name.** Lamotrigine — **rash and Stevens-Johnson syndrome if titrated too fast (halve the dose when combined with valproate)**. Levetiracetam — **irritability, aggression, depression and psychosis**. Topiramate — cognitive slowing, word-finding difficulty, weight loss, **renal stones, acute glaucoma and metabolic acidosis**. Oxcarbazepine and eslicarbazepine — **hyponatraemia**. Perampanel — serious psychiatric and behavioural reactions (boxed warning). Lacosamide — dizziness and **PR interval prolongation**. Zonisamide — stones and oligohidrosis. Gabapentinoids — oedema, sedation, weight gain and misuse potential.
5. **Geriatric considerations.** These agents are **preferred over the older drugs in old age** because they do not induce CYP450 enzymes and therefore do not compromise warfarin, DOACs, statins, antihypertensives, antiarrhythmics or chemotherapy, and they cause less bone loss. However, **levetiracetam, gabapentin, pregabalin and lacosamide all require dose reduction for creatinine clearance**, and every agent can cause dizziness, ataxia and falls. Sedative and anticholinergic burden must be counted under the **AGS Beers Criteria**.
6. **Limitations.** They are **substantially more expensive** — a major issue in Indian practice — and parenteral formulations are limited. Importantly, they do **not achieve higher seizure-freedom rates** than the older drugs: **SANAD II showed that levetiracetam was neither clinically superior nor cost-effective compared with lamotrigine for focal epilepsy**, and valproate remained best for generalised epilepsy. Evidence in the very old and the frail remains sparse.

Latest Update – 1 Point

7. **Cenobamate — the newest third-generation agent for drug-resistant focal epilepsy.** It has a **dual mechanism — inhibition of the persistent sodium current plus positive allosteric modulation of GABA-A receptors** — and produces unusually high seizure-freedom rates in adjunctive trials (around 20% at the highest dose, far above the usual 2–5% for add-on agents). Two cautions are examinable: it must be **titrated very slowly over at least 12 weeks starting at 12.5 mg daily to avoid DRESS (drug reaction with eosinophilia and systemic symptoms)**, and it **induces CYP3A4 and inhibits CYP2C19**, so interactions must be rechecked — a real consideration in the polypharmacy of old age. Brivaracetam, eslicarbazepine and perampanel are currently under study in older populations.

Examiner's Tip — Verbatim Mark-Earning Lines

- "The advantage of the newer agents is **predictable kinetics and freedom from enzyme induction**, not superior efficacy."
- "**Levetiracetam causes behavioural disturbance; lamotrigine causes rash; topiramate causes cognitive slowing and stones; oxcarbazepine causes hyponatraemia.**"
- "SANAD II: newer does not mean better — **lamotrigine remained the reference standard for focal epilepsy**."
- **Skeleton:** Definition/advantages → Mechanism table → Key drugs → Adverse effects → Geriatric prescribing → Cost and SANAD II → Cenobamate.

Question 4. Case — 80-Year-Old Hypertensive, Diabetic Woman with CAD and Atrial Fibrillation, Admitted with Altered Sensorium Following GTCS 15 MARKS

Answer

Textbook Core – 10 Points

1. **Frame the problem.** A generalised tonic-clonic seizure at 80 years is **an acute symptomatic (provoked) seizure until proved otherwise**, not idiopathic epilepsy. Two features dominate the differential: she is **on antithrombotic therapy for atrial fibrillation**, so **intracranial haemorrhage** must be excluded first; and she is **diabetic**, so **hypoglycaemia** must be excluded immediately.
2. **Immediate stabilisation (first 10 minutes).** Airway, left lateral position, oxygen, suction, IV access, cardiac and SpO_2 monitoring. **Check capillary blood glucose at the bedside** and give **IV thiamine 100 mg before any glucose**. If seizures continue: **IV lorazepam 0.1 mg/kg (maximum 4 mg per dose)**; if status persists, second-line **IV levetiracetam 60 mg/kg, fosphenytoin 20 PE/kg or valproate 40 mg/kg** — all equally effective; then anaesthesia with continuous EEG. Infuse slowly and watch blood pressure and respiration.
3. **Focused history from attendants.** The **exact drug list** — insulin or sulfonylurea, metformin, **warfarin versus a DOAC, antiplatelets**, beta-blocker, digoxin, amiodarone, statin, and pro-convulsant drugs such as tramadol or a quinolone; recent **falls or head injury**; fever; missed or doubled doses; alcohol; prior stroke, seizure or dementia; and her **baseline cognition and functional status**, which will shape every subsequent decision.
4. **Examination.** Vital signs and temperature, GCS, pupils, fundi, neck stiffness, and a search for **lateralising signs and Todd's paresis**. **Lateral tongue bite and urinary incontinence support a true GTCS**. Look for scalp haematoma, periorbital bruising and other signs of head injury; examine the heart for atrial fibrillation, murmur and failure; and screen for a septic focus — chest, urine, skin and pressure areas.
5. **Differential diagnosis to work through. Intracranial haemorrhage — anticoagulant-related intracerebral haemorrhage and chronic subdural haematoma**; acute ischaemic stroke or cardioembolic infarct with seizure; **hypoglycaemia**; **hyponatraemia**; uraemic or hepatic encephalopathy; sepsis-associated encephalopathy; hypoxic-ischaemic injury after an arrhythmia; CNS infection (meningoencephalitis, tuberculoma, neurocysticercosis); drug toxicity or withdrawal; hypertensive encephalopathy/PRES; and **non-convulsive status epilepticus**.
6. **Investigations — immediate.** Capillary and laboratory glucose; **serum sodium, calcium, magnesium**; urea, creatinine and electrolytes; LFT; arterial blood gas; full blood count; CRP or procalcitonin; **ECG and troponin** (silent myocardial infarction or arrhythmia is a real possibility with her CAD and AF); **coagulation profile and INR — mandatory given anticoagulation**; blood and urine cultures; chest X-ray.
7. **Investigations — neurological. Urgent non-contrast CT brain** is the first imaging study, looking for haemorrhage, subdural collection, infarct or mass. If the CT is non-diagnostic, proceed to **MRI with DWI ± MR angiography**. Lumbar puncture after imaging if infection is suspected and there is no contraindication — noting that anticoagulation must be reversed first. **EEG is essential if consciousness does not recover within 30–60 minutes, to exclude non-convulsive status epilepticus.**
8. **Managing the anticoagulated patient.** If intracranial haemorrhage is confirmed: **stop the anticoagulant and reverse it immediately — vitamin K plus 4-factor prothrombin complex concentrate for warfarin, idarucizumab for dabigatran, and andexanet alfa or PCC for a factor Xa inhibitor**; lower systolic blood pressure to about 130–140 mmHg; obtain a neurosurgical opinion; and decide on the timing of restarting anticoagulation later with the cardiology team. If instead there is an ischaemic stroke, a seizure at onset does not by itself preclude thrombolysis when the deficit is clearly attributable to the stroke.
9. **Antiseizure medication decision. An acute symptomatic seizure from a fully reversible metabolic cause does not require long-term antiseizure medication** — correct the cause. If a **structural lesion** (haemorrhage, infarct, subdural, tumour) is found, start maintenance therapy: **levetiracetam is the drug of choice** — intravenous formulation, no CYP450 interactions with her anticoagulant, statin or antiarrhythmic, with the dose adjusted for creatinine clearance. **Avoid phenytoin and carbamazepine**, which interact with warfarin, DOACs, statins and amiodarone and cause hypotension and arrhythmia on rapid infusion.
10. **Supportive and comprehensive geriatric care.** Aspiration precautions with a **formal swallow screen before oral intake**; VTE prophylaxis; pressure-area care; a **delirium prevention bundle** (orientation, sleep, mobilisation, avoidance of restraints, catheters and anticholinergics); nutrition; early physiotherapy; glycaemic control that avoids hypoglycaemia; deprescribing of pro-convulsant and anticholinergic drugs; falls, driving and home-safety advice; and an early, explicit **goals-of-care discussion with the family**, followed by comprehensive geriatric assessment before discharge.

Latest Updates – 2 Points

11. **ANNEXA-I (*NEJM* 2024) — reversal of factor Xa inhibitor-associated intracerebral haemorrhage.** In 530 patients (mean age 79 years) with ICH within 15 hours of a factor Xa inhibitor, **andexanet alfa achieved better haemostatic efficacy than usual care (67.0% vs 53.1%, p=0.003)** but caused **more thrombotic events (10.3% vs 5.6%, p=0.048), including ischaemic stroke (6.5% vs 1.5%)**, with **no difference in 30-day mortality or modified Rankin outcome**. Because of this, plus cost and availability, **4-factor PCC remains the widely used option in Indian practice** — a nuance worth writing.

12. **AHA/ASA 2026 Acute Ischaemic Stroke Guideline.** The approach to **thrombolysis contraindications has been modified**, and **seizure at stroke onset is no longer treated as an absolute barrier** when the deficit is attributable to the infarct. **Tenecteplase 0.25 mg/kg (maximum 25 mg) as a single bolus is now a Class 1 recommendation** alongside alteplase within 4.5 hours, thrombolysis should be given for disabling deficits regardless of NIHSS score, and thrombectomy eligibility has been broadened to **ASPECTS 3–10 and up to 24 hours** with mismatch imaging — all directly relevant to an 80-year-old whose first GTCS turns out to be a stroke.

Examiner's Tip — Verbatim Mark-Earning Lines

- "In an anticoagulated older patient, a new seizure is **intracranial haemorrhage until the CT proves otherwise**."
- "**Check the glucose before you check anything else**; give thiamine before glucose."
- "If she does not wake up within an hour, **get an EEG — non-convulsive status epilepticus is the commonest missed diagnosis**."
- "**Levetiracetam is the antiseizure drug of choice** in a patient on an anticoagulant, a statin and an antiarrhythmic."
- **Skeleton:** Frame the problem → ABC + glucose + benzodiazepine → History → Examination → Differentials → Labs → CT/MRI/EEG → Reversal of anticoagulation → ASM decision → CGA and goals of care → 2 updates.

Question 5. Transient Ischaemic Attack 7 MARKS • ASKED ×2

Answer

Textbook Core – 6 Points

1. **Definition.** A **transient episode of neurological dysfunction caused by focal brain, spinal cord or retinal ischaemia, without acute infarction on imaging** (tissue-based definition). The old time-based definition of "deficit resolving within 24 hours" is **obsolete**, because up to one-third of clinically transient events show a diffusion-weighted MRI lesion — and are therefore strokes.
2. **Clinical features by territory. Carotid** — contralateral hemiparesis, hemisensory loss, dysphasia, and **amaurosis fugax** (a curtain descending over one eye). **Vertebrobasilar** — vertigo *with* other brainstem signs, diplopia, dysarthria, dysphagia, ataxia, bilateral or crossed deficits, drop attacks. **Isolated vertigo, syncope, confusion, generalised weakness or a "funny turn" are NOT a TIA.**
3. **Risk stratification — ABCD^2 score. A**ge ≥60 = 1; **B**lood pressure ≥140/90 = 1; **C**linical features: unilateral weakness = 2, speech disturbance without weakness = 1; **D**uration ≥60 min = 2, 10–59 min = 1; **D**iabetes = 1. **A score of 4 or more is high risk.** Stroke risk is greatest in the **first 48 hours** — roughly 5% at 7 days and 10% at 90 days if untreated. **A TIA is a medical emergency, not an outpatient problem.**
4. **Investigations — all within 24 hours. MRI with diffusion-weighted imaging is the investigation of choice** (CT if MRI is unavailable); **carotid Doppler, CT angiography or MR angiography**; **ECG with prolonged cardiac monitoring** (Holter or implantable loop recorder) — occult atrial fibrillation is a major and treatable cause in the elderly; echocardiography; glucose and HbA1c, lipids, full blood count, renal function, and **ESR if amaurosis fugax (giant cell arteritis)**.
5. **Acute antithrombotic treatment.** For high-risk TIA (ABCD^2 ≥4) of non-cardioembolic origin, give **dual antiplatelet therapy — aspirin plus clopidogrel for 21 days, then a single agent** (loading aspirin 300 mg and clopidogrel 300 mg; CHANCE and POINT trials). Add a **high-intensity statin**, control blood pressure and glycaemia, and stop smoking. **If atrial fibrillation is present, the treatment is anticoagulation, not antiplatelets** — a DOAC is preferred over warfarin in older adults.
6. **Pathway and secondary prevention.** Assess in a **TIA clinic within 24 hours** for high-risk patients; carotid revascularisation **within 2 weeks** for symptomatic 70–99% stenosis; treat the whole vascular risk profile; give driving advice (in India, cease driving for at least one month and reassess); and screen for **cognitive impairment, depression and falls**, since a TIA in an older person is a sentinel event for both vascular dementia and frailty.

Latest Update – 1 Point

7. **INSPIRES trial (*NEJM* 2023; subgroup analyses 2024) — the DAPT window extends to 72 hours.** In 6,100 patients with **mild ischaemic stroke or high-risk TIA of presumed atherosclerotic cause**, **clopidogrel plus aspirin started within 72 hours reduced new stroke at 90 days compared with aspirin alone**, at the cost of a small excess of moderate-to-severe bleeding; efficacy was similar whether treatment began before 24 hours or between 24 and 72 hours. This **widens the previous 24-hour window to 72 hours** — a change of real practical value in India, where patients frequently present late. The AHA/ASA 2026 guideline likewise supports dual antiplatelet therapy for non-disabling stroke and high-risk TIA.

Extra Points to Remember (repeated question)

- **Crescendo TIA** (increasing frequency or duration) and **TIA while already on an antiplatelet** both mandate immediate admission.
- **TIA mimics** to exclude: migraine aura, focal seizure with Todd's paresis, hypoglycaemia, syncope, transient global amnesia, subdural haematoma, brain tumour, multiple sclerosis, peripheral vestibular disease.
- The **ABCD^2 score does not detect atrial fibrillation or carotid stenosis**, so a low score never justifies omitting vascular and cardiac imaging.
- **Limb-shaking TIA** — brief involuntary jerking of the contralateral limb on standing — indicates critical carotid stenosis with haemodynamic failure and is often misdiagnosed as a focal seizure.
- **ESUS** (embolic stroke of undetermined source): routine anticoagulation is **not** beneficial (NAVIGATE-ESUS, RE-SPECT ESUS) — look harder for atrial fibrillation instead.

Examiner's Tip — Verbatim Mark-Earning Lines

- "A TIA is defined by **absence of infarction on imaging, not by duration of symptoms**."
- "**A TIA is a warning shot — the highest risk of stroke is in the first 48 hours.**"
- "**Antiplatelets for atherothrombotic TIA; anticoagulation for atrial fibrillation** — never confuse the two."

Question 6. Lacunar Stroke 7 MARKS

Answer

Textbook Core – 6 Points

1. **Definition and pathology.** A **small subcortical infarct, less than 15–20 mm in diameter**, resulting from occlusion of a single **deep penetrating artery** (lenticulostriate, thalamoperforating or pontine perforator). It accounts for about **25% of all ischaemic strokes**. The underlying pathology is **lipohyalinosis and microatheroma** of the small vessel, driven above all by **hypertension and diabetes** — not by embolism.
2. **The five classical lacunar syndromes. Pure motor hemiparesis** (posterior limb of the internal capsule or pons — the commonest); **pure sensory stroke** (ventral posterolateral thalamus); **sensorimotor stroke**; **ataxic hemiparesis** (pons or internal capsule); and **dysarthria–clumsy hand syndrome** (pons or genu of the internal capsule).
3. **The defining negatives.** There are **no cortical signs** — **no aphasia, no neglect, no visual field defect, no altered consciousness and no seizure**. In pure motor hemiparesis, the **face, arm and leg are affected equally**. These negatives are what earn marks and what distinguish a lacune from a cortical infarct.
4. **Investigations. MRI with DWI is far superior to CT** for small deep infarcts, which are frequently invisible on CT. Look also for the accompanying markers of **cerebral small-vessel disease**: white matter hyperintensities (leukoaraiosis), **microbleeds on SWI**, enlarged perivascular spaces and old silent lacunes. Still perform ECG, carotid Doppler and echocardiography to exclude a cardioembolic or large-artery source, though the cause is usually intrinsic small-vessel disease.
5. **Treatment. Thrombolysis remains indicated within the time window** — lacunar strokes benefit. **Single antiplatelet therapy is the mainstay**, with a 21-day course of dual therapy only for high-risk minor stroke. Add a **high-intensity statin**, and above all **aggressive blood pressure control** — the SPS3 trial showed that a systolic target below 130 mmHg reduced intracerebral haemorrhage. Control diabetes and stop smoking.
6. **Prognosis and complications.** Early functional recovery is usually good, but there is a **high long-term risk of recurrence, vascular cognitive impairment and vascular parkinsonism**. The accumulated **multilacunar state** produces pseudobulbar palsy, emotional lability, *marche à petits pas*, gait apraxia and falls. **Long-term dual antiplatelet therapy must be avoided** — SPS3 showed no benefit but significantly more bleeding and higher mortality.

Latest Update – 1 Point

7. **AHA/ASA 2026 Acute Ischaemic Stroke Guideline — low NIHSS is no longer a reason to withhold thrombolysis.** The guideline emphasises **rapid thrombolysis for any eligible patient with a disabling deficit within 4.5 hours, regardless of NIHSS score and without waiting for advanced imaging**. This matters specifically for lacunar stroke, which characteristically scores low on the NIHSS yet may be severely disabling (isolated hand weakness, ataxic hemiparesis, dysarthria–clumsy hand). **Tenecteplase 0.25 mg/kg as a single bolus is now a Class 1 alternative to alteplase.** The INSPIRES trial additionally supports starting dual antiplatelet therapy up to 72 hours in minor strokes of atherosclerotic origin.

Examiner's Tip — Verbatim Mark-Earning Lines

- "**A lacunar syndrome is defined as much by what is absent as by what is present** — no aphasia, no neglect, no field defect, no altered consciousness."
- "Blood pressure control is the **single most important intervention** in small-vessel disease."
- "**Do not continue dual antiplatelet therapy long term after a lacunar stroke** — SPS3 showed harm."
- **Skeleton:** Definition/lipohyalinosis → Five syndromes → Cortical-sign negatives → MRI + small-vessel markers → BP, statin, antiplatelet → Recurrence/vascular cognitive impairment → AHA/ASA 2026.

Question 7. Thrombolysis and Mechanical Thrombectomy in Acute Stroke 7 MARKS

Answer

Textbook Core – 6 Points

1. **Principle.** "**Time is brain**" — reperfusion aims to salvage the **ischaemic penumbra** before infarction is complete. A **non-contrast CT to exclude haemorrhage is mandatory** before thrombolysis; the target **door-to-needle time is 60 minutes or less**. Every patient, whether or not eligible for reperfusion, benefits from **stroke unit care**.
2. **Intravenous thrombolysis. Alteplase 0.9 mg/kg (maximum 90 mg; 10% as bolus, remainder over 60 minutes) within 4.5 hours** of onset or last-known-well. The number needed to treat for improved outcome is about **10 when given within 3 hours**. **Age over 80 is not a contraindication** — IST-3 and pooled analyses confirm benefit in the very elderly.
3. **Contraindications and risks.** Intracranial haemorrhage; uncontrolled **BP >185/110 mmHg**; platelets <1,00,000/mm^3; **INR >1.7** or a DOAC taken within 48 hours; blood glucose <50 mg/dL; major surgery or gastrointestinal bleed within 21 days; stroke or head injury within 3 months; known AVM, aneurysm or intracranial neoplasm. The principal hazard is **symptomatic intracerebral haemorrhage in about 2–6%**; orolingual angio-oedema also occurs.
4. **Mechanical thrombectomy — indications.** Anterior circulation **large-vessel occlusion (ICA or M1)**, **NIHSS ≥6**, pre-stroke **mRS 0–1**, **ASPECTS ≥6**, within **6 hours**. The window extends to **6–24 hours with perfusion or clinical–core mismatch selection (DAWN and DEFUSE-3)**. With a **NNT of about 2.6** for reduced disability, it is the most powerful single intervention in acute stroke.
5. **Combined use and post-procedure care.** Patients eligible for both should receive **intravenous thrombolysis and proceed to thrombectomy** — **never delay the procedure to see whether symptoms improve**. After thrombolysis: no antiplatelet or anticoagulant for 24 hours, repeat CT at 24 hours, maintain BP below 180/105 mmHg, intensive neurological observation, early swallow screen and stroke unit admission.
6. **Indian and practical context.** The principal barriers are **delayed presentation, cost of alteplase and a limited number of thrombectomy-capable centres**. **Tenecteplase is single-bolus, cheaper and widely used in India**. Telestroke networks, pre-hospital notification and stroke-ready hospital certification under national programmes are the practical levers for improving access.

Latest Update – 1 Point

7. **AHA/ASA 2026 Guideline for the Early Management of Acute Ischaemic Stroke** (*Stroke*, 2026), replacing the 2018/2019 guidelines. Key changes: **tenecteplase 0.25 mg/kg (maximum 25 mg) as a single bolus is now a Class 1 recommendation, equivalent to alteplase within 4.5 hours**; thrombolyse eligible disabling strokes within 4.5 hours **regardless of NIHSS score and without waiting for advanced imaging**; extended-window thrombolysis at **4.5–9 hours or in wake-up stroke** using perfusion or DWI–FLAIR mismatch is Class 2a; thrombectomy eligibility is broadened to **ASPECTS 3–10**, admitting large-core infarcts; and mobile stroke units are endorsed.

Current Practice Update

Domain	Textbook	Current (AHA/ASA 2026)
Thrombolytic agent	Alteplase 0.9 mg/kg infusion	Alteplase **or** tenecteplase 0.25 mg/kg bolus — both Class 1
Thrombolysis window	0–4.5 hours	0–4.5 h standard; **4.5–9 h / wake-up** with mismatch imaging (Class 2a)
Thrombectomy core size	ASPECTS ≥6	**ASPECTS 3–10** — large-core infarcts now eligible
Thrombectomy window	0–6 hours	Up to **24 hours** with mismatch selection
Exam approach	Write the classical alteplase/6-hour framework first, then add one line: "The AHA/ASA 2026 guideline now gives tenecteplase a Class 1 recommendation and extends thrombectomy to ASPECTS 3–10 and to 24 hours."	

Examiner's Tip — Verbatim Mark-Earning Lines

- "**Age alone is never a contraindication to thrombolysis.**"
- "Thrombectomy has an **NNT of about 2.6** — the most effective acute intervention in medicine."
- "Give thrombolysis and go to thrombectomy — **do not wait to see if the patient improves**."
- **Skeleton:** Penumbra/CT/door-to-needle → Alteplase dose and window → Contraindications and sICH → Thrombectomy criteria and DAWN/DEFUSE-3 → Combined use and post-lysis care → Indian barriers → AHA/ASA 2026.

Question 8. Wallenberg Syndrome — Cause, Clinical Features and Treatment 7 MARKS

Answer

Textbook Core – 6 Points

1. **Definition.** Wallenberg syndrome is the **lateral medullary syndrome** — infarction of the dorsolateral medulla, often with the inferior cerebellum. Despite the name "PICA syndrome", the commonest vessel involved is the **vertebral artery (about 80%)**, with PICA occlusion accounting for the remainder.
2. **Causes in the elderly. Atherothrombosis of the vertebral artery** (commonest), cardioembolism (atrial fibrillation), **vertebral artery dissection** — which may follow neck manipulation, trauma or even trivial neck rotation and should be suspected when there is neck or occipital pain — vasculitis, and hypercoagulable states.
3. **Clinical features — the crossed sensory loss.** Loss of **pain and temperature sensation on the IPSILATERAL face** (spinal trigeminal nucleus and tract) with loss of **pain and temperature on the CONTRALATERAL body** (spinothalamic tract), while **touch, vibration and proprioception are preserved**. This dissociated, crossed pattern is the diagnostic signature.
4. **Other ipsilateral features. Horner's syndrome** — ptosis, miosis, anhidrosis (descending sympathetic fibres); **cerebellar ataxia and lateropulsion** (inferior cerebellar peduncle) — the body is pulled towards the side of the lesion; **vertigo, nystagmus, nausea and vomiting** (vestibular nuclei); **intractable hiccups**; and **dysphagia, hoarseness, absent gag reflex and ipsilateral palatal weakness** (nucleus ambiguus).
5. **The key negative and the practical dangers. There is NO hemiparesis** — the pyramidal tract lies medially and is spared. This is the single most useful discriminator from a hemispheric stroke. Two dangers dominate management: **severe dysphagia out of proportion to the rest of the deficit**, with a very high aspiration risk, and rarely **Ondine's curse** (loss of automatic respiration), which is life-threatening.
6. **Diagnosis and treatment. MRI with DWI is the investigation of choice** — CT frequently misses posterior fossa infarcts; add **CT or MR angiography of the vertebrobasilar system** to identify occlusion or dissection. Treatment: thrombolysis if within the window, stroke-unit care, and above all **aspiration prevention — nil by mouth, formal swallow assessment, nasogastric feeding and early speech-and-language therapy**. Secondary prevention with an antiplatelet (or anticoagulation for cardioembolism), high-intensity statin, and blood pressure and glycaemic control. Treat intractable hiccups (baclofen, chlorpromazine or gabapentin) and central post-stroke pain (amitriptyline or pregabalin); vestibular rehabilitation and physiotherapy for lateropulsion. **Prognosis is generally good**, with recovery over weeks to months.

Latest Update – 1 Point

7. **AHA/ASA 2026 Acute Ischaemic Stroke Guideline — posterior circulation stroke.** Two points are directly relevant. First, **thrombolysis is recommended for any disabling deficit within 4.5 hours regardless of NIHSS score** — important because posterior circulation strokes such as Wallenberg systematically score low on the NIHSS despite causing severe disability and dysphagia. Second, **endovascular thrombectomy is now recommended for basilar artery occlusion** (following the ATTENTION and BAOCHE trials), the condition to which progressive vertebrobasilar disease may lead. The guideline also contains updated recommendations on **dysphagia screening and management**, the dominant practical problem in this syndrome.

Examiner's Tip — Verbatim Mark-Earning Lines

- "**Ipsilateral face, contralateral body** — the crossed pain-and-temperature loss of the lateral medullary syndrome."
- "**Wallenberg syndrome causes no weakness** — that single negative separates it from a hemispheric stroke."
- "The immediate priority is not the drug chart but **the swallow** — keep the patient nil by mouth until formally assessed."
- "Consider **vertebral artery dissection** in any patient with neck pain and a lateral medullary syndrome."
- **Skeleton:** Definition/vertebral artery → Causes → Crossed sensory loss → Horner + ataxia + nucleus ambiguus → No hemiparesis, dysphagia danger → MRI/MRA + swallow + secondary prevention → AHA/ASA 2026.

Question 9. Subarachnoid Haemorrhage 7 MARKS

Answer

Textbook Core – 6 Points

1. **Definition and causes.** Bleeding into the subarachnoid space. About **85% follow rupture of a saccular (berry) aneurysm** — anterior communicating > posterior communicating > middle cerebral bifurcation; **10% are perimesencephalic non-aneurysmal** (benign course); the remainder are due to AVM, trauma, vasculitis, coagulopathy or reversible cerebral vasoconstriction syndrome. Risk factors: **hypertension, smoking, alcohol, family history, adult polycystic kidney disease** and connective tissue disorders.
2. **Clinical features. Sudden "worst headache of my life" (thunderclap headache) peaking within seconds**, with vomiting, neck stiffness, photophobia, transient loss of consciousness and seizures. A **sentinel or warning bleed** precedes the major event in up to 40%. Fundoscopy may show subhyaloid or vitreous (**Terson**) haemorrhage. **In the elderly the presentation is often atypical** — confusion, collapse or coma with less prominent headache and meningism.
3. **Grading. Hunt and Hess** (I — mild headache/nuchal rigidity, through V — deep coma and decerebrate posturing) and the **WFNS scale** (based on GCS plus motor deficit) grade clinical severity; the **modified Fisher scale** grades the volume and distribution of blood on CT and predicts vasospasm.
4. **Diagnosis. Immediate non-contrast CT head** — sensitivity exceeds 95% within the first 6 hours and falls steadily thereafter. If the CT is negative and suspicion persists, perform a **lumbar puncture after 12 hours**, looking for **xanthochromia** (ideally by spectrophotometry) and a red cell count that does not clear between successive bottles. Then **CT angiography or digital subtraction angiography** to define the aneurysm.
5. **Treatment.** Admit to a neurocritical care unit. **Secure the aneurysm within 24 hours** by **endovascular coiling** (preferred — ISAT showed better outcomes) or surgical clipping. Bed rest, analgesia, stool softeners; control blood pressure before the aneurysm is secured (systolic <160 mmHg) while avoiding hypotension; **nimodipine 60 mg orally every 4 hours for 21 days** to prevent delayed cerebral ischaemia; maintain **euvolaemia and normonatraemia**. Antiepileptics only if seizures occur.
6. **Complications. Rebleeding** (peaks in the first 24 hours; the highest-mortality complication); **delayed cerebral ischaemia from vasospasm** (days 4–14); **hydrocephalus** — acute obstructive needing an external ventricular drain, and chronic communicating needing a VP shunt; **hyponatraemia from cerebral salt wasting or SIADH**; neurogenic pulmonary oedema; **stunned myocardium / Takotsubo cardiomyopathy** with ECG changes and raised troponin; seizures; and long-term cognitive and mood impairment. Overall mortality is **30–40%**.

Latest Update – 1 Point

7. **AHA/ASA 2023 Guideline for Aneurysmal Subarachnoid Haemorrhage** (replacing the 2012 guideline). Key positions: treat the ruptured aneurysm **preferably within 24 hours**, aiming for complete obliteration; **early enteral nimodipine is beneficial**; **prophylactic hypervolaemia and haemodynamic augmentation are harmful and should NOT be used (Class 3: Harm)** — maintain euvolaemia instead, and induce hypertension only once symptomatic delayed cerebral ischaemia develops; **routine statin therapy and intravenous magnesium are not recommended**; and outcomes are better when patients are managed in **high-volume centres with dedicated neurocritical care units**, so timely transfer is recommended.

Current Practice Update

Item	Position
Textbook	"Triple-H therapy" — hypertension, hypervolaemia and haemodilution — is used to prevent and treat vasospasm. Statins and magnesium are considered.
Current recommendation	AHA/ASA 2023: **prophylactic hypervolaemia and haemodynamic augmentation are harmful**. Maintain euvolaemia; induce hypertension only for symptomatic delayed cerebral ischaemia. **Routine statins and IV magnesium are not recommended.**
Exam approach	Write the classical account, then add: "Triple-H therapy has been abandoned; the AHA/ASA 2023 guideline recommends euvolaemia, with induced hypertension reserved for established DCI."

Examiner's Tip — Verbatim Mark-Earning Lines

- "**A normal CT does not exclude subarachnoid haemorrhage** — do an LP after 12 hours and look for xanthochromia."
- "**Nimodipine improves outcome without abolishing angiographic vasospasm** — it is neuroprotective, not simply a vasodilator."
- "Rebleeding kills early; **vasospasm kills between days 4 and 14**."
- **Skeleton:** Definition/aneurysm → Thunderclap headache → Hunt-Hess/WFNS/Fisher → CT then LP then angiography → Coiling + nimodipine → Complications → AHA/ASA 2023.

Question 10. Treatment of Symptomatic and Asymptomatic Carotid Disease in the Elderly

7 MARKS

Answer

Textbook Core – 6 Points

1. **Definitions. Symptomatic carotid stenosis** = an ipsilateral stroke, TIA or amaurosis fugax **within the preceding 6 months**. **Asymptomatic** = stenosis detected on imaging or from a bruit, with no such event. Severity is quantified by the **NASCET method** on Doppler ultrasound, CT angiography or MR angiography, with a second modality used to confirm before intervention.
2. **Best medical therapy — for every patient, symptomatic or not. High-intensity statin** (atorvastatin 40–80 mg or rosuvastatin 20–40 mg; LDL target <70 mg/dL, and <55 mg/dL in very-high-risk patients), **antiplatelet therapy** (aspirin 75–150 mg or clopidogrel 75 mg, with a short course of dual therapy after TIA or minor stroke), **blood pressure <130/80 mmHg**, glycaemic control, smoking cessation, exercise and weight management.
3. **Symptomatic disease — revascularisation. Carotid endarterectomy (CEA) is clearly beneficial for 70–99% stenosis** — NASCET showed an absolute risk reduction of about 16% at 2 years (**NNT approximately 6**). Benefit for **50–69% stenosis is moderate** and greatest in **men, patients over 75, and hemispheric rather than retinal events**. There is **no benefit, and possible harm, below 50%**; near-occlusion and total occlusion are not revascularised.
4. **Timing and quality standards.** Revascularise **within 2 weeks of the index event** — benefit falls sharply thereafter, and delaying "to let things settle" is wrong. The procedure is only worthwhile if the unit's **perioperative stroke or death rate is below 6% for symptomatic and below 3% for asymptomatic disease**.
5. **Endarterectomy versus stenting in older adults. CEA is preferred in the elderly.** The CREST trial showed **significantly higher periprocedural stroke with stenting, particularly above the age of 70**, because of aortic arch atheroma and vessel tortuosity, while CEA carried a modestly higher risk of myocardial infarction. **Stenting is reserved for a hostile neck** (previous surgery or radiotherapy), surgically inaccessible high lesions, restenosis, or severe cardiorespiratory disease precluding anaesthesia.
6. **Asymptomatic disease — the geriatric decision.** The classical trials (ACAS, ACST-1) showed only about **1% absolute benefit per year**, and **only if life expectancy exceeds 5 years and the operative risk is under 3%**; that margin has narrowed further as medical therapy has improved. In a frail older patient with limited life expectancy, dementia or high operative risk, **best medical therapy alone is the correct answer** — the decision must follow a **comprehensive geriatric assessment, not the stenosis percentage alone**.

Latest Update – 1 Point

7. **CREST-2 (*NEJM*, 21 November 2025) — a paradigm shift in asymptomatic carotid stenosis.** Two parallel randomised, observer-blinded trials in **2,485 patients with asymptomatic stenosis ≥70%**, all receiving protocol-driven intensive medical therapy. **Stenting plus intensive medical therapy reduced the 4-year primary outcome (2.8% vs 6.0%, p=0.02), whereas endarterectomy showed no significant benefit over intensive medical therapy alone (3.7% vs 5.3%, p=0.24)**. Rates of disabling stroke were low in every group. Taken with SPACE-2 and ECST-2, accompanying editorials concluded there is **no longer a routine role for carotid endarterectomy in asymptomatic stenosis**, and advised starting intensive medical therapy immediately and deferring revascularisation until symptoms develop. **CREST-2 does not alter the strong indication for CEA in symptomatic 70–99% stenosis.**

Current Practice Update

Item	Position
Textbook	Carotid endarterectomy is offered for asymptomatic stenosis ≥60–70% when life expectancy exceeds 5 years and surgical risk is <3% (ACAS, ACST-1). Stenting is inferior in the elderly.
Current recommendation	**CREST-2 (2025):** against modern intensive medical therapy, **CEA adds no significant benefit in asymptomatic disease**; stenting gave a small absolute benefit. Intensive medical therapy first, and defer revascularisation until symptoms occur. **Symptomatic 70–99% stenosis is unchanged — CEA within 2 weeks.**
Exam approach	Answer symptomatic and asymptomatic disease separately, then close: "CREST-2 (NEJM 2025) has largely removed the routine role of endarterectomy in asymptomatic stenosis; intensive medical therapy is now the default."

Examiner's Tip — Verbatim Mark-Earning Lines

- "**Symptomatic 70–99% stenosis: endarterectomy within 2 weeks, NNT about 6.**"
- "**Asymptomatic stenosis is a medical, not a surgical, disease** in the modern era."
- "**CREST: stenting carries a higher periprocedural stroke risk in patients over 70** — endarterectomy is preferred in the elderly."
- "The decision in an older patient depends on **life expectancy and frailty, not on the stenosis figure**."

Question 11. Subdural Haematoma in the Elderly 7 MARKS

Answer

Textbook Core – 6 Points

1. **Definition and pathogenesis.** A collection of blood in the **subdural space** caused by tearing of the **bridging cortical veins**. **Cerebral atrophy in old age stretches these veins and widens the subdural space**, so a trivial or entirely unremembered head injury is sufficient. Classified as **acute (<3 days, hyperdense), subacute (3 days–3 weeks, isodense) and chronic (>3 weeks, hypodense)**.
2. **Risk factors.** Age over 65, brain atrophy, **recurrent falls**, alcohol excess, **anticoagulants and antiplatelet drugs**, previous subdural haematoma, epilepsy, coagulopathy, chronic liver disease, haemodialysis, and **CSF over-drainage after shunting**.
3. **Clinical presentation — "the great imitator". Fluctuating consciousness**, headache, gradual cognitive decline mimicking dementia, personality change, gait disturbance and falls, hemiparesis, dysphasia, or simply **delirium and unexplained functional decline**. A history of trauma is **absent in up to half of cases**, so a high index of suspicion is essential.
4. **Diagnosis. Non-contrast CT head** is the first-line investigation. Beware the **isodense subacute phase and bilateral collections**, where midline shift may be absent — look instead for **effaced sulci, compressed ventricles and loss of grey-white differentiation**. MRI is more sensitive when CT is equivocal. Check INR, coagulation profile and platelet count.
5. **Management. Reverse anticoagulation urgently** — vitamin K with **4-factor prothrombin complex concentrate** for warfarin, **idarucizumab** for dabigatran, and andexanet alfa or PCC for factor Xa inhibitors. Surgery: **burr-hole drainage with a subdural drain** for symptomatic chronic SDH (the drain reduces recurrence), and **craniotomy** for acute or organised clot. Small, asymptomatic collections may be managed conservatively with serial imaging.
6. **Complications and prognosis. Recurrence in 10–30%**; seizures, infection, tension pneumocephalus, venous thromboembolism and prolonged rehabilitation. Outcome depends more on the **GCS at presentation and premorbid frailty** than on the size of the clot. Restarting anticoagulation must be individualised — usually about **4 weeks**, earlier if there is a mechanical valve.

Latest Update – 1 Point

7. **Middle meningeal artery embolisation (MMAE).** Three randomised trials published together in the *New England Journal of Medicine* on **20 November 2024** — **EMBOLISE (n=400), STEM (n=310) and MAGIC-MT (n=722)** — showed that adjunctive MMAE **significantly reduces recurrence or progression of chronic subdural haematoma requiring reoperation**, without increasing disability. MMAE devascularises the inflammatory neomembranes that drive re-bleeding, and is now an established adjunct to surgery — **especially valuable in older, anticoagulated, high-surgical-risk patients**.

Current Practice Update

Item	Position
Textbook	Chronic SDH is managed by burr-hole drainage or craniotomy; recurrence in 10–30% is accepted and re-operated.
Current recommendation	Add **middle meningeal artery embolisation** as an adjunct to surgery (or as stand-alone treatment in selected non-surgical patients) to reduce recurrence — three RCTs, NEJM 2024.
Exam approach	After writing the surgical options, add: "Since the EMBOLISE, STEM and MAGIC-MT trials (NEJM 2024), MMAE is an evidence-based adjunct that reduces recurrence."

Examiner's Tip — Verbatim Mark-Earning Lines

- "Chronic subdural haematoma is a **reversible cause of dementia and delirium** in the elderly — CT the fluctuating patient."
- "**Absence of a history of head injury does not exclude the diagnosis.**"
- "Bilateral isodense collections may show **no midline shift** — look for effaced sulci and compressed ventricles."
- **Skeleton:** Bridging veins + atrophy → Risk factors → The great imitator → CT pitfalls → Reversal + burr hole → Recurrence/prognosis → MMAE trials.

Question 12. Post-Stroke Depression and Post-Stroke Pain 7 MARKS

Answer

Textbook Core – 6 Points

1. **Post-stroke depression (PSD) — definition and burden.** Depressive disorder occurring after stroke, affecting about **one-third of survivors** and peaking at **3–6 months**. Predictors are stroke severity, physical disability, aphasia, cognitive impairment, previous depression, female sex and social isolation. PSD independently causes **poorer functional recovery, reduced participation in rehabilitation and increased mortality**.
2. **Recognition of PSD.** Detection is difficult because of aphasia, apathy, emotionalism and anosognosia. Distinguish PSD from **apathy** (loss of motivation without sadness), **pseudobulbar affect / emotionalism** (pathological crying or laughing) and normal adjustment reaction. Screen with **PHQ-9, Geriatric Depression Scale or HADS**, and the Aphasic Depression Rating Scale in aphasic patients — at admission, at discharge and at follow-up.
3. **Management of PSD. SSRIs are first line** — escitalopram, sertraline or citalopram; **avoid tricyclics** because of anticholinergic burden, falls and arrhythmia. Watch for **hyponatraemia/SIADH, gastrointestinal bleeding (especially with antiplatelets), falls and fractures, and QT prolongation with citalopram**. Non-drug measures: structured psychosocial support, CBT or problem-solving therapy, early mobilisation, exercise, and treatment of pain and insomnia. Continue treatment at least 6 months after remission.
4. **Central post-stroke pain (Dejerine–Roussy thalamic pain).** Neuropathic pain in the body region corresponding to the lesion, typically after **thalamic or lateral medullary** strokes, appearing weeks to months later. It is **burning, aching or lancinating, with allodynia, hyperalgesia and dysaesthesia**, worsened by cold, touch and emotion. Treatment: **amitriptyline** (with caution in the elderly), **pregabalin or gabapentin**, lamotrigine or duloxetine. **Simple analgesics and opioids are largely ineffective.**
5. **Hemiplegic shoulder pain and other nociceptive post-stroke pain.** Caused by **glenohumeral subluxation, rotator cuff injury, adhesive capsulitis and spasticity**. **Prevention is by correct positioning, arm support with a lap tray or sling, and never pulling on the flaccid arm during transfers.** Treat with physiotherapy, analgesia, intra-articular steroid injection, and **botulinum toxin** for spasticity-related pain. Also consider shoulder–hand syndrome (complex regional pain syndrome) and post-stroke headache.
6. **Geriatric principles.** Mood and pain interact — **untreated pain worsens mood, sleep and rehabilitation outcome, and untreated depression amplifies pain**. Assess pain in aphasic or cognitively impaired patients with observational tools (**PAINAD, Abbey Pain Scale**). Apply “start low, go slow”, review anticholinergic burden and drug interactions, and remember that **multidisciplinary rehabilitation with early mobilisation is the single most effective intervention for both problems**.

Latest Update – 1 Point

7. **VA/DoD Clinical Practice Guideline for Stroke Rehabilitation, 2024 update.** It now **recommends against the routine use of antidepressants for the prevention of post-stroke depression** — a change from “neither for nor against” in 2019 to “weak against”. This follows the FOCUS, AFFINITY and EFFECTS trials and their **individual-patient-data meta-analysis (*Int J Stroke* 2024)**, which showed that fluoxetine 20 mg daily for 6 months **did not improve functional recovery** and **increased seizures, falls with injury and bone fractures**, although it did reduce the frequency of depression. The message is: **treat established post-stroke depression, but do not prescribe antidepressants prophylactically.**

Current Practice Update

Item	Position
Textbook	Early SSRIs after stroke may prevent depression and enhance motor recovery (based on FLAME and earlier small trials).
Current recommendation	Routine prophylactic antidepressants are **not** recommended; fluoxetine does not aid functional recovery and increases fractures, falls and seizures. Treat only established depression.
Exam approach	State: “SSRIs remain first-line *treatment* for post-stroke depression, but the 2024 VA/DoD guideline advises against their use for *prevention*.”

Examiner’s Tip — Verbatim Mark-Earning Lines

- “Post-stroke depression affects **about one-third of survivors** and independently worsens functional outcome and mortality.”
- “Central post-stroke pain responds to **antidepressants and anticonvulsants, not to analgesics**.”
- “**Never pull on the hemiplegic arm** — shoulder pain in stroke is largely preventable.”
- **Skeleton:** PSD burden/predictors → Recognition and screening → SSRI treatment and cautions → Central post-stroke pain → Hemiplegic shoulder → Geriatric principles → VA/DoD 2024.

Question 13. Normal Pressure Hydrocephalus 7 MARKS

Answer

Textbook Core – 6 Points

1. **Definition.** NPH is a syndrome of **gait disturbance, cognitive impairment and urinary incontinence** (Hakim–Adams triad) with **ventriculomegaly** but a **normal CSF opening pressure**. It is **idiopathic** in most older people, or **secondary** to subarachnoid haemorrhage, head injury, meningitis or infarction.
2. **Gait disturbance** is the earliest and most important feature. It is **magnetic or "glue-footed"** with reduced stride length, reduced step height, broad base and poor balance. Unlike Parkinson's disease, it does **not respond to external cues or to levodopa**.
3. **Cognitive impairment** is **frontal-subcortical** — inattention, slowed psychomotor speed, impaired recall more than recognition, and visuospatial dysfunction. Memory and orientation are relatively preserved, which separates it from Alzheimer's disease.
4. **Urinary dysfunction** begins as frequency and urgency and progresses to urge incontinence and then total incontinence as frontal control is lost. Mechanisms are a **small bladder capacity (mean 200 mL)** and a **hyperactive detrusor**. Faecal incontinence occurs late.
5. **Diagnosis.** CT/MRI shows ventricular enlargement out of proportion to atrophy (**Evans index >0.3**, DESH pattern). Patient selection is crucial: a positive **CSF tap test**, **external lumbar drainage** or raised **CSF outflow resistance** predicts shunt response. **Age is not a prognostic factor.**
6. **Treatment. Ventriculoperitoneal shunting** is standard care and succeeds in about **80%** of well-selected patients — gait improves in 81–86%, bladder control in 70%, cognition in only 40–44%. Serious adverse event risk is **11%**: shunt infection (a neurosurgical emergency), subdural effusion or haematoma, obstruction, over-drainage and hearing loss.

Latest Update – 1 Point

7. **PENS trial (NEJM 2025)** — the first **double-blind, placebo-controlled** trial of shunting in iNPH (n=99; open valve 110 mmH_2O vs placebo setting >400 mmH_2O). At 3 months, shunting significantly improved **gait velocity and Tinetti gait-and-balance score**, but showed **no significant benefit in cognition (MoCA) or incontinence**. This confirms that **gait is the shunt-responsive domain** and that response to temporary CSF drainage should drive patient selection.

Current Practice Update

Item	Position
Textbook	Shunting improves gait *and* urinary incontinence in most patients; cognition less consistently.
Current recommendation	Placebo-controlled evidence (PENS 2025) confirms benefit for **gait and balance only** at 3 months; cognition and continence were not significantly improved.
Exam approach	Quote the classical 80% success figure, then add that PENS 2025 has now placed shunting on randomised evidence with gait as the primary responsive domain.

Examiner's Tip — Verbatim Mark-Earning Lines

- "NPH is the only potentially **reversible cause of dementia** presenting with the triad of gait apraxia, cognitive slowing and urinary incontinence."
- "**Wet, wacky and wobbly** — but the wobble comes first and the wobble responds best."
- "NPH gait does not respond to visual cues or levodopa; Parkinsonian gait does."
- **Skeleton:** Definition/triad → Gait → Cognition → Bladder → Imaging + tap test → VP shunt and complications → PENS 2025.

Question 14. Progressive Supranuclear Palsy and Corticobasal Degeneration 7 MARKS

Answer

Textbook Core – 6 Points

1. **Definition.** Both are **Parkinson-plus (atypical parkinsonian) syndromes** and are **4-repeat tauopathies**. Both are frequently misdiagnosed as Parkinson's disease, and both show a **poor or absent response to levodopa**, which is the single most useful clinical discriminator.
2. **PSP — clinical features. Early postural instability with repeated backward falls** within the first year, **vertical supranuclear gaze palsy** (inability to look voluntarily downward, impaired saccades, eyelid-opening apraxia), **axial rigidity greater than limb rigidity**, retrocollis, and a startled or astonished facies. **Tremor is infrequent.**
3. **PSP — course and investigations.** Personality change, subcortical dementia and swallowing difficulty develop as the disease progresses. MRI shows **midbrain atrophy** — the normally concave upper midbrain becomes convex (**hummingbird sign**). Ocular electrophysiology demonstrates impaired saccades.
4. **CBD — clinical features.** A strikingly **asymmetrical akinetic-rigid syndrome** with limb **dystonia, myoclonus, cortical sensory loss, ideomotor apraxia** and the **alien (foreign) hand phenomenon**. MRI shows asymmetrical fronto-parietal atrophy contralateral to the affected limb.
5. **Differentiation from other causes of parkinsonism in old age.** Lewy body dementia — dementia within a year of parkinsonism, hallucinations, neuroleptic sensitivity. Multiple system atrophy — early severe dysautonomia with cerebellar or pyramidal signs. Vascular parkinsonism — lower-body predominance, multilacunar state, pseudobulbar palsy, stepwise worsening. NPH — magnetic gait with ventriculomegaly.
6. **Management is symptomatic; no disease-modifying therapy exists.** Trial levodopa (response usually poor or transient); amantadine may help. **Botulinum toxin** for limb dystonia, blepharospasm and sialorrhoea. Priorities are **falls prevention**, physiotherapy, weighted walkers, prism glasses, speech and swallow therapy, PEG feeding for aspiration, and **early advance care planning**. Avoid antipsychotics. Median survival is about **6–8 years**; death is usually from aspiration pneumonia.

Latest Update – 1 Point

7. **Tau-directed therapy has reached phase 3 in PSP (2024–2026).** Two agents have advanced to phase 3 — **NIO752**, an intrathecal **antisense oligonucleotide against the tau gene MAPT**, and **LM11A-31**, a p75 neurotrophin-receptor modulator. The NIH-funded **PSP Platform Trial** (UCSF, from 2025) is testing LM11A-31, the anti-tau vaccine AADvac1 and AZP2006 concurrently, and **tau-PET (^{18}F-PI-2620)** plus fluid biomarkers are being validated for early diagnosis. **No disease-modifying drug is yet approved** — management remains supportive.

Examiner's Tip — Verbatim Mark-Earning Lines

- "**Falls first, tremor never, levodopa fails** — think PSP."
- "**Asymmetry, apraxia and the alien hand** — think corticobasal degeneration."
- "PSP: **axial rigidity with vertical supranuclear gaze palsy and midbrain atrophy (hummingbird sign)**."
- **Skeleton:** Both tauopathies, levodopa-unresponsive → PSP features → PSP imaging → CBD features → Differential table → Symptomatic management → Tau trials.

Question 15. Motor Neuron Disease — Presentation, Evaluation and Treatment 7 MARKS

Answer

Textbook Core – 6 Points

1. **Definition and classification.** Progressive degeneration of **upper and lower motor neurons**. Variants: **amyotrophic lateral sclerosis** (commonest, UMN + LMN), **progressive bulbar palsy**, **progressive muscular atrophy** (pure LMN) and **primary lateral sclerosis** (pure UMN). About 90% are sporadic; familial forms involve **C9orf72, SOD1, TARDBP (TDP-43) and FUS**. The neuropathological hallmark of most cases is cytoplasmic **TDP-43 inclusions**.
2. **Presentation.** Painless, progressive, **asymmetrical weakness with wasting, fasciculations and cramps**, with **brisk reflexes in a wasted limb** (the mixed UMN–LMN signature). **Bulbar onset** — dysarthria, dysphagia, tongue wasting and fasciculation, nasal regurgitation — is commoner in older adults and carries a worse prognosis. There is **no sensory loss and no sphincter or extraocular involvement**.
3. **Cognitive and behavioural change** occurs in up to half of patients; **10–15% develop frank frontotemporal dementia** (ALS–FTSD revised criteria). The **ECAS** is the validated screen for motor-impaired patients. The frontotemporal syndrome is associated with **poor survival** and heavy caregiver burden.
4. **Evaluation.** Diagnosis is clinical plus electrophysiological, using the **El Escorial / revised Awaji criteria**. **EMG** shows active and chronic denervation in at least three of four regions (bulbar, cervical, thoracic, lumbosacral). MRI brain and whole spine excludes structural mimics; **CSF and serum neurofilament** serve as diagnostic and prognostic biomarkers.
5. **Exclude the treatable mimics** — a key examination point, since **many MND-mimic disorders are eminently treatable**: cervical spondylotic myelopathy, **multifocal motor neuropathy with conduction block**, myasthenia gravis, inclusion body myositis, Kennedy's disease, thyrotoxicosis and vitamin B12 deficiency.
6. **Treatment. Riluzole** (anti-glutamatergic, acts on Na^+/K^+ currents) is the only established oral disease-modifying drug — it prolongs survival by a median of about **3 months** and raised one-year survival from 49% to 58%; monitor liver enzymes. **Non-invasive ventilation prolongs survival and quality of life**; **PEG feeding** is offered for dysphagia and weight loss. Symptom control: glycopyrrolate/amitriptyline or botulinum toxin for sialorrhoea, baclofen for spasticity, quinine for cramps. **Multidisciplinary clinic care independently improves survival**, and early palliative care with advance directives is essential.

Latest Update – 1 Point

7. **Tofersen — the first gene-targeted therapy in ALS.** Tofersen is an **intrathecal antisense oligonucleotide** that reduces SOD1 protein synthesis, approved for **SOD1-mutation ALS** (about 2% of cases) under accelerated approval based on reduction of plasma neurofilament light chain. Long-term VALOR and open-label extension data (**JAMA Neurology 2026**) showed slowed functional decline and prolonged survival with **early initiation**. The **EAN 2024 ALS guideline** accordingly recommends **genetic testing for all patients with ALS**, alongside riluzole, early NIV and multidisciplinary care.

Examiner's Tip — Verbatim Mark-Earning Lines

- "**Mixed upper and lower motor neuron signs in the same limb, with no sensory loss**, is MND until proved otherwise."
- "Riluzole and non-invasive ventilation are the only two interventions that prolong survival."
- "Always exclude the treatable mimics — especially **cervical myelopathy and multifocal motor neuropathy**."
- **Skeleton:** Definition/variants → Presentation → Cognitive overlap → EMG/El Escorial → Mimics → Riluzole/NIV/PEG/MDT → Tofersen + EAN 2024.

Question 16. Restless Legs Syndrome 7 MARKS

Answer

Textbook Core – 6 Points

1. **Definition and diagnostic criteria (IRLSSG — all five essential; mnemonic URGE).** (i) **Urge to move the legs**, usually with unpleasant crawling or creeping sensations; (ii) begins or worsens at **Rest**; (iii) partially or completely **Relieved by movement**; (iv) worse in the **Evening or night**; (v) not solely explained by another condition (leg cramps, positional discomfort, neuropathy, akathisia, arthritis).
2. **Classification and mechanism. Primary** — idiopathic, often familial (BTBD9, MEIS1), earlier onset. **Secondary** — **iron deficiency, chronic kidney disease and dialysis, diabetes and peripheral neuropathy, Parkinson's disease, rheumatoid arthritis** and pregnancy. The core pathophysiology is **brain iron deficiency with dopaminergic dysfunction**.
3. **Aggravating drugs — a high-yield geriatric point. Antihistamines** (including sedating antiemetics), **metoclopramide and prochlorperazine**, antipsychotics, **SSRIs, SNRIs, mirtazapine and tricyclics**, and lithium; plus **caffeine, alcohol and nicotine**. **Always review the drug chart before starting new treatment.**
4. **Consequences in the elderly.** Sleep-onset insomnia and non-restorative sleep, daytime fatigue, depression, and **nocturnal wandering with falls and confusion**. It is commonly misdiagnosed as anxiety, akathisia, nocturnal cramps or agitation in dementia, and it worsens the burden of coexisting cardiovascular disease.
5. **Investigations.** The diagnosis is **clinical**. Measure **serum ferritin and transferrin saturation** (morning, fasting sample), renal function, blood glucose and TSH. **Periodic limb movements of sleep** accompany RLS in about 80% of patients; polysomnography is needed only if the diagnosis is uncertain or obstructive sleep apnoea is suspected.
6. **Treatment.** Treat the cause and **withdraw offending drugs**. Non-pharmacological: sleep hygiene, moderate regular exercise, leg massage, avoidance of caffeine, alcohol and nicotine. **Iron replacement** if iron stores are low. Drugs: **alpha-2-delta ligands — gabapentin, gabapentin enacarbil, pregabalin** (sedation, dizziness, ataxia, ankle oedema; reduce dose in renal impairment); **dopamine agonists** (pramipexole, ropinirole) which cause **augmentation, rebound and impulse-control disorders**; and low-dose opioids for refractory disease.

Latest Update – 1 Point

7. **AASM Clinical Practice Guideline, J Clin Sleep Med 2025 — a paradigm shift. Dopamine agonists are no longer recommended as first-line therapy** because long-term harm from **augmentation** outweighs short-term benefit. **Alpha-2-delta ligands (gabapentin, gabapentin enacarbil, pregabalin) and iron therapy (oral ferrous sulfate or intravenous iron) are now first line**, targeting **serum ferritin >100 ng/mL and transferrin saturation >20%**. Extended-release oxycodone/opioids and **bilateral high-frequency peroneal nerve stimulation** are conditionally recommended additional options.

Current Practice Update

Item	Position
Textbook	Dopamine agonists (pramipexole, ropinirole) are first-line drug therapy for moderate-to-severe RLS; iron replacement only if ferritin is frankly low.
Current recommendation	AASM 2025: alpha-2-delta ligands and iron are first line; dopamine agonists are demoted because of augmentation in 50–70% of long-term users. Ferritin target is **>100 ng/mL**, not merely "normal".
Exam approach	Write the classical treatment ladder, then state: "The AASM 2025 guideline has replaced dopamine agonists with alpha-2-delta ligands and iron as first-line therapy because of augmentation."

Examiner's Tip — Verbatim Mark-Earning Lines

- "RLS is a **clinical diagnosis based on the five IRLSSG criteria** — urge, rest, relief, evening, and exclusion of mimics."
- "**Augmentation** — earlier onset, greater intensity and spread to the arms — is the defining long-term complication of dopamine agonists."
- "In an older patient with RLS, the **drug chart is the first investigation**."
- **Skeleton:** URGE criteria → Primary vs secondary → Drug triggers → Geriatric consequences → Ferritin/TSAT → Treatment ladder → AASM 2025.

Question 17. Benign Paroxysmal Positional Vertigo — Diagnosis and Epley Manoeuvre

7 MARKS

Answer

Textbook Core – 6 Points

1. **Definition and mechanism.** BPPV is the **commonest cause of recurrent vertigo in older adults** — brief episodic vertigo provoked by a change in head position relative to gravity, caused by **displaced otoconia from the utricular macula entering a semicircular canal**. **Canalolithiasis** (free-floating debris) is commoner than **cupulolithiasis** (debris adherent to the cupula). The **posterior canal is involved in 85–90%**, the horizontal canal in 5–15%, and the anterior canal rarely.
2. **Clinical features.** Sudden spinning vertigo lasting **less than one minute**, triggered by turning over in bed, lying down, sitting up, bending forward or looking up ("top-shelf vertigo"), with nausea and residual unsteadiness between attacks. **There is no hearing loss, tinnitus or neurological deficit.** In older people it more often presents as **falls, unsteadiness and fear of falling** than as classical rotatory vertigo.
3. **Diagnosis — the Dix–Hallpike manoeuvre** (posterior canal). The patient sits upright, the head is rotated **45° towards the ear being tested**, and the patient is laid rapidly supine with the head extended **20–30° below the horizontal**. A positive test shows, after a **latency of 1–5 seconds**, **up-beating torsional (geotropic) nystagmus lasting less than 60 seconds**, which **reverses on sitting up** and **fatigues on repetition**. The **supine roll (Pagnini–McClure) test** diagnoses horizontal-canal BPPV.
4. **Red flags for central positional vertigo.** Nystagmus with **no latency, no fatigability**, that is **purely vertical or down-beating** or direction-changing; disproportionate truncal ataxia; other brainstem or cerebellar signs; new headache; or vertigo unresponsive to repositioning. **These require urgent MRI to exclude posterior fossa stroke or tumour.**
5. **The Epley (canalith repositioning) manoeuvre — four steps, each held about 30 seconds or until nystagmus stops.** (a) Dix–Hallpike position with the head turned 45° to the affected side; (b) rotate the head **90° to the opposite side** while supine; (c) roll the patient onto that shoulder and turn the head a further 90° so the **nose points to the floor**; (d) sit the patient upright with the chin tucked. **Success is 80–90% in one or two sessions.** Alternatives: **Semont liberatory manoeuvre**; **Lempert barbecue roll** for horizontal canal; **Brandt–Daroff exercises** for home use.
6. **Precautions and drug therapy in the elderly.** Before repositioning, check for **cervical spondylosis, severe kyphosis, vertebrobasilar insufficiency, carotid disease, retinal detachment or recent neck surgery**, and modify with a shoulder roll and reduced neck extension. **Vestibular sedatives — betahistine, prochlorperazine, cinnarizine — do not treat BPPV**; they delay central compensation and cause drowsiness, falls and drug-induced parkinsonism, so they should not be continued beyond 24–48 hours. Recurrence occurs in 15–50%.

Latest Update – 1 Point

7. **Vitamin D supplementation reduces BPPV recurrence.** Randomised and meta-analytic evidence from 2024–2025 — including a randomised controlled trial in older adults (*Otolaryngol Head Neck Surg* 2025), a placebo-controlled RCT (*Laryngoscope Investig Otolaryngol* 2024) and a systematic review (*Nutrients* 2024) — shows that **vitamin D supplementation significantly lowers the recurrence rate of BPPV in patients with serum 25(OH)D below 20 ng/mL**. Recognised recurrence risk factors are advanced age, female sex, hypertension, diabetes, hyperlipidaemia, **osteoporosis and vitamin D deficiency**. Therefore **check vitamin D in recurrent BPPV and replace it** — a cheap intervention that simultaneously addresses osteoporosis and falls risk.

Current Practice Update

Item	Position
Textbook	BPPV is treated with repositioning manoeuvres; recurrence is accepted and simply re-treated.
Current recommendation	Add **secondary prevention**: measure serum 25(OH)D in recurrent BPPV and supplement if deficient, which significantly reduces recurrence.
Exam approach	Close the answer with: "In recurrent BPPV, correct vitamin D deficiency — recent RCT evidence shows a significant reduction in recurrence."

Examiner's Tip — Verbatim Mark-Earning Lines

- "**Latency, torsional up-beating nystagmus, duration <60 seconds, reversal and fatigability** — the five features of a positive Dix–Hallpike test."
- "BPPV is **diagnosed by a manoeuvre and treated by a manoeuvre, not by a drug**."
- "In the elderly, BPPV presents as **falls, not as vertigo**."
- **Skeleton:** Definition/canalolithiasis → Symptoms → Dix–Hallpike → Central red flags → Epley in four steps → Precautions and why vestibular sedatives are wrong → Vitamin D.

Question 18. Myasthenia Gravis in the Older Adult 7 MARKS

Answer

Textbook Core – 6 Points

1. **Definition.** An **antibody-mediated disorder of the neuromuscular junction** in which antibodies against the postsynaptic **nicotinic acetylcholine receptor (AChR, ~85%)**, or against **MuSK** or **LRP4**, impair neuromuscular transmission. **Late-onset MG (>50 years) is increasing in incidence**, shows a male predominance, is usually AChR-antibody positive, and is associated with thymoma in 10–15%.
2. **Clinical features. Fatigable, fluctuating weakness** that worsens through the day and with repeated use. Ocular onset in more than half — **ptosis and diplopia**; bulbar features (dysphagia, nasal regurgitation, nasal speech, jaw fatigue on chewing); proximal limb and neck-extensor weakness ("dropped head"); and respiratory muscle weakness. **Reflexes are normal and there is no sensory loss.**
3. **Geriatric pitfall — MG is under-diagnosed in the elderly.** It is mislabelled as stroke, bulbar palsy, motor neuron disease, myopathy or simply "old age". Older patients present more often with **bulbar and respiratory involvement**. **Consider MG in any older person with ptosis, diplopia, dysphagia or unexplained fatigable weakness**, because it is a highly treatable disease.
4. **Diagnosis. Serum AChR antibody** first; test **MuSK antibody** if negative. **Repetitive nerve stimulation** shows a decremental response of more than 10%; **single-fibre EMG** is the most sensitive test. The **ice-pack test** and the sleep test are safe bedside tests for ptosis and are preferred to edrophonium in older people. **CT/MRI thorax is mandatory to exclude thymoma.** Screen for associated thyroid and other autoimmune disease.
5. **Treatment. Pyridostigmine** for symptomatic relief (cholinergic side effects — colic, diarrhoea, bradycardia, secretions). **Prednisolone** is the mainstay immunotherapy — **start low and increase slowly** to avoid the early steroid-induced deterioration. Steroid-sparing agents: **azathioprine** (check TPMT), mycophenolate, methotrexate. **Thymectomy** is indicated for thymoma at any age; in non-thymomatous MG it is usually reserved for generalised, AChR-positive patients under 50–60 years, and benefit appears only after 6–12 months.
6. **Crisis, precautions and prognosis. Myasthenic crisis** (weakness needing ventilation) is precipitated by infection, surgery or too-rapid reduction of immunotherapy; the rarer **cholinergic crisis** follows excessive anticholinesterase — if in doubt, ventilate and stop the anticholinesterase. Treat with **IVIG or plasma exchange** plus aggressive treatment of infection. **Avoid aminoglycosides, macrolides, fluoroquinolones, beta-blockers, magnesium, procainamide and iodinated contrast.** Prognosis is good — **more than 90% achieve remission or substantial improvement**.

Latest Update – 1 Point

7. **Targeted biologics have transformed refractory generalised MG.** Two classes are now available: **complement C5 inhibitors** — eculizumab, ravulizumab and zilucoplan (meningococcal vaccination is mandatory) — and **FcRn blockers** that accelerate IgG catabolism — efgartigimod, rozanolixizumab, and **nipocalimab, approved by the FDA in April 2025 and the European Commission in December 2025** for AChR- or MuSK-antibody-positive generalised MG. These give rapid, steroid-sparing IgG reduction (about 70% fall) and are particularly relevant in older adults who tolerate long-term corticosteroids poorly.

Current Practice Update

Item	Position
Textbook	Refractory MG is managed with escalating conventional immunosuppression, IVIG/plasma exchange, and eculizumab as the sole biologic option.
Current recommendation	A defined biologic pathway now exists: C5 inhibitors (eculizumab, ravulizumab, zilucoplan) and FcRn blockers (efgartigimod, rozanolixizumab, nipocalimab) for AChR/MuSK-positive generalised MG.
Exam approach	Finish the treatment paragraph with one line: "In refractory disease, complement C5 inhibitors and FcRn blockers are now established steroid-sparing options."

Examiner's Tip — Verbatim Mark-Earning Lines

- "MG incidence **rises with age but is under-diagnosed in the elderly**, where it is mistaken for stroke or MND."
- "**Fatigable ptosis, diplopia and bulbar weakness with normal reflexes and no sensory loss.**"
- "Single-fibre EMG is the most sensitive test; the **ice-pack test** is the safest bedside test in old age."
- **Skeleton:** Definition/antibodies → Fatigability → Geriatric under-diagnosis → Diagnosis + thymoma imaging → Pyridostigmine/steroids/thymectomy → Crisis and drugs to avoid → Biologics.

Question 19. Subacute Combined Degeneration of the Cord 7 MARKS

Answer

Textbook Core – 6 Points

1. **Definition.** A **vitamin B12 (cobalamin) deficiency myelopathy** causing symmetrical demyelination of the **dorsal (posterior) columns and lateral corticospinal tracts**, chiefly in the cervical and upper thoracic cord. It is frequently combined with a **peripheral neuropathy**, and occasionally with optic atrophy and dementia — hence "combined".
2. **Causes in the elderly. Pernicious anaemia** (autoimmune atrophic gastritis), **food-cobalamin malabsorption from atrophic gastritis** (the commonest cause in old age), gastrectomy or bariatric surgery, ileal resection or Crohn's disease, **chronic metformin and proton-pump inhibitor use**, strict vegetarian or vegan diet (highly relevant in India), tropical sprue and nitrous oxide exposure.
3. **Clinical features.** Symmetrical distal **paraesthesiae and numbness** first, then **sensory ataxia with a positive Romberg sign** and loss of vibration and joint-position sense. The classical examination finding is a **spastic paraparesis with extensor plantar responses but absent ankle jerks** — pyramidal signs coexisting with an abolished reflex because of the accompanying neuropathy. Lhermitte's phenomenon may occur.
4. **Systemic and neuropsychiatric features.** Macrocytic **megaloblastic anaemia**, glossitis, angular stomatitis and skin hyperpigmentation; and "**megaloblastic madness**" — irritability, depression, psychosis and reversible dementia. Critically, **neurological disease can occur with a normal haemoglobin and a normal MCV**, so a normal blood count never excludes SCD.
5. **Investigations.** Serum B12 (low or low-normal). If equivocal, measure **serum methylmalonic acid and plasma homocysteine** — **both are raised in B12 deficiency; only homocysteine is raised in folate deficiency**. Anti-intrinsic-factor and anti-parietal-cell antibodies for pernicious anaemia. Peripheral smear shows macro-ovalocytes and **hypersegmented neutrophils**. MRI spine shows T2 hyperintensity of the posterior columns — the **"inverted V" or rabbit-ear sign**.
6. **Treatment.** Parenteral **hydroxocobalamin or cyanocobalamin 1000 µg intramuscularly on alternate days for 1–2 weeks, then weekly until no further improvement, then monthly for life**. **Never give folic acid alone** — it corrects the anaemia but precipitates or worsens the myelopathy. Monitor **serum potassium** during early recovery (hypokalaemia) and co-supplement iron. Recovery takes months and is **often incomplete if treatment is delayed beyond 6–12 months**, so treat on suspicion.

Latest Update – 1 Point

7. **NICE guideline NG239 — Vitamin B12 deficiency in over-16s (March 2024).** Where serum B12 is equivocal, use **plasma homocysteine or serum methylmalonic acid as the initial confirmatory test**; take all diagnostic samples **before** starting replacement. Crucially, **do not delay B12 replacement while awaiting results** in anyone with suspected megaloblastic anaemia or neurological symptoms, **especially subacute combined degeneration of the cord**. Lifelong intramuscular replacement is advised after total gastrectomy, complete terminal ileal resection, or when autoimmune gastritis is the cause.

Examiner's Tip — Verbatim Mark-Earning Lines

- "**Extensor plantars with absent ankle jerks** — the classical sign of subacute combined degeneration."
- "Neurological damage may occur **before, and without,** any haematological abnormality."
- "**Never give folate before B12** — it precipitates or worsens the myelopathy."
- "MMA *and* homocysteine raised = B12; homocysteine alone = folate."
- **Skeleton:** Definition/tracts → Causes → Signs (Romberg, extensor plantars, absent ankle jerks) → Haematological/psychiatric → MMA + MRI → B12 regimen and the folate trap → NICE NG239.

Question 20. Peripheral Neuropathy in Older Adults — Aetiology, Clinical Features, Investigations and Management 15 MARKS • ASKED ×2

Answer

Textbook Core – 10 Points

1. **Definition and classification.** A disorder of peripheral nerves, classified by **fibre type** (sensory, motor, autonomic, mixed), **distribution** (mononeuropathy, mononeuritis multiplex, polyneuropathy, radiculopathy, plexopathy), **pathology** (axonal versus demyelinating) and **time course** (acute, subacute, chronic). Prevalence rises steeply with age — around **8–15% of people over 55**.

2. **Aetiology — mnemonic DANG THERAPIST. D**iabetes; **A**lcohol; **N**utritional (B12, thiamine, folate, vitamin E, copper); **G**uillain–Barré syndrome; **T**oxins and drugs; **H**ereditary (Charcot–Marie–Tooth); **E**ndocrine and metabolic (hypothyroidism, uraemia, hepatic failure); **R**heumatological and vasculitic; **A**myloidosis; **P**araneoplastic; **I**nfections (leprosy, HIV, hepatitis C, Lyme); **S**arcoidosis; **T**umour and paraproteinaemia (MGUS, myeloma, POEMS). **In India, diabetes and leprosy lead; in older adults, diabetes, B12 deficiency, alcohol, CKD, hypothyroidism, paraproteinaemia and drugs account for most cases.**

3. **Drug causes to review at every consultation. Isoniazid** (always co-prescribe pyridoxine), **metronidazole, nitrofurantoin, phenytoin, amiodarone, colchicine, statins, linezolid, dapsone, chloroquine, thalidomide**, and chemotherapy — **vincristine, cisplatin, oxaliplatin, taxanes and bortezomib**. Also **excess pyridoxine** (a sensory ataxic neuropathy), and **long-term metformin and proton-pump inhibitors** acting through B12 depletion.

4. **Clinical features. Sensory** — symmetrical distal **"glove-and-stocking" numbness, tingling, burning and allodynia**, ascending over months to years. **Motor** — distal weakness and wasting, foot drop with a high-stepping gait, and **loss of ankle jerks**. **Autonomic** — postural hypotension, gastroparesis, diarrhoea or constipation, bladder dysfunction, erectile dysfunction and impaired sweating. **Trophic** — dry skin, foot deformity, **painless ulcers and Charcot joints**.

5. **Why neuropathy matters in geriatrics.** It is a major cause of **falls, fractures, gait instability and loss of independence**; sensory loss leads to **unnoticed foot ulceration and amputation**; autonomic involvement causes syncope; and neuropathic pain destroys sleep and mood. Importantly, **ankle jerks and vibration sense decline physiologically with age**, so isolated minor abnormalities must be interpreted with care and not over-investigated.

6. **A structured approach — five questions.** (i) **What is the distribution?** symmetrical distal, multifocal (mononeuritis multiplex) or proximal. (ii) **Which fibres are involved?** small fibre — pain and autonomic features with normal reflexes; large fibre — loss of vibration, proprioception and reflexes. (iii) **Axonal or demyelinating?** (iv) **What is the time course?** acute onset suggests **Guillain–Barré syndrome, vasculitis, porphyria or a toxin**. (v) **Is there a systemic clue?** weight loss, rash, organomegaly or a monoclonal band.

7. **First-line investigations. Fasting and postprandial glucose and HbA1c**, with an **oral glucose tolerance test if these are normal** — impaired glucose tolerance alone causes small-fibre neuropathy; **serum vitamin B12, with methylmalonic acid or homocysteine if borderline**; TSH; renal and liver function; full blood count and **ESR**; and **serum protein electrophoresis with immunofixation**, which is essential in older adults to detect a paraproteinaemia. Add HIV and hepatitis serology where indicated.

8. **Second-line and specialist investigations. Nerve conduction studies and EMG** confirm the diagnosis, localise the lesion and separate axonal from demyelinating pathology. A **demyelinating** pattern should prompt consideration of **CIDP — a treatable cause** (CSF shows albuminocytological dissociation). Further directed tests: ANA, ANCA and cryoglobulins; anti-MAG and anti-ganglioside antibodies; skeletal survey; **skin biopsy for intraepidermal nerve fibre density** in small-fibre neuropathy; genetic testing; and **sural nerve biopsy**, reserved for suspected vasculitis, amyloidosis or leprosy.

9. **Treatment of the cause. Glycaemic control** — tight but not aggressive, since hypoglycaemia is more dangerous than modest hyperglycaemia in the elderly; **vitamin B12 replacement**; alcohol cessation with thiamine; thyroid replacement; dialysis or transplantation for uraemia; **multidrug therapy for leprosy**; **immunotherapy (corticosteroids, IVIG, plasma exchange) for CIDP, Guillain–Barré syndrome and vasculitic neuropathy**; treatment of an underlying malignancy or paraproteinaemia; and **withdrawal of the offending drug**.

10. **Symptomatic and supportive management.** For **neuropathic pain**, first-line agents are **amitriptyline or nortriptyline, duloxetine, gabapentin and pregabalin**; second line, topical **capsaicin 8% patch** and **lidocaine 5% plaster**; **avoid opioids for chronic neuropathic pain**. Non-pharmacological care is equally important: **daily foot inspection, protective well-fitting footwear, podiatry and callus/ulcer care**, physiotherapy with strength and balance training, an **ankle-foot orthosis for foot drop**, walking aids, home hazard assessment and structured **falls prevention**, vitamin D and bone protection, and treatment of coexisting depression and insomnia.

Latest Updates – 2 Points

11. **AAN practice guideline on painful diabetic neuropathy (2022, reaffirmed through 2023–2025).** Four drug classes have **comparable efficacy** — **tricyclic antidepressants, SNRIs (duloxetine, venlafaxine), gabapentinoids, and sodium-channel blockers (lamotrigine, lacosamide, oxcarbazepine)** — so the choice should be driven by comorbidity and adverse-effect profile rather than a fixed ladder. Critically, **opioids, including tramadol, should NOT be offered for painful diabetic neuropathy**. In older adults this pushes

practice towards **duloxetine or a reduced-dose gabapentinoid** and away from amitriptyline, which appears on the **AGS Beers Criteria 2023**.

12. **Two shifts in disease-modifying thinking (2023–2025).** (i) **Glycaemic control alone is insufficient in type 2 diabetes** — the components of the metabolic syndrome (obesity, hypertriglyceridaemia, hypertension) are now recognised as **independent, modifiable drivers of neuropathy**, so lifestyle and lipid management form part of neuropathy treatment, not merely of cardiovascular prevention. (ii) **Hereditary transthyretin amyloid polyneuropathy (ATTRv)** — which classically presents in older adults as a **painful small-fibre and autonomic neuropathy with cardiomyopathy and carpal tunnel syndrome** — is now treatable with **RNA-silencing agents (patisiran, vutrisiran) and the stabiliser tafamidis**. Amyloid neuropathy must therefore be actively sought rather than assumed untreatable.

Examiner's Tip — Verbatim Mark-Earning Lines

- "**Distribution, fibre type, axonal or demyelinating, time course, systemic clue** — the five questions that structure any neuropathy answer."
- "**A demyelinating pattern on nerve conduction studies means look for CIDP — a treatable neuropathy.**"
- "Serum protein electrophoresis is **mandatory in any older patient with an unexplained neuropathy**."
- "In the elderly, the outcome that matters is not the pain score but **the ulcer, the fall and the fracture**."

Converting this to the 7-Mark Version (6 textbook points + 1 update)

- This question has been asked at both 15 and 7 marks. For **7 marks**, compress the ten points into six: **(1)** definition and classification (fibre type, distribution, axonal vs demyelinating, course; 8–15% over 55); **(2)** common causes — diabetes commonest, then B12 deficiency, alcohol, CKD, hypothyroidism, paraproteinaemia, drugs, and **leprosy in India**; **(3)** clinical features — glove-and-stocking sensory loss, absent ankle jerks, autonomic features, painless ulcers and Charcot joints; **(4)** geriatric significance — falls, fractures, ulceration and amputation, plus the physiological age-related loss of ankle jerks and vibration sense; **(5)** investigations — glucose/HbA1c with OGTT, B12 with MMA, TSH, renal and liver function, ESR, **serum protein electrophoresis (mandatory)**, then NCS/EMG with CIDP considered if demyelinating; **(6)** management — treat the cause, neuropathic-pain agents avoiding opioids, and foot care with falls prevention.
- Retain the same latest update (AAN painful diabetic neuropathy guideline plus ATTRv amyloid neuropathy) as the seventh point.
- Points **6 (the five-question approach)** and **8 (second-line and specialist investigations)** are the ones to sacrifice first — they carry the least marks per line in a short answer.

Question 21. Temporal Arteritis and its Neurological Complications 7 MARKS

Answer

Textbook Core – 6 Points

1. **Definition.** Temporal (giant cell) arteritis is a **granulomatous large- and medium-vessel vasculitis** affecting the extracranial branches of the carotid artery, occurring **almost exclusively above the age of 50** (peak 70–80 years). It is the commonest primary systemic vasculitis of old age and is closely associated with **polymyalgia rheumatica**.
2. **Clinical features. New-onset persistent unilateral temporal headache**, **scalp tenderness** (pain on combing the hair or resting the head on a pillow), **jaw claudication — the most specific symptom**, and a **tender, thickened, nodular or pulseless temporal artery**. Constitutional features include **fever of unknown origin**, weight loss, anorexia and malaise, with pain and morning stiffness of the shoulder and hip girdles if PMR coexists.
3. **Neuro-ophthalmic complications — the core of the answer. Sudden, painless, irreversible monocular visual loss** from **anterior ischaemic optic neuropathy** (commonest), central retinal artery occlusion, or preceding **amaurosis fugax**; and **diplopia** from ocular motor palsy. If untreated, **the second eye is affected in 25–50% within 1–2 weeks**. Other neurological complications: **transient ischaemic attack and stroke (characteristically vertebrobasilar)**, scalp and tongue necrosis, occipital neuropathy, and rarely mononeuritis multiplex, depression and dementia.
4. **Investigations. Raised ESR** (often >50 mm/h, classically >75 mm/h) and **CRP**, with normochromic normocytic anaemia, thrombocytosis and raised alkaline phosphatase. **Temporal artery biopsy** of a 1–2 cm segment, ideally within 1–2 weeks of starting steroids, shows granulomatous panarteritis with giant cells and fragmentation of the internal elastic lamina — but **skip lesions cause false negatives, so a negative biopsy does not exclude the diagnosis**. Colour Doppler ultrasound shows the **"halo sign"**; PET-CT or MR angiography detects large-vessel and aortic involvement.
5. **Treatment — a medical emergency. Start corticosteroids immediately on clinical suspicion; never wait for the biopsy.** Without visual symptoms: **prednisolone 40–60 mg/day**. With visual loss or amaurosis: **intravenous methylprednisolone 500–1000 mg/day for 3 days**, then oral prednisolone. Add **low-dose aspirin** and a proton-pump inhibitor. Taper slowly over **12–24 months**, guided by symptoms and CRP rather than ESR alone.
6. **Steroid toxicity and steroid-sparing therapy.** Older patients are highly vulnerable to **osteoporosis and fragility fracture, diabetes, hypertension, cataract, glaucoma, infection, proximal myopathy and delirium**. Start **calcium, vitamin D and a bisphosphonate from day one**, and monitor blood pressure and glucose. **Tocilizumab** (IL-6 receptor blocker) is the established steroid-sparing agent for relapsing or refractory disease; methotrexate is an alternative. A late complication is **thoracic aortic aneurysm**, requiring long-term surveillance.

Latest Update – 1 Point

7. **Upadacitinib — the first oral JAK inhibitor approved for giant cell arteritis** (European Commission April 2025; FDA 29 April 2025), at **15 mg once daily with a corticosteroid taper**. Approval was based on the phase 3 **SELECT-GCA trial (n=428)**, in which **46.4% achieved sustained remission from week 12 to week 52 with a 26-week steroid taper, versus 29.0% on placebo with a 52-week taper (p=0.002)**; sustained complete remission was 37.1% versus 16.1%. It provides an oral steroid-sparing alternative to tocilizumab — **particularly relevant in older patients at high risk of glucocorticoid toxicity**.

Current Practice Update

Item	Position
Textbook	High-dose corticosteroids are the treatment; methotrexate is used as a steroid-sparing agent in relapsing disease.
Current recommendation	Steroids remain the emergency treatment, but **tocilizumab** and now **upadacitinib 15 mg once daily** are approved steroid-sparing agents that increase sustained remission and shorten steroid exposure.
Exam approach	Write "steroids immediately, biopsy later", then close with: "Tocilizumab and upadacitinib (SELECT-GCA, 2025) are now the recognised steroid-sparing options."

Examiner's Tip — Verbatim Mark-Earning Lines

- "Giant cell arteritis is a **medical emergency — steroids first, biopsy afterwards**; delay costs the sight of the second eye."
- "**Jaw claudication is the most specific symptom; visual loss is the most feared complication.**"
- "A **negative temporal artery biopsy does not exclude GCA** because of skip lesions."
- "Any older patient with new headache and an **ESR above 50 mm/h** has GCA until proved otherwise."
- **Skeleton:** Definition/PMR link → Headache, scalp tenderness, jaw claudication → Visual and neurological complications → ESR/CRP/biopsy/halo sign → Steroid regimen → Steroid toxicity and bone protection → Upadacitinib.

Question 22. Trigeminal Neuralgia 7 MARKS

Answer

Textbook Core – 6 Points

1. **Definition and classification (ICHD-3).** Recurrent **unilateral, brief, electric shock-like facial pain**, abrupt in onset and termination, confined to one or more divisions of the trigeminal nerve and triggered by innocuous stimuli. Three categories: **classical** (neurovascular compression, usually by the superior cerebellar artery) in 80–90%; **secondary** (multiple sclerosis, cerebellopontine angle tumour, skull-base lesion, stroke); and **idiopathic**.
2. **Clinical features.** Paroxysms lasting from a fraction of a second to **2 minutes**, affecting **V2 and V3 most often and V1 least**, right more than left, and **almost never crossing the midline**. **Trigger zones around the nasolabial fold and chin** — pain provoked by chewing, talking, brushing the teeth, shaving, washing the face or cold wind. There is a refractory period after an attack, and remissions may last months. Some patients have superimposed continuous background pain ("type 2").
3. **Red flags suggesting secondary trigeminal neuralgia.** Age below 40, **bilateral pain**, **sensory loss in the trigeminal territory, an absent corneal reflex**, other cranial nerve palsies, deafness, and **failure to respond to carbamazepine**. These mandate detailed imaging.
4. **Investigations and differential diagnosis. MRI brain with dedicated high-resolution trigeminal sequences** (3D FIESTA/CISS with MR angiography) should be performed in all patients to demonstrate neurovascular compression and exclude a secondary cause; **trigeminal reflex (blink reflex) testing** is an alternative where MRI is unavailable. Differentials: **dental pain, temporomandibular joint dysfunction, giant cell arteritis, post-herpetic neuralgia, cluster headache, glossopharyngeal neuralgia and sinusitis**.
5. **Medical management. Carbamazepine is the drug of choice (Level A evidence)** — start **100 mg twice daily and titrate to 600–1200 mg/day**; **70–80% obtain initial relief**. **Oxcarbazepine (Level B)** is equally effective with better tolerability and fewer drug interactions. Second-line and add-on agents: **lamotrigine, baclofen, gabapentin and pregabalin**. Note that **more than half of initial responders lose their response over 5–10 years**.
6. **Geriatric precautions and surgical options.** Carbamazepine in older adults causes **hyponatraemia (SIADH), ataxia, falls, drowsiness, diplopia, bradycardia and AV block, leucopenia and aplastic anaemia, and rash including Stevens–Johnson syndrome** (test **HLA-B*1502** in Asian patients); it is a potent CYP450 inducer, reducing levels of warfarin, DOACs, statins and other antiepileptics — **monitor sodium, full blood count and liver function**. For drug-refractory pain: **microvascular decompression** gives the best long-term relief (about 70–80% pain-free at 10 years) but needs posterior fossa craniotomy, so frailty must be assessed; **percutaneous procedures** (radiofrequency thermocoagulation, balloon compression, glycerol rhizolysis) and **Gamma Knife stereotactic radiosurgery** are preferred in frail or high-risk elderly patients.

Latest Update – 1 Point

7. **Botulinum toxin type A for refractory trigeminal neuralgia.** An updated systematic review with meta-analysis (*Clin J Pain*, 2024) and a comprehensive review of 35 studies (*Toxins*, 2024) confirm that **subcutaneous or submucosal BTX-A significantly reduces VAS pain scores and attack frequency in patients refractory to, or intolerant of, medical and surgical therapy**, with **transient facial asymmetry** as the main adverse effect. A 2026 umbrella review positions BTX-A as an **adjunctive third-line option for focal peripheral neuropathic pain** — a useful, low-risk choice in frail older patients unsuitable for craniotomy.

Examiner's Tip — Verbatim Mark-Earning Lines

- "**Carbamazepine is both the drug of choice and a diagnostic test** — failure to respond suggests a secondary cause."
- "**Sensory loss or bilateral pain means it is not classical trigeminal neuralgia** — image the patient."
- "In the elderly, the commonest reason to abandon carbamazepine is **hyponatraemia and ataxia with falls**."
- "Microvascular decompression gives the best durability; **percutaneous and radiosurgical options suit the frail**."
- **Skeleton:** ICHD-3 definition and three types → Paroxysms and triggers → Red flags → MRI and differentials → Carbamazepine/oxcarbazepine → Geriatric toxicity and surgery → Botulinum toxin A.

Question 23. Cluster Headache 7 MARKS • ASKED ×2

Answer

Textbook Core – 6 Points

1. **Definition (ICHD-3).** A **trigeminal autonomic cephalalgia** characterised by attacks of severe, strictly **unilateral orbital, supraorbital and/or temporal pain lasting 15–180 minutes**, occurring from once every other day to **eight times a day**, accompanied by ipsilateral cranial autonomic features and/or a sense of restlessness or agitation.
2. **Classification and epidemiology.** Male predominance of about 3:1; onset usually between 20 and 40 years, but it can begin in old age. **Episodic cluster headache (80–90%)** — bouts lasting weeks to months separated by remissions of at least 3 months; **chronic cluster headache (10–20%)** — no remission, or remissions shorter than 3 months within a year.
3. **Clinical features.** The pain is **excruciating, boring or stabbing and retro-orbital**, with striking **circadian and circannual periodicity** — attacks wake the patient 1–2 hours after falling asleep and recur at the same time each day ("alarm-clock headache"), with bouts recurring in the same season each year. Ipsilateral autonomic features: **lacrimation, conjunctival injection, nasal congestion or rhinorrhoea, eyelid oedema, forehead and facial sweating, and miosis with ptosis (partial Horner's syndrome)**. Unlike migraine, the patient is **restless and pacing, not lying still**. Alcohol reliably triggers attacks during a bout but not during remission.
4. **Differential diagnosis.** Migraine; other trigeminal autonomic cephalalgias — **paroxysmal hemicrania** (shorter, more frequent, and **absolutely responsive to indomethacin**) and **SUNCT/SUNA** (attacks lasting seconds); trigeminal neuralgia; acute angle-closure glaucoma; **giant cell arteritis**; sinusitis; and **secondary cluster-like headache** from a pituitary lesion or carotid dissection. **A first presentation over the age of 50, or any abnormal neurological sign, mandates MRI brain with pituitary views.**
5. **Acute (abortive) treatment. High-flow 100% oxygen at 12–15 L/min through a non-rebreathing mask for 15–20 minutes** aborts about 70% of attacks (avoid in severe COPD with carbon dioxide retention), and **subcutaneous sumatriptan 6 mg** (fastest acting; maximum two doses in 24 hours) or **intranasal zolmitriptan 5 mg**. Oral triptans and simple analgesics act far too slowly to be useful. **Triptans are contraindicated in uncontrolled hypertension, ischaemic heart disease, previous stroke and peripheral vascular disease** — a major limitation in older patients, for whom **oxygen becomes the treatment of choice**.
6. **Preventive treatment. Verapamil is first line** (240–960 mg/day in divided doses); an **ECG must be done before starting and before every dose increase** because of bradycardia and heart block — critically important in older patients. Give **transitional therapy** with a short tapering course of oral prednisolone, or a **greater occipital nerve block**, to break the bout while verapamil is titrated. Alternatives: lithium (especially for the chronic form), topiramate and melatonin. For refractory chronic disease: occipital nerve stimulation or sphenopalatine ganglion stimulation.

Latest Update – 1 Point

7. **Galcanezumab — the only agent specifically approved for the prevention of episodic cluster headache.** A monoclonal antibody against **calcitonin gene-related peptide (CGRP)**, given as **300 mg subcutaneously at the onset of a bout and repeated monthly through the cluster period**; it **failed to show benefit in chronic cluster headache**. **Real-world data and an expert consensus published in 2024** (*Journal of Headache and Pain*) support its use as an add-on when verapamil is ineffective, not tolerated, or contraindicated — precisely the situation in older patients with conduction disease, in whom high-dose verapamil is hazardous. This sits alongside the American Headache Society 2024 position statement establishing CGRP-targeting therapies as a first-line option in migraine prevention.

Converting this to the 6-Mark Version (5 textbook points + 1 update)

- **Omit point 4 (differential diagnosis)** and retain points 1, 2, 3, 5 and 6 plus the latest update — giving exactly **6 points**.
- Carry the one indispensable line from point 4 into point 1 instead: "**A first presentation after the age of 50 requires MRI brain with pituitary views to exclude secondary cluster-like headache.**"
- Keep the numbers — **15–180 minutes, up to 8 attacks a day, oxygen 12–15 L/min, sumatriptan 6 mg SC, verapamil 240–960 mg** — whichever version is asked.

Examiner's Tip — Verbatim Mark-Earning Lines

- "**The migraine patient lies still in a dark room; the cluster patient paces the floor.**"
- "**Oxygen and subcutaneous sumatriptan abort; verapamil prevents; steroids bridge.**"
- "**ECG before every verapamil dose increase** — the doses used in cluster headache are far higher than cardiological doses."
- "An **indomethacin trial** should be given to exclude paroxysmal hemicrania before labelling any TAC as cluster headache."

Question 24. Tension Headache 6 MARKS

Answer

Textbook Core – 5 Points

1. **Definition (ICHD-3).** The commonest primary headache disorder. Pain is **bilateral, pressing or tightening (non-pulsating), of mild to moderate intensity, lasting 30 minutes to 7 days, and NOT aggravated by routine physical activity**. There is **no nausea or vomiting**; photophobia *or* phonophobia may be present, but not both. The classic description is a **"tight band" around the head**.
2. **Classification. Infrequent episodic** (fewer than 1 day per month), **frequent episodic** (1–14 days per month) and **chronic** (15 or more days per month for over 3 months). Chronic tension-type headache very commonly coexists with, and is perpetuated by, **medication-overuse headache**.
3. **Contributing factors.** Psychological stress, **anxiety and depression**, sleep disturbance and obstructive sleep apnoea, **pericranial muscle tenderness (the hallmark physical sign)**, cervical spondylosis and poor posture, eye strain, and caffeine or analgesic overuse. In older adults the dominant contributors are **cervical degenerative disease, depression and analgesic overuse**.
4. **Red flags mandating investigation in an older patient. Any new or changed headache after the age of 50**, thunderclap onset, progressive or positional headache, systemic features (fever, weight loss), **scalp tenderness or jaw claudication — check ESR and CRP for giant cell arteritis**, focal neurological signs, papilloedema, immunosuppression or malignancy, and **headache in an anticoagulated patient (subdural haematoma)**. In their absence, no imaging is required.
5. **Management. Non-pharmacological measures come first**: reassurance, a headache diary, sleep hygiene, regular meals, stress management, relaxation training, cognitive behavioural therapy, physiotherapy, posture correction and regular exercise. **Acute** — paracetamol, or an NSAID used cautiously in the elderly because of gastrointestinal, renal and cardiovascular risk; **limit acute analgesic use to fewer than 15 days a month to prevent medication-overuse headache**; **avoid opioids and butalbital combinations entirely**. **Preventive** for frequent or chronic forms — **amitriptyline is first line** starting at 10 mg at night, but it is anticholinergic, sedating, orthostatic and arrhythmogenic in older people; nortriptyline, mirtazapine or venlafaxine are alternatives.

Latest Update – 1 Point

6. **Current guidance and evidence syntheses (2023–2025)** place **non-pharmacological therapy at the centre of tension-type headache management**, with the strongest evidence for **cognitive behavioural therapy, relaxation training and supervised exercise or physiotherapy**. This matters especially in older adults, because **amitriptyline appears on the AGS Beers Criteria 2023** as a drug to avoid in older people (anticholinergic burden, sedation, orthostatic hypotension and falls). The second key message is the active identification and **withdrawal of medication-overuse headache**, which is a common, reversible and frequently missed cause of what is labelled "chronic tension headache" in this population.

Examiner's Tip — Verbatim Mark-Earning Lines

- "**Bilateral, pressing, mild to moderate, not worsened by activity, no vomiting** — the four-part ICHD-3 definition."
- "**Any new headache after 50 is not tension headache until giant cell arteritis and a space-occupying lesion have been excluded.**"
- "**Amitriptyline is first-line prophylaxis but is on the Beers list** — use the lowest dose, or choose nortriptyline."
- "Ask about analgesic frequency — **medication-overuse headache is the commonest reversible cause of chronic daily headache**."

Question 25. Hypoxic-Ischaemic Encephalopathy — Causes, Clinical Manifestations, Diagnosis and Management 15 MARKS

Answer

Textbook Core – 10 Points

1. **Definition.** Diffuse brain injury caused by a **global reduction in oxygen delivery (hypoxia) and/or cerebral blood flow (ischaemia)**, most often after cardiac arrest. The injury is **biphasic**: **primary ischaemic injury** during the arrest, followed by **secondary reperfusion injury** after return of spontaneous circulation — and it is the secondary phase that is modifiable.
2. **Causes. Cardiac arrest is the commonest**. Others: respiratory arrest and severe hypoxaemia (COPD exacerbation, drowning, choking, strangulation, hanging), profound or prolonged shock and haemorrhage, **carbon monoxide and cyanide poisoning**, severe anaemia, status asthmaticus, anaesthetic and airway mishaps, and prolonged status epilepticus.
3. **Pathophysiology and selective vulnerability.** Energy failure → **glutamate excitotoxicity** → calcium influx → free-radical generation, mitochondrial dysfunction and apoptosis. Certain neurons are selectively vulnerable: **the hippocampal CA1 sector, cerebellar Purkinje cells, cortical layers 3, 5 and 6, and the basal ganglia**. Prolonged hypotension additionally causes **watershed (border-zone) infarction**.
4. **Clinical spectrum.** Outcomes range from full recovery through **persistent coma, vegetative state (unresponsive wakefulness syndrome), minimally conscious state, and brain death**. Milder injury leaves an **amnestic (Korsakoff-like) syndrome from hippocampal damage**, cortical blindness with denial (**Anton's syndrome**), the watershed **"man-in-a-barrel"** pattern of proximal arm weakness with preserved distal function, and post-hypoxic parkinsonism or dystonia.
5. **Post-hypoxic myoclonus.** Two distinct forms. **Acute myoclonic status** within 24 hours of arrest is a poor prognostic sign but **is no longer regarded as uniformly fatal** and must not alone justify withdrawal of treatment. **Lance–Adams syndrome** is a delayed **action myoclonus** appearing days to weeks later in a patient who has regained consciousness; it responds to clonazepam, valproate, levetiracetam or piracetam.
6. **Delayed post-hypoxic leukoencephalopathy.** An apparent recovery followed by relapse after **2–4 weeks**, with cognitive decline, parkinsonism and akinetic mutism, and diffuse white matter change on MRI — classically after **carbon monoxide poisoning**, but also after opioid overdose and prolonged hypoxia.
7. **Clinical assessment. Serial neurological examination is central**: GCS or FOUR score, **pupillary and corneal reflexes**, oculocephalic and oculovestibular responses, motor response to a standardised painful stimulus, breathing pattern, and the presence of myoclonus. **Confounders must be excluded before any conclusion is drawn** — residual sedatives (propofol, opioids, neuromuscular blockers), hypothermia, metabolic and electrolyte derangement, unrecognised seizures, and organ failure.
8. **Investigations. EEG** — malignant patterns are burst-suppression, a suppressed background and status epilepticus, whereas a **continuous reactive background is favourable**. **Somatosensory evoked potentials** — **bilaterally absent N20 responses indicate a very poor prognosis**. **Serum neuron-specific enolase (NSE)** and neurofilament light chain. **CT brain** — loss of grey–white differentiation, effaced sulci and the "pseudo-subarachnoid haemorrhage" sign. **MRI with DWI at 2–7 days is the most sensitive imaging test.**
9. **Management — prevent secondary injury.** Treat the underlying cause (coronary angiography and PCI where indicated). Maintain **MAP ≥65–80 mmHg**; **avoid both hypoxaemia and hyperoxaemia**; maintain normocapnia; keep **normoglycaemia while strictly avoiding hypoglycaemia**; **prevent fever aggressively**; treat seizures (levetiracetam or valproate) while avoiding deep prolonged sedation that will later confound prognostication; correct electrolytes; and provide standard critical-care supportive measures.
10. **Prognostication and geriatric/ethical issues.** Prognostication must be **multimodal and delayed to at least 72 hours after normothermia and after sedation has cleared**; **no single test may be used alone**. Markers of poor outcome: absent pupillary and corneal reflexes at ≥72 hours, bilaterally absent N20 SSEPs, a malignant EEG, myoclonic status, very high NSE, and diffuse injury on CT or MRI. In older adults, discuss **goals of care, advance directives and surrogate decision-making early**. Because **withdrawal of life-sustaining therapy is the commonest mode of death**, premature pessimism creates a **self-fulfilling prophecy** and must be avoided.

Latest Updates – 2 Points

11. **ERC/ESICM 2025 Post-Resuscitation Care Guidelines (adopting the 2024 ILCOR recommendations).** The goal is now **active fever prevention with a target temperature at or below 37.5 °C**, rather than routine therapeutic hypothermia at 33 °C — following the TTM2 and HYPERION trials. Temperature control should be maintained for **36–72 hours** using devices with feedback-controlled temperature monitoring, and **routine prehospital cooling with cold intravenous fluid is not recommended**. Neuroprognostication should be **multimodal and delayed**, with a **repeat brain CT at 72–96 hours** if the first scan showed no hypoxic-ischaemic change, and MRI ideally at **2–7 days**.

12. **AHA Post-Cardiac Arrest Care Guideline (*Circulation*, 2025).** The neuroprognostication section has been expanded to include **predictors of favourable outcome**, not only of poor outcome, and **serum neurofilament light chain (NfL) has been added alongside NSE as a biomarker** — when used **in combination with other prognostic tests**, high NSE or NfL within 72 hours supports a prediction of unfavourable neurological outcome. Temperature control should be maintained for **at least 36 hours** in patients who remain unresponsive to verbal commands after ROSC, and CT plus echocardiography are reasonable after ROSC to establish the cause of arrest and detect resuscitation complications.

Current Practice Update

Item	Position
Textbook	Therapeutic hypothermia to 32–34 °C for 12–24 hours is recommended for comatose survivors of cardiac arrest.
Current recommendation	ERC/ESICM 2025 and AHA 2025: the aim is **fever prevention with a target ≤37.5 °C**, maintained 36–72 hours; deep hypothermia at 33 °C is no longer routine (TTM2, HYPERION). **No prehospital cold-fluid cooling.**
Exam approach	State: "Targeted temperature management has shifted from therapeutic hypothermia at 33 °C to **active fever prevention at or below 37.5 °C** for 36–72 hours."

Examiner's Tip — Verbatim Mark-Earning Lines

- "The injury is **biphasic — primary ischaemia and secondary reperfusion injury**; only the second is modifiable."
- "**Hippocampal CA1, cerebellar Purkinje cells and the basal ganglia** are the selectively vulnerable regions."
- "**Prognostication must be multimodal and never before 72 hours** off sedation and at normothermia."
- "Withdrawal of life-sustaining therapy is the commonest mode of death — **beware the self-fulfilling prophecy**."
- **Skeleton:** Definition/biphasic → Causes → Selective vulnerability → Clinical spectrum → Myoclonus → Delayed leukoencephalopathy → Examination → EEG/SSEP/NSE/MRI → Prevent secondary injury → Prognostication + ethics → 2 updates.

Question 26. Post-Acute Management of Traumatic Brain Injury 7 MARKS

Answer

Textbook Core – 6 Points

1. **Scope and geriatric context.** The post-acute phase begins once the patient is medically and neurosurgically stable, and covers **rehabilitation, prevention of secondary complications, and management of long-term sequelae**. In older adults, TBI is usually caused by **falls**, is frequently complicated by antiplatelet or anticoagulant use, and carries **higher mortality and worse functional outcome for any given injury severity**.
2. **Structured multidisciplinary rehabilitation.** Refer early to a team comprising a rehabilitation physician, physiotherapist, occupational therapist, speech and language therapist, **neuropsychologist**, dietitian and social worker. Set goal-directed, individualised targets and measure progress with **GOS-E, FIM or Barthel Index** and formal cognitive testing. **Early, intensive, specialist inpatient rehabilitation produces better functional outcomes** than generic ward care.
3. **Cognitive rehabilitation.** Measure the **duration of post-traumatic amnesia** (a key prognostic marker) with the GOAT or Westmead scale. Deficits in **attention, memory, processing speed and executive function** are managed with compensatory strategies, memory aids and diaries, environmental structuring and errorless learning. Formal screening is essential in older adults, in whom these deficits are easily mislabelled as dementia.
4. **Neurobehavioural and psychiatric sequelae.** Agitation and aggression, apathy, disinhibition, emotional lability, **depression (up to 50%)**, anxiety, post-traumatic stress, disturbed sleep–wake cycle and fatigue. Manage behaviour **non-pharmacologically first** — routine, environmental modification, identifying triggers. Use SSRIs for depression; **avoid typical antipsychotics and benzodiazepines, which impair motor and cognitive recovery**. Methylphenidate or amantadine may improve arousal and attention in selected patients.
5. **Medical complications to anticipate. Post-traumatic seizures** — prophylaxis only for the first 7 days; longer-term antiseizure medication (preferably levetiracetam) only if seizures actually occur. **Post-traumatic hydrocephalus** — suspect when recovery plateaus or reverses; image and consider shunting. Also spasticity and contractures, heterotopic ossification, **dysphagia and aspiration**, malnutrition, bladder and bowel dysfunction, pressure ulcers, venous thromboembolism, chronic pain and post-traumatic headache, **post-traumatic hypopituitarism** (growth hormone, thyroid, cortisol and gonadal deficiency — commonly missed), and **chronic subdural haematoma appearing weeks later**.
6. **Long-term, social and geriatric issues.** Assess **mental capacity** and undertake advance care planning; give explicit **driving cessation and return-to-activity advice**; train and support caregivers and arrange respite. Prevent the next injury — **falls prevention: home hazard assessment, vision and hearing, footwear, gait aids, medication review, bone health** — and decide with the cardiology team on reintroducing antithrombotics. Counsel that TBI raises the long-term risk of **dementia, epilepsy and Parkinson's disease**, and arrange disability certification and welfare access under NPHCE and the **Rights of Persons with Disabilities Act 2016**.

Latest Update – 1 Point

7. **Contemporary guidance and evidence syntheses (2023–2025)** add three practice points. (i) **Blood biomarkers GFAP and UCH-L1** (available as an FDA-cleared point-of-care assay) assist triage and prognostication after TBI and reduce unnecessary CT scanning — useful in frail patients in whom repeated imaging is undesirable. (ii) **Systematic screening for post-traumatic hypopituitarism at 3–6 and 12 months** is recommended, since untreated hypopituitarism mimics and worsens post-concussive fatigue, apathy and cognitive slowing. (iii) **Age alone must not be used to withhold or limit rehabilitation** — outcome data consistently show that older TBI survivors achieve meaningful functional gains when given adequate rehabilitation intensity.

Examiner's Tip — Verbatim Mark-Earning Lines

- "**Seizure prophylaxis is for 7 days only** — it prevents early, not late, post-traumatic seizures."
- "When recovery **plateaus or reverses, think post-traumatic hydrocephalus or chronic subdural haematoma**."
- "**Screen for hypopituitarism** — it is the most commonly missed treatable cause of poor recovery after TBI."
- "**Avoid antipsychotics and benzodiazepines**; they slow motor and cognitive recovery."

Question 27. Wernicke Encephalopathy 7 MARKS

Answer

Textbook Core – 6 Points

1. **Definition.** An **acute, potentially reversible neuropsychiatric emergency caused by thiamine (vitamin B1) deficiency**, with lesions in the **mammillary bodies, medial thalami, periaqueductal grey matter and the floor of the fourth ventricle**. If untreated, it progresses to the largely irreversible **Korsakoff syndrome** — together, the Wernicke–Korsakoff syndrome.
2. **Causes in the elderly.** Chronic alcoholism is the classical cause, but in older adults the **non-alcoholic causes dominate and are routinely overlooked**: malnutrition and starvation, prolonged vomiting, **bariatric surgery and gastrectomy**, malignancy and chemotherapy, dialysis, **prolonged parenteral nutrition without vitamin supplementation**, severe systemic illness and sepsis, thyrotoxicosis, and critically — **administration of glucose to a thiamine-deplete patient**.
3. **Classical triad. Confusion, ophthalmoplegia and ataxia**. However, the **complete triad is present in only about 10–16% of cases**, so waiting for it is a diagnostic error. Ocular signs: **horizontal nystagmus, bilateral lateral rectus palsy and conjugate gaze palsy**; the ataxia is predominantly truncal and affects gait. In older adults it frequently presents simply as **unexplained delirium, apathy or "failure to thrive"**.
4. **Caine criteria — the practical bedside rule.** Diagnose (and treat) if **any TWO** of the following four are present: (i) **dietary deficiency**, (ii) **oculomotor abnormality**, (iii) **cerebellar dysfunction**, (iv) **either altered mental state or mild memory impairment**. This substantially increases sensitivity over the classical triad and should be quoted in the answer.
5. **Investigations. The diagnosis is clinical and treatment must never await results.** Serum thiamine or erythrocyte transketolase activity may be sent, but **take the sample before giving thiamine**. **MRI** (about 53% sensitive, 93% specific) shows **symmetrical T2/FLAIR hyperintensity of the medial thalami, periaqueductal grey, mammillary bodies and tectal plate**. Also check **magnesium** (an essential cofactor — thiamine will not work while hypomagnesaemia persists), sodium, glucose, liver function and vitamin B12.
6. **Treatment. Give parenteral thiamine BEFORE any glucose load**. Standard regimen: **thiamine 500 mg IV three times daily for 2–3 days**, then **250 mg IV/IM daily for 3–5 days**, then oral 100 mg daily long term. **Correct magnesium**, give the remaining B-complex vitamins, treat the underlying cause and provide nutritional rehabilitation. Ocular signs improve within hours to days and ataxia over weeks, but **confusion and memory often do not recover — about 80% of survivors develop Korsakoff syndrome** with anterograde amnesia and confabulation.

Latest Update – 1 Point

7. **Reviews and practice guidance published 2023–2025** converge on three messages. First, the **low-dose oral or 100 mg intramuscular thiamine still given in many hospitals is inadequate** for suspected Wernicke encephalopathy — **high-dose parenteral thiamine (500 mg IV three times daily) is the standard**, and prophylactic parenteral thiamine (200–300 mg daily) should be given to any at-risk patient before glucose or refeeding. Second, there is growing emphasis on **non-alcoholic Wernicke encephalopathy in older patients** — after bariatric surgery, prolonged vomiting, chemotherapy and prolonged ICU stay — which is where most missed cases now occur. Third, **magnesium must be corrected concurrently**, or thiamine replacement fails.

Examiner's Tip — Verbatim Mark-Earning Lines

- "**Never give glucose before thiamine** in a malnourished or alcoholic patient — it can precipitate Wernicke encephalopathy."
- "**The complete triad occurs in only 10–16%** — use the Caine criteria (any two of four) and treat on suspicion."
- "Wernicke encephalopathy is a **clinical diagnosis and a therapeutic emergency**; a normal MRI does not exclude it."
- "**Correct magnesium** — thiamine cannot work without it."

Question 28. CSF Findings in Pyogenic, Tuberculous, Fungal and Carcinomatous Meningitis

7 MARKS

Answer

Textbook Core – 6 Points

1. **Normal CSF and pre-requisites.** Opening pressure **8–18 cm H_2O**; clear and colourless; **0–5 lymphocytes/mm³**; protein **15–45 mg/dL**; glucose **45–80 mg/dL, or at least 60% of a simultaneous blood glucose** — **always send a paired blood sugar, or the CSF glucose cannot be interpreted**. Contraindications to lumbar puncture: raised intracranial pressure with focal signs or papilloedema, coagulopathy or anticoagulation, and local sepsis. **CT brain before LP** in any older, immunocompromised, seizing or focally abnormal patient.
2. **The comparison table — the core of the answer.** See table below.
3. **Pyogenic (bacterial) meningitis. Turbid** fluid at high pressure; **neutrophilic pleocytosis, typically 1000–5000/mm³**; protein 100–500 mg/dL; **glucose markedly reduced (<40% of blood glucose)**; **raised CSF lactate (>3.5 mmol/L)**; Gram stain positive in 60–90% before antibiotics; culture and multiplex PCR. **Partially treated meningitis** shifts towards a lymphocytic picture with a less dramatic fall in glucose, and is a common diagnostic trap.
4. **Tuberculous meningitis.** Clear or ground-glass fluid at high pressure, forming a **cobweb (spider-web) clot on standing**; **lymphocytic pleocytosis of 100–500/mm³** (neutrophils may predominate in the first few days); **protein markedly raised, 100–500 mg/dL, and may exceed 1 g/dL with spinal block (Froin's syndrome)**; **glucose low**; **adenosine deaminase raised**. AFB smear has a low yield; **Xpert MTB/RIF Ultra is the recommended rapid test**; culture takes weeks.
5. **Fungal (especially cryptococcal) meningitis.** Clear fluid, often at a **very high opening pressure requiring therapeutic drainage**; **lymphocytic pleocytosis of 20–500/mm³**, which may be minimal in the profoundly immunosuppressed; protein raised; **glucose low**; **India ink positive in about 50–70%**; and **CSF cryptococcal antigen (CrAg) is the test of choice with sensitivity above 95%**. Fungal culture confirms.
6. **Carcinomatous (neoplastic/leptomeningeal) meningitis.** Clear or xanthochromic; **mononuclear pleocytosis, usually modest (<100/mm³)**; **protein raised**; and **glucose low (hypoglycorrhachia) — making it the great mimic of tuberculous meningitis**. Diagnosis rests on **CSF cytology from repeated large-volume samples** (a single sample is only about 50% sensitive; three samples approach 90%), flow cytometry for haematological malignancy, and **contrast MRI of the whole neuraxis** showing leptomeningeal enhancement.

Table 1 — Comparative CSF Findings

Parameter	Pyogenic	Tuberculous	Fungal	Carcinomatous
Appearance	Turbid / purulent	Clear, ground-glass; **cobweb clot**	Clear	Clear or xanthochromic
Pressure	Raised	Raised	**Very high**	Normal or raised
Cell count	1000–5000/mm³	100–500/mm³	20–500/mm³	Usually <100/mm³
Cell type	**Neutrophils**	Lymphocytes	Lymphocytes	Lymphocytes + **malignant cells**
Protein	100–500 mg/dL	**100–500, may exceed 1 g/dL**	Raised	Raised
Glucose	**Markedly low**	Low	Low	**Low**
Key test	Gram stain, culture, lactate	**Xpert MTB/RIF Ultra**, ADA	**CrAg**, India ink	**Cytology ×3**, flow cytometry
Onset	Hours	Weeks	Weeks	Weeks–months

Table 2 — Contrast with Viral and Normal CSF

Parameter	Viral meningitis	Normal
Cells	10–500/mm³, lymphocytic	0–5 lymphocytes/mm³
Protein	Normal or mildly raised (<100 mg/dL)	15–45 mg/dL
Glucose	**NORMAL** — the key discriminator	45–80 mg/dL (≥60% of blood)

Latest Update – 1 Point

7. **Two diagnostic and therapeutic changes with direct Indian relevance.** (i) **WHO now recommends Xpert MTB/RIF Ultra as the initial diagnostic test on CSF for suspected tuberculous meningitis**, in preference to smear microscopy and culture, and India's **NTEP has scaled up Ultra and Truenat testing** accordingly. (ii) For cryptococcal disease, WHO guidance (2022, implemented through 2023–2025) recommends the **CrAg lateral flow assay as the preferred rapid test** and a **single high-dose liposomal amphotericin B (10 mg/kg) plus**

flucytosine and fluconazole induction regimen, based on the **AMBITION-cm** trial — markedly less toxic and more feasible than 7–14 days of conventional amphotericin in resource-limited settings.

Examiner's Tip — Verbatim Mark-Earning Lines

- "**Low CSF glucose in a lymphocytic meningitis means TB, fungus or malignancy — never a virus.**"
- "**Always send a simultaneous blood glucose**; the CSF glucose is meaningless without it."
- "**Carcinomatous meningitis is the great mimic of tuberculous meningitis** — send cytology on three large-volume samples."
- "**Partially treated pyogenic meningitis** looks lymphocytic — ask about prior antibiotics."

Question 29. Serotonin Syndrome 7 MARKS

Answer

Textbook Core – 6 Points

1. **Definition.** A **predictable, dose-related toxic state caused by excess serotonergic activity** at central and peripheral 5-HT receptors, chiefly **5-HT$_{2A}$**. It is **not idiosyncratic** — it follows starting or increasing a serotonergic drug, or more commonly **adding a second serotonergic agent**. Onset is rapid: **within 6–24 hours** in most cases.
2. **Causative drugs.** SSRIs, SNRIs, **tricyclics**, **MAO inhibitors — including linezolid and methylene blue**, trazodone, mirtazapine, **tramadol, fentanyl, pethidine and dextromethorphan**, triptans, ondansetron and metoclopramide, lithium, valproate, **St John's wort**, amphetamines, and enzyme inhibitors that raise SSRI levels. In geriatric practice the two classic culprits are **an SSRI plus tramadol** and **an SSRI plus linezolid**.
3. **Clinical triad.** (i) **Neuromuscular hyperactivity** — **clonus (spontaneous, inducible and ocular), hyperreflexia, tremor and rigidity, all more marked in the LOWER limbs**; (ii) **autonomic instability** — hyperthermia, tachycardia, hypertension, diaphoresis, **mydriasis**, flushing, and **diarrhoea with hyperactive bowel sounds**; (iii) **altered mental state** — agitation, anxiety, restlessness and confusion.
4. **Diagnosis — Hunter Serotonin Toxicity Criteria.** In a patient on a serotonergic agent, the diagnosis is made if **any one** of the following is present: **spontaneous clonus; inducible clonus with agitation or diaphoresis; ocular clonus with agitation or diaphoresis; tremor with hyperreflexia; or hypertonia with temperature above 38 °C plus ocular or inducible clonus** — **sensitivity 84%, specificity 97%**. There is no confirmatory laboratory test; investigations (CK, renal function, electrolytes, coagulation, ABG, ECG) are for complications and mimics.
5. **Differential diagnosis. Neuroleptic malignant syndrome** — slower onset over days, **lead-pipe rigidity greater in the upper body, bradyreflexia, normal pupils**, dopamine antagonist exposure; **anticholinergic toxicity** — **dry skin, absent bowel sounds, normal reflexes**; malignant hyperthermia (anaesthetic exposure); sympathomimetic toxicity; meningitis or encephalitis; thyroid storm; and alcohol or benzodiazepine withdrawal.
6. **Management. Stop all serotonergic drugs immediately** — this alone resolves most cases within 24 hours. Supportive care is the mainstay: IV fluids, cardiac monitoring, and **benzodiazepines (diazepam or lorazepam) for agitation, tremor and rigidity**, which also lower temperature. Use **active external cooling** for hyperthermia and **avoid antipyretics — the fever is muscular, not hypothalamic**. Control blood pressure with short-acting agents (esmolol, nitroprusside); **avoid bromocriptine, dantrolene and physical restraints**. For severe or refractory cases give **cyproheptadine, a 5-HT$_{2A}$ antagonist — 12 mg orally, then 2 mg every 2 hours**. Temperature above 41.1 °C requires intubation, sedation and **non-depolarising neuromuscular paralysis**.

Latest Update – 1 Point

7. **Prevention has become the focus, not new antidotes.** The **AGS Beers Criteria 2023** specifically flags the combination of an **SSRI or SNRI with tramadol** as a drug–drug interaction to avoid in older adults — both for serotonin toxicity and for the compounded risks of **hyponatraemia, falls and fracture**. **Linezolid**, a reversible monoamine oxidase inhibitor, now carries an explicit requirement for a formal serotonergic-drug review before prescription, an issue that arises frequently when treating MRSA or VRE infections in older inpatients already on an antidepressant. Electronic interaction alerts and structured deprescribing, rather than pharmacological rescue, are the current practice emphasis.

Examiner's Tip — Verbatim Mark-Earning Lines

- "**Clonus is to serotonin syndrome what lead-pipe rigidity is to neuroleptic malignant syndrome.**"
- "**Rapid onset, lower-limb predominance, hyperreflexia, mydriasis and diarrhoea** — serotonin syndrome."
- "**Do not give antipyretics** — the hyperthermia is generated by muscle activity, not by the hypothalamic set point."
- "The commonest cause in geriatric practice is **an SSRI plus tramadol**."

Question 30. Neuroleptic Malignant Syndrome 7 MARKS

Answer

Textbook Core – 6 Points

1. **Definition.** A rare, life-threatening **idiosyncratic reaction to dopamine (D_2) receptor blockade**, or to abrupt withdrawal of dopaminergic therapy, characterised by hyperthermia, rigidity, autonomic instability and altered consciousness. Onset is **subacute, over 1–3 days**, and most cases occur **within the first two weeks** of starting or increasing the drug. Mortality has fallen to about **5–10%** with early recognition.
2. **Causes. Typical antipsychotics — haloperidol is the classic culprit**; atypical antipsychotics (risperidone, olanzapine, quetiapine), often with milder rigidity; and the frequently forgotten **antiemetics — metoclopramide, prochlorperazine, promethazine and domperidone**. Also **abrupt withdrawal of levodopa or a dopamine agonist in Parkinson's disease** (the parkinsonism–hyperpyrexia syndrome). Risk factors: dehydration, agitation, high or rapidly escalated dose, parenteral or depot administration, male sex, a previous episode, and pre-existing extrapyramidal disease.
3. **Clinical tetrad.** (i) **Severe generalised "lead-pipe" rigidity** with tremor and dysphagia; (ii) **hyperthermia above 38 °C, often exceeding 40 °C**; (iii) **autonomic instability** — labile blood pressure, tachycardia, tachypnoea, profuse diaphoresis, urinary incontinence; (iv) **altered mental state** ranging from agitated confusion to stupor and coma. Crucially, **reflexes are normal or reduced and there is no clonus** — the key discriminator from serotonin syndrome.
4. **Investigations. Markedly raised creatine kinase — often above 1000 IU/L and sometimes above 10,000** — reflecting rhabdomyolysis; leucocytosis; raised LDH and transaminases; **low serum iron**; metabolic acidosis; deranged renal function with myoglobinuria; and disseminated intravascular coagulation in severe cases. Exclude mimics with CT or MRI brain, lumbar puncture, blood and urine cultures, thyroid function and toxicology. **The diagnosis remains clinical** (DSM-5 criteria).
5. **Differential diagnosis. Serotonin syndrome** (rapid onset, clonus, hyperreflexia, lower-limb predominance, diarrhoea, mydriasis); **malignant hyperthermia** (anaesthetic exposure); **malignant catatonia** (may be clinically indistinguishable); anticholinergic delirium; CNS infection; heat stroke; thyroid storm; tetanus; status epilepticus; and in older patients, **drug-induced parkinsonism complicated by an intercurrent infection**.
6. **Management. Stop the offending antipsychotic immediately — or restart the withdrawn dopaminergic drug** — and transfer to intensive care. Supportive care dominates: **aggressive intravenous fluids to prevent acute kidney injury from rhabdomyolysis**, active cooling, correction of electrolytes, VTE and aspiration prophylaxis, and **benzodiazepines (lorazepam)** for agitation and for mild cases. For moderate to severe disease: **bromocriptine 2.5–5 mg 8-hourly** (or amantadine) to restore dopaminergic tone, and **dantrolene 1–2.5 mg/kg IV** for severe rigidity and hyperthermia. **Electroconvulsive therapy** is used for refractory cases and when malignant catatonia cannot be excluded. Recovery takes 7–14 days; if an antipsychotic is later essential, **wait at least 2 weeks and restart with a low dose of a low-potency or atypical agent**.

Latest Update – 1 Point

7. **Prevention is the dominant geriatric message, reinforced by the AGS Beers Criteria 2023.** Antipsychotics should be **avoided for the behavioural and psychological symptoms of dementia** unless non-pharmacological measures have failed and the patient poses a danger to self or others, because of the **boxed warning of increased mortality and stroke**, together with falls, aspiration and NMS. Recent reviews (2023–2025) additionally emphasise that **NMS from atypical antipsychotics is increasingly common and often presents atypically in older adults — with lower fever and less prominent rigidity**. The practical corollary: **a raised creatine kinase in a rigid, confused older patient on any antipsychotic or antiemetic should trigger the diagnosis**, rather than waiting for the classical tetrad.

Table — Serotonin Syndrome versus Neuroleptic Malignant Syndrome

Feature	Serotonin syndrome	Neuroleptic malignant syndrome
Drug	Serotonergic agent (SSRI, tramadol, linezolid)	Dopamine antagonist, or dopaminergic withdrawal
Onset	**Hours (<24 h)**	**Days (1–3 days)**
Reflexes	**Hyperreflexia and clonus**	**Normal or reduced; no clonus**
Rigidity	Lower limbs > upper limbs	Generalised **"lead-pipe"**, upper body prominent
Pupils	**Mydriasis**	Normal
Bowel sounds	**Hyperactive, diarrhoea**	Normal or reduced
Resolution	Within 24 hours of stopping the drug	7–14 days
Specific drug	**Cyproheptadine**	**Bromocriptine ± dantrolene**

Question 31. Rhabdomyolysis 7 MARKS

Answer

Textbook Core – 6 Points

1. **Definition.** A syndrome of **skeletal muscle necrosis with release of intracellular contents — myoglobin, creatine kinase, potassium, phosphate and urate — into the circulation**. It is diagnosed by a **creatine kinase more than five times the upper limit of normal (usually >1000 IU/L)**, and a **CK above 5000 IU/L** marks significant risk of acute kidney injury.
2. **Causes in older adults. The "long lie" after an unwitnessed fall is the single most important cause in geriatric practice**, along with prolonged immobility and pressure necrosis. Others: **drugs — statins, especially when combined with fibrates, amiodarone, macrolides, azoles, ciclosporin or colchicine**; alcohol; antipsychotics via NMS; **infection and sepsis**; **hypokalaemia, hypophosphataemia and hyponatraemia**; hypothyroidism; diabetic ketoacidosis and hyperosmolar state; **heat stroke**; seizures and status epilepticus; trauma and crush injury; compartment syndrome; and inflammatory myopathies.
3. **Clinical features.** The classical triad of **myalgia, weakness and dark "cola-coloured" urine is present in fewer than 10%** of cases; muscles may be swollen and tender. In older patients the presentation is typically **non-specific — a fall, confusion, immobility or being "off legs"** — so the diagnosis depends on **actively checking the CK** rather than waiting for classical features. Examine for pressure sores and dependent oedema.
4. **Investigations. Serum CK** — rises within 12 hours, peaks at 24–72 hours and falls over 3–5 days. **Urine dipstick positive for blood but with NO red cells on microscopy** — this mismatch indicates myoglobinuria and is the classic examination point. Also serum and urine myoglobin; **potassium, calcium, phosphate, urate and bicarbonate**; urea and creatinine; liver enzymes (a raised AST/ALT of muscle origin); coagulation screen for DIC; **ECG for hyperkalaemia**; and a search for the underlying cause.
5. **Complications. Acute kidney injury in 10–50%** — the mechanism is renal vasoconstriction, direct myoglobin tubular toxicity and **intratubular cast obstruction**, all worsened by hypovolaemia and acidic urine. **Hyperkalaemia with life-threatening arrhythmia** is the immediate danger. There is **early hypocalcaemia** (calcium deposits in damaged muscle) followed by **late rebound hypercalcaemia** — so calcium should NOT be given unless there is symptomatic hypocalcaemia or hyperkalaemic ECG change. Also hyperphosphataemia, hyperuricaemia, metabolic acidosis, **compartment syndrome** and DIC.
6. **Management. Early, aggressive intravenous fluid resuscitation is the single most important intervention.** Start isotonic saline as soon as possible — initially **1–1.5 L/hour**, then titrated to a **urine output of 200–300 mL/hour** — while monitoring carefully for fluid overload in older patients with heart failure or chronic kidney disease. Stop the causative drug; treat hyperkalaemia; correct the precipitating cause; monitor for compartment syndrome and refer for fasciotomy if it develops; and use **renal replacement therapy** for refractory hyperkalaemia, severe acidosis, uraemia or fluid overload. **Urinary alkalinisation with bicarbonate and the use of mannitol are NOT routinely recommended** — the evidence is weak and both carry risk (hypocalcaemia, volume overload).

Latest Update – 1 Point

7. **Contemporary reviews and guidance (2023–2025)** reaffirm that **volume resuscitation alone — without bicarbonate or mannitol — is the standard of care**, and place new emphasis on **early risk stratification using the McMahon score**, which combines age, sex, initial creatinine, calcium, phosphate, bicarbonate, CK and cause to identify patients likely to need renal replacement therapy, allowing earlier escalation. The corresponding **geriatric preventive message** is equally examinable: **falls prevention, personal alarm systems and a rapid response to the "long lie"**, together with a **statin interaction review** (particularly with fibrates, macrolides, azoles and amiodarone) at every medication reconciliation.

Examiner's Tip — Verbatim Mark-Earning Lines

- "**Urine positive for blood on dipstick with no red cells on microscopy is myoglobinuria until proved otherwise.**"
- "**Fluids, fluids and more fluids** — and neither bicarbonate nor mannitol."
- "**Early hypocalcaemia, late hypercalcaemia** — do not correct the calcium unless the patient is symptomatic."
- "In geriatrics, rhabdomyolysis is the **metabolic consequence of the long lie** — check the CK in every patient found on the floor."

Appendix — Rapid Revision Grid

Consolidated from both source volumes. Where a fact appears in more than one answer it is listed once here.

A. Examinable Numbers

Topic	Number to reproduce
Epilepsy in the elderly	Incidence >100/1,00,000/yr after 65; cerebrovascular cause 30–50%; EEG sensitivity 26–38%; ~80% seizure-free on low-dose monotherapy
Complex partial seizure	Duration 30 s–3 min; post-ictal confusion 5–30 min; temporal lobectomy seizure-free in ~2/3 of selected cases
Status epilepticus	Lorazepam 0.1 mg/kg; levetiracetam 60 mg/kg; fosphenytoin 20 PE/kg; valproate 40 mg/kg
TIA	ABCD² ≥4 = high risk; stroke risk ~5% at 7 days, 10% at 90 days; DAPT 21 days; DWI lesion in up to 1/3
Lacunar stroke	Infarct <15–20 mm; ~25% of ischaemic strokes; SPS3 systolic target <130 mmHg
Subarachnoid haemorrhage	85% aneurysmal; CT sensitivity >95% within 6 h; LP after 12 h; nimodipine 60 mg 4-hourly × 21 days; vasospasm days 4–14; mortality 30–40%
Carotid disease	Symptomatic 70–99% → CEA, ARR ~16%, NNT ~6, within 2 weeks; risk thresholds <6% symptomatic, <3% asymptomatic
Hypoxic-ischaemic encephalopathy	Prognosticate at ≥72 h off sedation, normothermic; temperature target ≤37.5 °C for 36–72 h; MRI at 2–7 days
Traumatic brain injury	Seizure prophylaxis 7 days only; depression up to 50%; screen pituitary at 3–6 and 12 months
Wernicke encephalopathy	Complete triad in only 10–16%; Caine criteria = any 2 of 4; thiamine 500 mg IV tds × 2–3 days; ~80% progress to Korsakoff
Cluster headache	Attacks 15–180 min, up to 8/day; O_2 12–15 L/min effective in ~70%; sumatriptan 6 mg SC; verapamil 240–960 mg/day; galcanezumab 300 mg
Tension headache	Chronic = ≥15 days/month for >3 months; limit analgesics to <15 days/month; amitriptyline from 10 mg nocte
Peripheral neuropathy	Prevalence 8–15% over age 55
CSF	Normal: 0–5 cells, protein 15–45 mg/dL, glucose ≥60% of blood; pyogenic 1000–5000 neutrophils; TB 100–500 lymphocytes; CSF lactate >3.5 mmol/L in bacterial
Serotonin syndrome	Onset <24 h; Hunter criteria sensitivity 84%, specificity 97%; cyproheptadine 12 mg then 2 mg 2-hourly
Neuroleptic malignant syndrome	Onset 1–3 days; mortality 5–10%; CK often >1000 IU/L; bromocriptine 2.5–5 mg tds; dantrolene 1–2.5 mg/kg; wait ≥2 weeks before rechallenge
Rhabdomyolysis	CK >5× ULN (>1000 IU/L); AKI risk above 5000 IU/L; AKI in 10–50%; fluids 1–1.5 L/h, urine output 200–300 mL/h
NPH	Evans index >0.3; shunt success ~80%; gait 81–86%, bladder 70%, cognition 40–44%; serious adverse events 11%
PSP / CBD	Falls within first year; median survival 6–8 years; midbrain “hummingbird sign”
MND	Riluzole prolongs survival ~3 months; 1-year survival 58% vs 49%; FTD in 10–15%; SOD1 ~2% of ALS
Myasthenia gravis	AChR antibody in ~85%; thymoma 10–15%; RNS decrement >10%; >90% remission or major improvement
SCD of cord	B12 1000 µg IM alternate days × 1–2 weeks → weekly → monthly lifelong; recovery poor if delayed >6–12 months
RLS	Ferritin target >100 ng/mL, transferrin saturation >20%; augmentation in 50–70% of long-term dopamine agonist users; PLMS in ~80%
BPPV	Posterior canal 85–90%; nystagmus latency 1–5 s, duration <60 s; Epley success 80–90%; recurrence 15–50%; vitamin D threshold <20 ng/mL
Subdural haematoma	Acute <3 days / subacute 3 days–3 weeks / chronic >3 weeks; recurrence 10–30%; anticoagulation restart ~4 weeks
Stroke reperfusion	Alteplase 0.9 mg/kg (max 90 mg); tenecteplase 0.25 mg/kg (max 25 mg); sICH 2–6%; thrombectomy NNT ~2.6; BP limits 185/110 pre-lysis, 180/105 post-lysis
Post-stroke depression	Affects ~one-third of survivors; peaks 3–6 months; continue antidepressant ≥6 months after remission
Giant cell arteritis	ESR often >50 (classically >75) mm/h; prednisolone 40–60 mg/day; IV methylprednisolone 500–1000

	mg × 3 days; second eye involved in 25–50% within 1–2 weeks; taper 12–24 months
Trigeminal neuralgia	Pain <2 minutes; classical type 80–90%; carbamazepine 600–1200 mg/day, 70–80% initial response; MVD ~70–80% pain-free at 10 years

B. Named Trials, Guidelines and Regulatory Decisions (2023–2026)

Trial / Guideline	One-line message
ILAE Elderly Task Force (Epilepsia 2023)	Lamotrigine, levetiracetam and lacosamide first line in older adults; gabapentin last resort.
ELUCID (2025)	Late-onset unexplained epilepsy is a marker of future dementia and stroke, not just of seizures.
SANAD II	Levetiracetam was neither superior nor cost-effective versus lamotrigine in focal epilepsy.
Cenobamate	High seizure-freedom rates in drug-resistant focal epilepsy; slow 12-week titration to avoid DRESS.
ANNEXA-I (NEJM 2024)	Andexanet improved haemostasis (67.0% vs 53.1%) but doubled thrombotic events (10.3% vs 5.6%); no mortality benefit.
INSPIRES (NEJM 2023)	Clopidogrel + aspirin up to 72 h reduces 90-day stroke after minor stroke or high-risk TIA.
CHANCE / POINT	DAPT for 21 days within 24 h after high-risk TIA or minor stroke.
SPS3	After lacunar stroke: systolic <130 mmHg helps; long-term DAPT causes harm.
AHA/ASA 2023 aSAH Guideline	Secure the aneurysm within 24 h; early enteral nimodipine; **prophylactic hypervolaemia is harmful**; no routine statin or magnesium.
CREST	Stenting carries higher periprocedural stroke than endarterectomy, especially over age 70.
CREST-2 (NEJM 2025)	Asymptomatic ≥70% stenosis: stenting 2.8% vs 6.0% (p=0.02); **endarterectomy no better than intensive medical therapy** (3.7% vs 5.3%).
AHA/ASA 2026 AIS Guideline	Tenecteplase Class 1; thrombolyse disabling deficits regardless of NIHSS; thrombectomy ASPECTS 3–10 and up to 24 h; thrombectomy for basilar occlusion.
ERC/ESICM 2025 Post-Resuscitation	Fever prevention at ≤37.5 °C for 36–72 h replaces hypothermia at 33 °C; no prehospital cold fluids.
AHA Post-Cardiac Arrest 2025 (Circulation)	NfL added alongside NSE for neuroprognostication; temperature control ≥36 h; predictors of *favourable* outcome now included.
Galcanezumab (consensus 2024)	Only approved preventive for **episodic** cluster headache; failed in chronic cluster headache.
AAN Painful Diabetic Neuropathy Guideline	TCAs, SNRIs, gabapentinoids and sodium-channel blockers are equally effective; **do not offer opioids or tramadol**.
AMBITION-cm / WHO	Single high-dose liposomal amphotericin B 10 mg/kg + flucytosine + fluconazole for cryptococcal meningitis; CrAg is the preferred rapid test.
WHO / NTEP	Xpert MTB/RIF Ultra is the initial CSF test for suspected tuberculous meningitis.
AGS Beers Criteria 2023	Avoid SSRI + tramadol; avoid antipsychotics for BPSD; amitriptyline is potentially inappropriate in older adults.
PENS (NEJM 2025)	First placebo-controlled trial of shunting in iNPH — improves gait and balance, not cognition or continence.
NIO752 / LM11A-31; PSP Platform Trial	Tau-directed therapies in phase 3 for PSP; none yet approved.
VALOR + OLE (JAMA Neurol 2026); **EAN 2024**	Tofersen slows decline and prolongs survival in SOD1-ALS; genetic testing now recommended for all ALS.
Nipocalimab (FDA Apr 2025, EC Dec 2025)	FcRn blocker for AChR/MuSK-positive generalised MG; joins efgartigimod, rozanolixizumab and the C5 inhibitors.
NICE NG239 (Mar 2024)	MMA or homocysteine as confirmatory test for B12 deficiency; never delay replacement when SCD is suspected.
AASM RLS Guideline (JCSM 2025)	Alpha-2-delta ligands and iron are first line; dopamine agonists demoted because of augmentation.
Vitamin D RCTs (2024–2025)	Vitamin D supplementation reduces BPPV recurrence when 25(OH)D <20 ng/mL.
EMBOLISE, STEM, MAGIC-MT (NEJM, 20 Nov 2024)	Middle meningeal artery embolisation reduces recurrence of chronic subdural haematoma.
AHA/ASA 2026 AIS Guideline	Tenecteplase Class 1; thrombolysis 4.5–9 h with mismatch; thrombectomy ASPECTS 3–10 and up to 24 h.
VA/DoD Stroke Rehab 2024; FOCUS/AFFINITY/EFFECTS IPD meta-	Do not use antidepressants to *prevent* post-stroke depression; fluoxetine increases falls, fractures and seizures.

analysis (Int J Stroke 2024)	
SELECT-GCA (upadacitinib, 2025)	Sustained remission 46.4% vs 29.0%; first oral JAK inhibitor approved for giant cell arteritis.
BTX-A meta-analysis (Clin J Pain 2024)	Botulinum toxin A reduces pain and attack frequency in refractory trigeminal neuralgia.

C. Drug or Intervention of Choice — One Line Each

Condition	Drug / intervention of choice
New-onset focal epilepsy in the elderly	Levetiracetam or lamotrigine (avoid enzyme inducers)
Status epilepticus	IV lorazepam, then IV levetiracetam / fosphenytoin / valproate
Seizure in a patient on an anticoagulant and a statin	Levetiracetam (no CYP450 interaction; IV form available)
Drug-resistant focal epilepsy	Cenobamate, or referral for pre-surgical evaluation
High-risk TIA / minor stroke (non-cardioembolic)	Aspirin + clopidogrel for 21 days, then single agent
TIA with atrial fibrillation	DOAC anticoagulation — not antiplatelet therapy
Lacunar stroke	Single antiplatelet + high-intensity statin + systolic BP <130 mmHg
Aneurysmal subarachnoid haemorrhage	Endovascular coiling within 24 h + oral nimodipine 21 days
Symptomatic carotid stenosis 70–99%	Carotid endarterectomy within 2 weeks
Asymptomatic carotid stenosis	Intensive medical therapy (CREST-2)
Wallenberg syndrome	Stroke unit care + nil by mouth and formal swallow assessment
Comatose survivor of cardiac arrest	Temperature control ≤37.5 °C for 36–72 h + delayed multimodal prognostication
Suspected Wernicke encephalopathy	IV thiamine 500 mg tds, given before any glucose
Acute cluster headache attack	High-flow oxygen 12–15 L/min (or subcutaneous sumatriptan 6 mg)
Cluster headache prevention	Verapamil (galcanezumab for episodic cluster if verapamil fails)
Chronic tension-type headache	CBT and exercise first; amitriptyline (or nortriptyline) as preventive
Painful diabetic neuropathy in the elderly	Duloxetine or a reduced-dose gabapentinoid — never an opioid
Tuberculous meningitis	Xpert MTB/RIF Ultra on CSF + ATT with adjunctive corticosteroids
Cryptococcal meningitis	Single-dose liposomal amphotericin B 10 mg/kg + flucytosine + fluconazole
Serotonin syndrome	Stop the drug + benzodiazepines (cyproheptadine if severe)
Neuroleptic malignant syndrome	Stop the drug + fluids + bromocriptine ± dantrolene
Rhabdomyolysis	Aggressive isotonic saline — no bicarbonate, no mannitol
Normal pressure hydrocephalus	Ventriculoperitoneal shunt after a positive tap test or lumbar drainage
PSP / CBD	No disease-modifying drug — trial of levodopa, botulinum toxin for dystonia, falls prevention
Motor neuron disease	Riluzole + non-invasive ventilation (tofersen if SOD1 mutation)
Myasthenia gravis	Pyridostigmine + prednisolone (azathioprine as steroid-sparing); IVIG or plasma exchange in crisis
Subacute combined degeneration	Parenteral vitamin B12 — never folate first
Restless legs syndrome	Iron replacement + alpha-2-delta ligand (gabapentin or pregabalin)
BPPV	Epley canalith repositioning manoeuvre — not vestibular sedatives
Chronic subdural haematoma	Burr-hole drainage with drain ± middle meningeal artery embolisation
Acute ischaemic stroke <4.5 h	Tenecteplase or alteplase; thrombectomy for large-vessel occlusion
Post-stroke depression	SSRI (escitalopram or sertraline) — avoid tricyclics
Central post-stroke pain	Amitriptyline or pregabalin — not simple analgesics
Giant cell arteritis	Immediate high-dose corticosteroid (IV methylprednisolone if visual loss) + aspirin + bone protection
Trigeminal neuralgia	Carbamazepine (oxcarbazepine if poorly tolerated)

Format Reminder for the Examination Hall

- **20 marks = 17 points** (14 + 3) • **15 marks = 12 points** (10 + 2) • **7 marks = 7 points** (6 + 1) • **6 marks = 6 points** (5 + 1). Do not pad beyond the count.
- Bold or box the keywords the examiner scans for: definition, diagnostic criterion, cut-off value, first-line drug, drug of choice, dangerous complication.
- **One named trial with a year and a number**, correctly quoted, outscores three vague sentences.
- Where textbook and current practice differ — dopamine agonists in RLS, triple-H therapy in SAH, surgery for asymptomatic carotid stenosis, targeted temperature management, prophylactic antidepressants after stroke, enzyme-inducing antiseizure drugs — **say so explicitly in one line**. Examiners reward it.

MD GERIATRIC MEDICINE

EXAMINATION ANSWER COMPILATION

BPSD · CAREGIVER SUPPORT · REVERSIBLE DEMENTIAS · MCI · VASCULAR DEMENTIA

Questions 21–28

Strict mark-wise format observed throughout

7-mark answer = **6 textbook points + 1 latest update = exactly 7 points** 15-mark answer = **10 textbook points + 2 latest updates = exactly 12 points**

Primary source: *Pathy's Principles and Practice of Geriatric Medicine, 6th edition · Kane's Essentials of Clinical Geriatrics* **Supplemented with:** guidelines and evidence 2023 – present, with Indian clinical and policy context

Prepared for: Sudarsan — MD Geriatric Medicine, Postgraduate Examination Revision

QUESTION 21

What are the behavioural and psychological symptoms of dementia? Describe their types, etiology, investigations and line of management, including the modes of action, indications and side effect profile of the drugs used.

RECURRING TOPIC — ASKED 3 TIMES. Appears both as a 15-mark essay and as a 7-mark note. **Both versions are given below**, followed by extra recall scaffolding.

15-Mark Answer

Textbook Core — 10 Points

1. Definition. Neuropsychiatric symptoms (NPSs), also called **behavioural and psychological symptoms of dementia (BPSD)**, are **signs and symptoms of disturbed perception, thought content, mood or behaviour** occurring in a person with dementia. They may appear **before, during or after** the diagnosis and may be the **earliest sign of incipient cognitive decline**.

2. Epidemiology. 90–98% of people with dementia develop at least one NPS over the disease course. NPSs occur in **all dementia types and all stages**. In AD the commonest is **apathy**, followed by **depression, aggression, anxiety, sleep disturbance, irritability, appetite disturbance, motor problems, delusions, disinhibition, hallucinations and euphoria**.

3. Types — five clusters. (i) Psychosis — delusions (commonly theft, infidelity, misidentification, phantom boarder) and hallucinations; **(ii) Affective** — depression, anxiety, apathy, elation; **(iii) Hyperactive/agitation** — aggression, restlessness, pacing, wandering, disinhibition, screaming; **(iv) Vegetative** — sleep–wake reversal, sundowning, appetite and eating change, hyperorality; **(v) Care-related** — rejection of care, resistance to bathing and dressing.

4. Aetiology — patient factors. Neurodegeneration with cholinergic, serotonergic and frontal–subcortical circuit disruption, plus **acute medical issues — pain, infection (urinary, respiratory), constipation, urinary retention, dehydration, drug change, poor sleep and sensory impairment**. **Pain is a common and frequently missed cause of agitation**, and sometimes a trial of scheduled analgesia is the only way to prove it.

5. Aetiology — caregiver and environmental factors. Unmet needs (hunger, boredom, toileting, need for company), **pre-existing personality and psychiatric illness**, **caregiver factors** (communication style, unrealistic expectations, countertherapeutic approach) and **environmental factors** (clutter, poor lighting, over- or under-stimulation, unfamiliar surroundings, lack of routine).

6. Investigations. Exclude delirium first using the **Confusion Assessment Method (CAM)** — acute onset and fluctuating course, plus inattention, plus disorganised thinking or altered consciousness. Then: **urinalysis and culture, complete blood count, blood glucose, blood urea, electrolytes (sodium, potassium in mEq/L), calcium (mg/dL), liver function tests, TSH (μIU/mL), vitamin B12 (pg/mL), oxygen saturation, ECG (QTc before any antipsychotic), chest radiograph if indicated, and a full medication review**. **Pain assessment** using observational tools (PAINAD, Abbey) in non-verbal patients, and imaging only if focal signs, head injury or rapid change.

7. Symptom assessment tools. Neuropsychiatric Inventory (NPI, NPI-Q, NPI-C) for the symptom profile; **Cohen-Mansfield Agitation Inventory (CMAI)** for agitation; **Cornell Scale for Depression in Dementia (CSDD)** for mood.

8. Management — the DICE framework. Describe the behaviour and its context and triggers; **Investigate** modifiable patient, caregiver and environmental causes; **Create** a plan built on **five strategies — caregiver education, effective communication, meaningful activity, task simplification with structured routine, and a safe simplified environment**; **Evaluate** effectiveness, caregiver distress and unintended effects, and **consider dose reduction or withdrawal of any psychotropic used**.

9. Non-pharmacological management is first line. Family and paid caregiver interventions have the strongest evidence and a greater effect size (0.34) than antipsychotics for behavioural symptoms. Efficacy is best in **mild and moderate stages**. **Music therapy, exercise and tailored activity** work best embedded within caregiver-directed programmes. **Training and supervising paid caregivers in communication or person-centred care skills is effective for severe agitation, immediately and up to six months afterward.**

10. Pharmacological principles. Drugs are reserved for **three "medication as first-line" scenarios — major depression, psychosis, or aggression with risk of harm to self or others** — and for acute safety crises. **International Delphi consensus** endorses an escalating approach: identify causes → caregiver training, environmental adaptation, person-centred care and tailored activities → then **citalopram and analgesia before antipsychotics**; but for **psychosis specifically, risperidone is prioritised**. **Valproate and carbamazepine are ineffective and harmful; benzodiazepines are for crises only; cholinesterase inhibitors and memantine do not help NPS in AD but do help in DLB and PDD.**

Drug Table — Mode of Action, Indications and Side Effects

Drug	Mode of action	Indication in BPSD	Key adverse effects
Risperidone	D2 and 5-HT2A antagonist	**Best evidence for short-term (6–12 wk) agitation and aggression**; licensed in EU/Canada for severe aggression	**Stroke, increased mortality**, extrapyramidal symptoms, hyperprolactinaemia, sedation, falls
Olanzapine	Multi-receptor (D2, 5-HT2, H1, M1) antagonist	Agitation, psychosis	Sedation, **anticholinergic and metabolic effects**, weight gain, hyperglycaemia, stroke, mortality
Quetiapine	D2/5-HT2A antagonist, low D2 affinity	**Agent of choice with clozapine in DLB and PDD** psychosis	Sedation, postural hypotension, falls; least extrapyramidal risk
Aripiprazole	D2 partial agonist, 5-HT1A partial agonist	Agitation, psychosis	Akathisia, sedation, stroke, mortality
Haloperidol	Potent D2 antagonist	**Avoid in dementia**; delirium only, lowest effective dose	**Severe extrapyramidal effects; catastrophic neuroleptic sensitivity in DLB**; QTc prolongation
Citalopram	SSRI	**Agitation** (CitAD trial, target 30 mg), anxiety, irritability	**QTc prolongation and cognitive decline at 30 mg**; hyponatraemia (SIADH), falls, GI upset
Sertraline	SSRI	**Major depression in dementia** (DIADS)	Hyponatraemia, GI upset, bleeding risk with NSAIDs, tremor
Trazodone	Serotonin antagonist and reuptake inhibitor	**Night-time agitation and sleep disturbance**; stabilises circadian rhythm	**Sedation**, postural hypotension, falls, rarely priapism
Lorazepam / oxazepam	GABA-A positive allosteric modulator	**Crisis use only**, short- to intermediate-acting preferred	Sedation, falls, **paradoxical agitation, respiratory depression, worsened cognition, dependence**
Cholinesterase inhibitors	Inhibit acetylcholinesterase, raising synaptic acetylcholine	**Not for NPS in AD**; effective for psychiatric and cognitive symptoms in **DLB and PDD**	Nausea, vomiting, diarrhoea, **bradycardia and syncope**, weight loss; **worsens tremor** in PD
Memantine	**Uncompetitive, voltage-dependent, low-affinity NMDA receptor antagonist**	Moderate–severe AD cognition; **cognitive and global benefit in DLB but no psychiatric benefit**	Dizziness, headache, confusion; **case reports of worsening delusions and hallucinations in DLB**
Pimavanserin	Selective **5-HT2A inverse agonist**, no dopaminergic action	**Parkinson's disease psychosis only**	QTc prolongation, peripheral oedema, confusion

Latest Updates (2023–Present)

11. Agitation in AD now has two licensed drugs. Brexpiprazole (Rexulti) — a **serotonin–dopamine activity modulator (D2 and 5-HT1A partial agonist, 5-HT2A antagonist)** — received **FDA approval in May 2023**, the first approval for any neuropsychiatric symptom of dementia, showing about **31% reduction in agitation versus placebo (CMAI effect size ~0.35)**, with benefit on CGI-S significant only at 3 mg. It retains the **class boxed warning for increased mortality in elderly patients with dementia-related psychosis**, plus akathisia, somnolence and weight gain.

12. The first non-antipsychotic option has arrived. Dextromethorphan–bupropion (AXS-05, Auvelity) was FDA-approved in 2026 for agitation in AD dementia: **dextromethorphan is an NMDA receptor antagonist and sigma-1 agonist**, while **bupropion inhibits CYP2D6** to sustain dextromethorphan levels. **ADVANCE-1** showed CMAI improvement versus placebo at week 5; **ACCORD-2** showed **delayed relapse (HR 0.276) and reduced relapse (8.4% vs 28.6%)** over 26 weeks. It carries a **boxed warning for suicidal thoughts and behaviours** and is contraindicated in **seizure disorder, eating disorders, recent alcohol or benzodiazepine withdrawal, and with MAOIs**.

7-Mark Answer

Textbook Core — 6 Points

1. BPSD/NPS = disturbed perception, thought content, mood or behaviour in dementia; **90–98%** of patients develop at least one, across **all dementia types and stages**; **apathy is commonest in AD**, followed by depression and aggression.

2. Five symptom clusters: psychosis (delusions, hallucinations), **affective** (depression, anxiety, apathy), **hyperactive/agitation** (aggression, wandering, disinhibition), **vegetative** (sleep reversal, sundowning, eating change) and **care-related** (rejection of care).

3. Aetiology is multifactorial: neurodegeneration; **acute medical triggers — pain, infection, constipation, retention, dehydration, drug change, poor sleep, sensory loss**; unmet needs; caregiver communication and expectations; and environmental over- or under-stimulation.

4. Investigations: rule out delirium with the CAM, then urinalysis and culture, CBC, **glucose, urea, electrolytes (mEq/L), calcium (mg/dL)**, LFT, **TSH (μIU/mL)**, **B12 (pg/mL)**, oxygen saturation, **ECG for QTc before any antipsychotic**, medication review, and observational pain assessment (PAINAD, Abbey). Quantify with **NPI, CMAI and Cornell Scale**.

5. Non-pharmacological measures are first line and follow the **DICE approach (Describe, Investigate, Create, Evaluate)**, with **caregiver education, communication training, meaningful activity, task simplification with routine, and a safe simplified environment**. **Caregiver interventions (effect size 0.34) outperform antipsychotics.**

6. Drugs are reserved for major depression, psychosis, or aggression risking harm. Risperidone has the best short-term evidence for agitation (stroke and mortality risk); **quetiapine or clozapine** in DLB/PDD; **citalopram** helps agitation but causes **QTc prolongation and cognitive decline at 30 mg**; **trazodone** for night-time agitation; **benzodiazepines for crises only**; **valproate and carbamazepine are ineffective**; **ChEIs and memantine do not help NPS in AD** but do in DLB and PDD.

Latest Update — 1 Point

7. Two drugs are now formally licensed for agitation in AD dementia: **brexpiprazole (FDA 2023)**, a serotonin–dopamine activity modulator with an effect size of about 0.35 and a boxed mortality warning; and **dextromethorphan–bupropion (AXS-05, FDA 2026)** — **NMDA antagonist plus sigma-1 agonist with CYP2D6 inhibition** — the **first non-antipsychotic** option, which delayed relapse in **ACCORD-2 (HR 0.276)**.

Current Practice Update

Area	Textbook	Current recommendation	Exam approach
Licensed drugs for agitation	**None approved**; all off-label except pimavanserin in PD psychosis	**Brexpiprazole (2023)** and **dextromethorphan–bupropion (2026)** approved	State the textbook position first, then add both approvals as the update
First-line management	Non-pharmacological, caregiver-directed	**Unchanged and reinforced** — DICE, person-centred care, tailored activity, music therapy	Always open the management section with non-pharmacological measures
SSRIs for agitation	CitAD positive at 30 mg with QTc and cognitive cost	**S-CitAD (escitalopram, 2024) negative** — SSRI role remains uncertain	Quote both trials to show balance

Extra Recall Box — Recurring Topic

Mnemonic for the reversible triggers to check before prescribing: "PINCH ME" **P**ain · **I**nfection · **N**utrition and dehydration · **C**onstipation · **H**ydration/**H**ypoxia · **M**edication · **E**nvironment

Numerical anchors: NPS in **90–98%** · caregiver intervention effect size **0.34** · risperidone efficacy window **6–12 weeks** · brexpiprazole effect size **~0.35** · ACCORD-2 relapse **8.4% vs 28.6%**

Named trials to quote: DIADS and DIADS-2 (sertraline) · **HTA-SADD** (sertraline vs mirtazapine) · **CitAD** and **S-CitAD** (citalopram, escitalopram) · **WHELD** (person-centred care) · **ADVANCE-1 and ACCORD-2** (AXS-05) · **HARMONY** (pimavanserin)

Examiner traps: 1. Reaching for an antipsychotic before excluding **pain, infection, constipation and retention**. 2. Forgetting that **delirium must be excluded first** — the CAM takes two minutes. 3. Prescribing **haloperidol in DLB** — potentially catastrophic neuroleptic sensitivity. 4. Claiming ChEIs help behaviour in AD — **they do not**; they help in DLB and PDD. 5. Omitting the **ECG/QTc** before citalopram or an antipsychotic. 6. Continuing psychotropics indefinitely — **the "E" of DICE demands a withdrawal trial**.

15-second answer skeleton: Define → 90–98% prevalence → five clusters → aetiology in three domains (patient, caregiver, environment) → exclude delirium and PINCH ME triggers with named investigations → **DICE** and caregiver-directed non-pharmacological care first → drugs only for depression, psychosis or dangerous aggression, with the mode-of-action/indication/side-effect table → the two new licensed agents → Indian context (family caregiving, DMHP, unregulated antipsychotic access).

Exam One-Liners

- **90–98%** of people with dementia develop at least one NPS.
- **Apathy is the commonest NPS in AD.**
- **DICE = Describe, Investigate, Create, Evaluate.**
- Caregiver interventions **(effect size 0.34) beat antipsychotics**.
- **Risperidone** = best short-term evidence; **quetiapine/clozapine** in DLB.
- **Citalopram 30 mg** = agitation benefit at the cost of **QTc prolongation and cognitive decline**.
- **Valproate and carbamazepine are ineffective and harmful.**
- **ChEIs and memantine help NPS in DLB/PDD, not in AD.**
- Memantine = **uncompetitive NMDA antagonist**; may worsen hallucinations in DLB.
- New licences: **brexpiprazole 2023, dextromethorphan–bupropion 2026**.

QUESTION 22

Non-pharmacological management of behavioural disturbances in dementia.

RECURRING TOPIC — ASKED 3 TIMES as a 7-mark note. Extra recall scaffolding is given after the answer.
Pivot from Question 21: Question 21 covers the whole BPSD syndrome including drugs. This answer is pivoted entirely to **the non-drug evidence base and its practical delivery**, and should not repeat the pharmacology.

7-Mark Answer

Textbook Core — 6 Points

1. Rationale and evidence. Non-pharmacological measures are **first-line for all behavioural disturbance in dementia**. A meta-analysis of **23 randomised trials in nearly 3300 community-dwelling patient–caregiver dyads** confirmed that caregiver-directed interventions **significantly reduce behavioural symptoms with an effect size of 0.34 — greater than that of antipsychotics for behavioural symptoms and of cholinesterase inhibitors for memory**. Efficacy is best in **mild and moderate stages**, when cognition and communication are better preserved.

2. The DICE framework. Describe the behaviour, its context and its triggers with the caregiver; **I**nvestigate modifiable causes across patient, caregiver and environmental domains; **C**reate a collaborative plan; **E**valuate whether the target symptom improved, caregiver distress fell and no unintended effects occurred. DICE was designed for **any health professional working within a team**.

3. The five caregiver-training strategies with the strongest evidence base. Train caregivers to **(i) use problem-solving skills to manage NPS; (ii) increase activity for the person with dementia; (iii) enhance communication within the dyad; (iv) reduce environmental complexity; and (v) simplify tasks for the person with dementia** — supplemented by **establishing structured routines and ensuring safety**.

4. Interventions targeting the person with dementia. Music therapy, aromatherapy, exercise, tailored activity programmes, reminiscence and multisensory stimulation. Individual approaches work best **embedded within tailored-activity programmes** rather than delivered alone. **Bright light therapy with good sleep hygiene is first-line for circadian rhythm disorder**, for which there is no evidence for drug treatment.

5. Interventions targeting professional caregivers. In nursing homes, **training and supervising paid caregivers in communication or person-centred care skills is effective for symptomatic and severe agitation, immediately and up to six months afterward**. A **systematic review and meta-analysis of 11 randomised trials** showed psychosocial interventions in nursing homes **reduced psychotropic drug prescribing**, using three intervention classes — **staff training on psychotropic prescribing and NPS management, consultation with external experts for complex NPS, and person-centred psychosocial interventions**.

6. Interventions targeting the environment. Address **over- or under-stimulation, safety problems, lack of activity and structure, and absence of established routine**; add **architectural modification improving secure access to the outdoors and helping navigation** of the home or institution. Because strategies are used in combination, **no single approach can be identified as preferred — a combination of environmental adjustments yields the behavioural change**, and individual environmental studies remain too small and disparate for firm conclusions.

Latest Update — 1 Point

7. International Delphi expert consensus now formally places **caregiver training, environmental adaptation, person-centred care and tailored activities ahead of any pharmacological approach** for overall NPS and agitation, and identifies **DICE and music therapy as the two most promising non-pharmacological approaches**. This is reinforced by **person-centred care packages such as WHELD** and by **Cochrane reviews of music-based interventions (2022–2025)** showing consistent modest benefit on agitation and depressive symptoms **with no drug-related harm** — while **digital and telehealth delivery of caregiver skills training** (web-based psychoeducation, telecoaching, multicomponent platforms) has been shown feasible and effective, greatly widening access in resource-limited settings including India.

Current Practice Update

Area	Textbook	Current recommendation	Exam approach
Position in the treatment ladder	First-line, but no formal consensus ranking	**Delphi consensus formally ranks non-drug measures ahead of drugs**; DICE and music therapy top the list	Say “first-line by expert consensus, not merely by default”
Delivery mode	Face-to-face caregiver programmes	**Digital and telehealth delivery** validated — critical for India’s dispersed families	Add one line on telehealth to modernise the answer
Environmental interventions	Too small and disparate to draw conclusions	Position largely unchanged; **combination approaches** remain the recommendation	Do not overclaim environmental evidence

Extra Recall Box — Recurring Topic

Mnemonic for the five caregiver-training targets: “PACES” Problem-solving skills · **A**ctivity increased for the patient · **C**ommunication enhanced within the dyad · **E**nvironmental complexity reduced · **S**implified tasks

Numerical anchors: caregiver intervention meta-analysis — **23 trials, ~3300 dyads, effect size 0.34** · nursing-home psychosocial meta-analysis — **11 trials, reduced psychotropic prescribing** · paid-caregiver training benefit sustained **up to 6 months**

Named entities: DICE (Kales, Gitlin and Lyketsos, BMJ 2015) · **Brodaty and Arasaratnam meta-analysis** · **WHELD** · **Cochrane music-based interventions**

Examiner traps: 1. Listing activities without the **framework** — DICE is what earns the structure marks. 2. Forgetting that the intervention target is often **the caregiver, not the patient**. 3. Claiming strong evidence for **environmental modification alone** — the textbook explicitly says the studies are too small and disparate. 4. Omitting the **nursing-home/paid-caregiver** limb of the evidence. 5. Missing **bright light therapy and sleep hygiene** as first-line for circadian disturbance.

15-second answer skeleton: State first-line status with the effect size 0.34 → **DICE** → five caregiver-training targets (**PACES**) → patient-directed therapies (music, exercise, tailored activity, bright light) → professional caregiver training reduces psychotropic prescribing → environmental modification in combination → Delphi consensus and digital delivery → Indian application through family caregivers, ARDSI and DMHP.

Exam One-Liners

- Caregiver-directed interventions: **effect size 0.34, greater than antipsychotics**.
- **DICE = Describe, Investigate, Create, Evaluate.**

- Five training targets: **problem-solving, activity, communication, reduced complexity, simplified tasks**.
- Best efficacy in **mild and moderate** stages.
- Paid-caregiver **person-centred care training works for severe agitation, sustained to 6 months**.
- Nursing-home psychosocial interventions **reduce psychotropic prescribing**.
- **Bright light therapy + sleep hygiene** = first-line for circadian rhythm disorder.
- **Environmental interventions work in combination**, not singly.
- **DICE and music therapy** are the two consensus-endorsed approaches.

QUESTION 23

How will you address caregiver stress in dementia?

7-Mark Answer

Textbook Core — 6 Points

1. Recognise the scale and the domains of impact. Caregiving affects the carer's **physical health, mental health, financial well-being, social support and spiritual well-being**. Caregivers of people with dementia are particularly vulnerable, and **caregiver counselling and support is listed among the evidence-based preventive interventions in geriatric psychiatry, with the specific outcome of preventing caregiver depression**.

2. Screen and quantify the burden. Use validated instruments: the **Zarit Burden Interview (22 items, with validated 3-item and shortened versions and translations into many languages)**; the **Caregiver Self-Assessment Questionnaire (CSAQ)**, an 18-item American Medical Association tool **also validated as a screen for depression**; the **Rapid Screen for Caregiver Burden (7 items)**; and the **Caregiver Well-Being Scale**. **These tools allow a quick assessment of needs and stress levels so that appropriate referrals can be made.**

3. Psychoeducation and skills training. The largest body of evidence supports **psychoeducational programmes that teach problem-solving techniques for behavioural problems**, including **cognitive behavioural family intervention**, structured behaviour-management programmes, and **combined education and support programmes supplemented by follow-up telephone support**. In one three-country randomised trial, **five sessions of counselling plus ad hoc telephone support combined with cholinesterase inhibitor therapy in the patient reduced caregiver depression from moderate to lower levels**.

4. Mindfulness, yoga and meditation. Mindfulness-Based Stress Reduction (MBSR) — formal practice (mindful movement, body scan, sitting meditation) plus informal practice in everyday life — improves psychological well-being in healthy and clinical populations. A **twice-weekly online mindfulness intervention reduced caregiver burden and improved quality of life**, and **yoga and compassion-meditation programmes improved quality of life and self-compassion in family caregivers of people with Alzheimer's disease**.

5. Support groups and respite. Support groups are of two types: **time-limited groups training caregivers in care delivery and role management**, and **ongoing forums for sharing experience and mutual support**, the latter providing lasting social support and social activity. Caregivers generally evaluate them positively, but **not all are comfortable meeting others, and substitute care arrangements may be needed to allow the caregiver to attend**. **Respite and adult day care** are core components of multidimensional caregiver programmes, and **adult day programmes reduce the risk of accelerated cognitive decline associated with nursing home placement**.

6. Treat the dyad, not the patient alone, and respect culture. Interventions have traditionally focused on one party rather than **the dyad of caregiver and care recipient**, and new strategies must support **both**. Caregiving practices vary across cultures: in **traditional Asian cultures the family is expected to care for elders at home, and suggesting a care facility may be perceived as abandonment and is culturally insensitive** — directly relevant to Indian practice. Other proven measures include **family intervention to delay institutionalisation** and **exercise plus behavioural management to reduce disability in the person with dementia**, both of which reduce the demands placed on the caregiver.

Latest Update — 1 Point

7. Digital and telehealth caregiver support has been validated and is the most scalable answer for India. Randomised and systematic-review evidence (2024–2026) for **web-based psychoeducation, telecoaching, multicomponent online platforms and telehealth-adapted caregiver skills training** shows reductions in burden and depression comparable to face-to-face delivery. Indian-specific work is now available: **LASI-DAD (2025)** provides the first nationally representative data on dementia caregiving in India, analysing **4196 informants** and demonstrating that the care recipient's cognitive impairment measurably worsens caregiver **stress, mental health and positive affect**; and a **coproduced digital media caregiver intervention developed at NIMHANS (CTRI-registered)** has established feasibility and acceptability for Indian family caregivers.

Current Practice Update

Area	Textbook	Current recommendation	Exam approach
Delivery of caregiver support	Face-to-face programmes, telephone follow-up noted as promising	**Digital, web-based and telehealth delivery validated** as effective and scalable	Mention telephone support from the textbook, then extend to digital platforms
Indian evidence	Cultural discussion, largely Western data	**LASI-DAD (2025)** — first nationally representative Indian caregiving data	Quote LASI-DAD to anchor the Indian angle
Target of intervention	Caregiver-focused	**Dyadic interventions** supporting caregiver and care recipient together	Use the word "dyad" — it is the current framing

Practical Indian Framework to Quote

- **Assess:** Zarit Burden Interview (Hindi and regional-language versions validated) at every dementia review
- **Educate:** diagnosis, expected course, behavioural management, safety, driving, finances, advance planning
- **Support: ARDSI** chapters — caregiver training, memory clinics, day care and support groups
- **Relieve:** respite through day care, planned short-stay admission, shared family rotas
- **Refer: District Mental Health Programme** for caregiver depression; **NPHCE** geriatric clinics; **Elder Line 14567**
- **Protect: Maintenance and Welfare of Parents and Senior Citizens Act, 2007** where caregiving has broken down; **AB-PMJAY / Ayushman Vay Vandana Card** for those 70+; **Rashtriya Vayoshri Yojana** for assistive devices
- **Anticipate:** screen the caregiver's **own** blood pressure, glycaemia, sleep and mood — the caregiver is frequently an older adult too

Exam One-Liners

- Screen with the **Zarit Burden Interview**; **CSAQ (18 items) also screens for depression**; **Rapid Screen for Caregiver Burden = 7 items**.
- **Caregiver counselling and support prevents caregiver depression** — an evidence-based preventive intervention.
- Five sessions of counselling plus telephone support **reduced caregiver depression** in a three-country RCT.
- **MBSR, yoga and compassion meditation** improve caregiver quality of life.
- **Adult day programmes reduce accelerated cognitive decline** linked to nursing home placement.
- **Family intervention delays institutionalisation.**
- Treat the **dyad**, not the caregiver alone.
- In Asian cultures, **suggesting a care facility may be read as abandonment** — counsel with cultural sensitivity.
- **LASI-DAD (2025)**: first nationally representative Indian dementia caregiving data, **4196 informants**.

QUESTION 24

What are the innovations you would suggest to ease caregiver burden?

Pivot from Question 23: Question 23 asks how you would *address* caregiver stress with established interventions. This answer is pivoted to **innovation and service design** — technology, new models of delivery, workforce and policy — and should not simply repeat the psychoeducation and support-group list.

7-Mark Answer

Textbook Core — 6 Points

1. Information and communication technology as a delivery channel. The textbook already identifies that **the use of telephone support highlights the potential of alternative modes of delivery to enhance or replace face-to-face interventions**, and reviews **the impact of information and communication technology on family carers of older people and professionals**. Innovation therefore begins by **moving proven interventions onto remote platforms** rather than inventing new content — **telephone, video, app and web-based delivery of psychoeducation, counselling and behaviour-management training**.

2. Online and remote psychological interventions. A **twice-weekly mindfulness intervention delivered online was correlated with a decrease in caregiver burden and an increase in quality of life** — direct textbook support for remote delivery of stress-reduction programmes, extendable to **online yoga and compassion meditation**, which improved quality of life and self-compassion in caregivers of people with Alzheimer's disease.

3. Telemedicine and telepsychiatry for the care recipient. Telemedicine prevents psychiatric admissions from long-term care homes, and **geriatric telepsychiatry has a role in long-term care psychiatry, in the assessment and management of dementias in primary care, in geriatric emergencies in the emergency department, and in managing depression in primary care through collaborative care**. Every avoided admission or avoided journey is caregiver burden removed.

4. Ambient and assistive technology in the home. Context-aware computing and context-awareness — technology that senses the situation and responds — together with **design and technology for older people** and **assistive devices**, reduce supervision demand. Practical applications are **wandering alerts and GPS location, fall detection, medication reminders and automated dispensers, stove and door sensors, and video check-in**. **Security must be built in**, since these systems carry recognised risks of **denial of service and identity theft**.

5. Restructured services and new models of care. Community-based, multidisciplinary geriatric mental health treatment teams are the ideal model of community psychiatry service delivery, supported by **hospital-based consultation-liaison services**, **care home liaison services** and **memory clinics** giving **informed discussion of diagnosis and prognosis with arrangements for ongoing review, support and management**. **Adult day programmes reduce the risk of accelerated cognitive decline associated with nursing home placement**, and **respite and adult day care** are core components of multidimensional caregiver programmes.

6. Reduce demand at source and fund the workforce. Burden falls when the patient's condition is better managed: **exercise plus behavioural management reduces disability in people with dementia**; **comprehensive nutritional treatment prevents weight loss**; **family intervention delays institutionalisation**; and **DICE training for family and professional caregivers manages agitation and aggression without psychotropic drugs**. Structurally, **more funding and new payment models are needed to train health professionals, primary care providers and family caregivers in assessing and managing NPS**, and **primary care providers should be reimbursed for time spent on approaches like DICE and given better access to backup specialist services**.

Latest Update — 1 Point

7. Digital caregiver platforms have moved from pilot to evidence. Randomised trials and systematic reviews (2024–2026) of **home-based digital interventions — web psychoeducation, telecoaching, digital cognitive training, assistive technology and multicomponent platforms** — show reductions in caregiver burden and depression, with dedicated platforms (**GamePlan4Care, TeleCARE**) tested in randomised designs, and **nature-based and community respite models** under formal evaluation. For India specifically, a **coproduced digital media caregiver resource developed at NIMHANS and registered with CTRI** has demonstrated feasibility and acceptability for Indian family caregivers — the model most likely to scale, given that **India has one of the world's fastest-growing dementia populations, over 90% of care is family-provided, and formal respite and long-term care infrastructure is scarce**.

Innovations Grid — Ready-Made Answer Structure

Level	Innovation	Burden relieved
Technology	App and web psychoeducation, telecoaching, online MBSR, video support groups	Access, isolation, travel time
Home	GPS/wandering alerts, fall detection, automated pill dispensers, door and stove sensors, video check-in	Supervision load, night-time vigilance
Clinical service	Memory clinic with case manager, telepsychiatry, care home liaison, single point of contact	Fragmented care, repeated hospital visits
Respite	Adult day care, planned short-stay respite, community and nature-based respite programmes	Physical exhaustion, loss of employment
Workforce	DICE training for family and paid caregivers; reimbursement for caregiver-facing time	Behavioural crises, antipsychotic prescribing
Peer	Dementia-friendly communities, self-help and peer-caregiver networks, employer flexibility	Social isolation, financial strain
Policy (India)	NPHCE geriatric clinics, DMHP linkage, Elder Line 14567, Ayushman Vay Vandana Card (70+), Rashtriya Vayoshri Yojana, ARDSI day care	Cost, legal insecurity, unmet need

Exam One-Liners

- Start with **ICT delivery of proven interventions** — do not invent new content.
- **Online mindfulness reduced caregiver burden and improved quality of life.**
- **Telemedicine prevents psychiatric admissions from long-term care homes.**
- **Context-aware computing** and assistive technology reduce supervision demand — but carry **DoS and identity-theft risks**.
- **Adult day programmes reduce accelerated cognitive decline** linked to nursing home placement.
- **Family intervention delays institutionalisation**; **exercise plus behavioural management reduces disability**.
- **DICE training manages agitation without psychotropics.**
- Structural need: **funding, training and reimbursement for caregiver-facing time**.
- India: **>90% family-provided care**, scarce respite — **digital and community models are the realistic innovation**.

QUESTION 25

What are the reversible dementias? How would you investigate and treat them?

RECURRING TOPIC — ASKED 3 TIMES as a 7-mark note. Extra recall scaffolding follows the answer.

7-Mark Answer

Textbook Core — 6 Points

1. Definition and scale. Dementia is grouped into **reversible or partially reversible** and **non-reversible** types. **Reversible dementia accounts for probably less than 4% of cases**, but **concurrent medical conditions causing mild cognitive disturbance are much commoner and are potentially treatable**; identifying them **avoids unnecessary disability and misdiagnosis**. Crucially, **finding a reversible cause does not guarantee that the dementia will improve once the cause is treated**.

2. The causes. Drugs (anticholinergics and antihistamines, analgesics including narcotics and NSAIDs, anticonvulsants, antihypertensives, antiparkinsonian agents, digoxin, antiarrhythmics, H2 blockers, antianxiety drugs, antidepressants, antipsychotics, sedative-hypnotics, muscle relaxants, steroids, alcohol); **psychiatric — depression ("depressive pseudodementia")**; **metabolic and endocrine** (hypothyroidism, hyperparathyroidism, hyponatraemia, hypercalcaemia, hepatic and renal failure); **nutritional** (**vitamin B12**, folate, thiamine — Wernicke–Korsakoff, pellagra); **neoplasms**; **trauma — chronic subdural haematoma**; **toxins** (alcohol, heavy metals, organic poisons); **infections** (HIV and opportunistic infection, neurosyphilis, tuberculous meningitis); **autoimmune** (CNS vasculitis, temporal arteritis, SLE, multiple sclerosis); and **other — normal-pressure hydrocephalus**.

3. Clinical evaluation. These disorders are detected by careful history, physical examination and selected laboratory studies. Take a **complete drug history including over-the-counter agents**, maintain a **high index of suspicion for excessive alcohol intake** (easily missed, and a cause of dementia, delirium, depression and falls), and screen for depression. **Pointers to**

depressive pseudodementia are **prominent complaints of memory loss, patchy and inconsistent cognitive deficits, and frequent "don't know" answers**.

4. Laboratory investigation. Complete blood count, glucose, blood urea nitrogen, electrolytes (sodium and potassium in mEq/L), calcium and phosphorus (mg/dL), liver function tests, thyroid-stimulating hormone (μIU/mL), vitamin B12 (pg/mL), serological test for syphilis if suspected, and HIV antibodies if suspected. These blood tests are **directed at finding common comorbidities rather than at identifying the cause of the underlying dementia**.

5. Imaging and neuropsychology. CT or MRI of the head should be done in **dementia of recent onset with no other clinical finding to explain it, and in those with focal neurological signs or symptoms**, to exclude **subdural haematoma, tumour and normal-pressure hydrocephalus**. **CT and MRI are roughly equivalent for detecting most remediable structural lesions**; MRI shows more lesions in multi-infarct dementia but also white-matter changes of uncertain significance. **Neuropsychological testing** is particularly useful for **differentiating depression from dementia** and where mental-status score and functional status are discordant.

6. Treatment. Discontinue whenever feasible every drug known to impair cognition, especially strong anticholinergics. **Treat depression energetically** — an SSRI such as **citalopram or sertraline**, or the NaSSA **mirtazapine**, all with minimal cognitive side effects; where doubt persists, **a careful trial of an antidepressant is justified** both diagnostically and therapeutically. **Correct B12 deficiency, hypothyroidism, hyponatraemia and hypercalcaemia; refer normal-pressure hydrocephalus for shunt assessment and chronic subdural haematoma for neurosurgery; treat neurosyphilis, HIV and tuberculous meningitis specifically; and control vascular risk factors, especially hypertension**, which may favourably influence the course of both degenerative and vascular cognitive impairment. Note that **thyroid hormone and vitamin B12 replacement may be important for general health but have not been shown to reverse cognitive deficits**, and that **older patients with reversible cognitive impairment during depression are at high risk of later dementia and need close follow-up**.

Latest Update — 1 Point

7. Shunting for idiopathic normal-pressure hydrocephalus now has randomised, placebo-controlled proof. The **PENS trial (NEJM, 2025)** randomised **99 patients across 17 centres** to open versus placebo (closed) shunt valve settings: **gait velocity improved by 0.21 m/s more with an open shunt (95% CI 0.12–0.31; p<0.001)**, with **80% versus 24% achieving meaningful improvement**, better balance and fewer falls — but **no significant improvement in cognition or continence at 3 months**. Alongside this, **antibody-mediated autoimmune encephalitis (LGI1, CASPR2, NMDAR, GAD65)** is now an established, immunotherapy-responsive cause of subacute cognitive decline, to be suspected with **rapid progression over weeks to months, seizures, hyponatraemia, movement disorder or psychiatric prominence**.

Current Practice Update

Area	Textbook	Current recommendation	Exam approach
NPH shunting	Surgery, with risk–benefit carefully assessed; efficacy debated	**PENS (2025)** — randomised proof of gait benefit; cognition and continence do not reliably improve	Quote the numbers and the nuance
Autoimmune causes	CNS vasculitis, SLE, MS	**Antibody-mediated encephalitis** a distinct, treatable category	Add autoantibody panel to the workup for rapid decline
B12 and thyroid replacement	Not shown to reverse cognitive deficits	**Unchanged** — treat for general health, not expecting cognitive reversal	State this explicitly; it is a marking point

Extra Recall Box — Recurring Topic

Mnemonic — "DEMENTIA": Drugs and **D**epression · **E**motional/**E**ndocrine · **M**etabolic · **E**yes and **E**ars (sensory deprivation) · **N**utritional and **N**PH · **T**umour and **T**rauma (subdural) · **I**nfection and **I**nflammatory · **A**lcohol and toxins

Numerical anchors: reversible dementia **<4%** · depression coexists in **>1/3** of dementia outpatients · rapidly progressive dementia = decline within **12 months** · **PENS: 0.21 m/s gait gain; 80% vs 24%**

Examiner traps: 1. Overstating frequency — it is **<4%**. 2. Claiming B12 or thyroid replacement reverses dementia — **it has not been shown to**. 3. Forgetting that **drugs are the commonest single reversible contributor**. 4. Omitting **NPH and chronic subdural haematoma** — both surgically treatable. 5. Diagnosing dementia while delirium is present — **the diagnosis must await treatment of all reversible causes of delirium**.

15-second answer skeleton: Define and state <4% → causes by system (drugs, depression, metabolic/endocrine, nutritional, structural, infective, autoimmune, NPH) → history, drug review, alcohol history, depression screen → named blood panel plus CT/MRI → treat each specifically, deprescribe first → caveat that reversal is not guaranteed → PENS and autoimmune encephalitis as the update → Indian angle (**B12 deficiency, hypothyroidism, neurosyphilis, HIV, TB meningitis are all commoner here**).

Exam One-Liners

- **<4%** of dementias are truly reversible.
- **Finding the cause does not guarantee reversal.**
- **Drugs first** — anticholinergics are the commonest culprit.
- Depressive pseudodementia: **prominent memory complaints, patchy deficits, "don't know" answers**.
- Panel: **CBC, glucose, urea, electrolytes, calcium/phosphorus, LFT, TSH, B12, VDRL and HIV if suspected + CT/MRI**.
- Blood tests find **comorbidities, not the cause of the dementia**.
- **CT and MRI are equivalent** for remediable structural lesions.
- Structural causes: **subdural haematoma, tumour, NPH**.
- **PENS 2025**: shunting improves **gait**, not cognition or continence.

QUESTION 26

Mild cognitive impairment — evaluation, treatment and outcome.

RECURRING TOPIC — ASKED TWICE as a 7-mark note. Extra recall scaffolding follows the answer.
Pivot: the emphasis here is on **evaluation, treatment and outcome** rather than on the full biomarker classification — the criteria are stated compactly and the weight is placed on the clinical workup, management and prognosis.

7-Mark Answer

Textbook Core — 6 Points

1. Definition and criteria. MCI is **(i) a subjective disorder of memory and/or other cognitive domains, (ii) objective impairment of memory and/or other cognitive domains, and (iii) no significant impact on activities of daily living** — so patients **do not meet criteria for dementia**. The **NIA-AA core clinical criteria** are **concern about a change in cognition** (from patient, informant or clinician), **impairment in one or more cognitive domains greater than expected for age and education**, **preservation of independence in functional abilities** (mild difficulty with complex tasks is allowed, with minimal aids), and **not demented**. **No thresholds are defined** for the degree of impairment. The DSM-5 equivalent is **mild neurocognitive disorder**.

2. Clinical evaluation. History from **both patient and an informant**, focused on **the impact on usual activities, lifestyle, the history of the cognitive disorder, and above all whether functional decline has occurred** — the point that separates MCI from dementia. **Old age alone does not cause cognitive impairment severe enough to render a person dysfunctional**, so decline must not be dismissed as ageing.

3. Cognitive and functional testing. A **standardised mental status test** plus **neuropsychological testing**, which is especially useful when the mental status score is normal but function or behaviour has changed (high baseline intelligence) or the score is low without functional deficit (low educational level), and for **differentiating depression from dementia**. Function is documented with **Lawton IADL and basic ADL scales**; mood with the **Geriatric Depression Scale**.

4. Exclude the treatable — the same three questions used for any confusional presentation. (i) Has the onset been acute (hours to days)? (ii) Are there physical factors — medical illness, sensory deprivation, drugs? (iii) Are psychological factors — depression, psychosis — contributing? Investigate with **complete blood count, glucose, blood urea, electrolytes (mEq/L), calcium and phosphorus (mg/dL), liver function tests, TSH (μIU/mL), vitamin B12 (pg/mL), syphilis and HIV serology if suspected**, and **CT or MRI head** where indicated. MCI is **heterogeneous** and may be due to **AD, other neurodegenerative disease, depression, vascular lesions or traumatic brain injury**.

5. Biomarkers where available. NIA-AA research criteria stratify by **amyloid markers (CSF Aβ42, amyloid PET)** and **neuronal injury markers (CSF total tau and p-tau, MRI atrophy, FDG-PET/SPECT hypometabolism)**: **"high likelihood" MCI due to AD = both positive; "intermediate likelihood" = one positive with the other untested; "unlikely due to AD" = both negative**. The **IWG criteria for prodromal AD** require **abnormal CSF Aβ1-42 and tau or an abnormal amyloid PET (IWG-2)**. **Most countries, including India, lack national guidelines on biomarker use**, which limits harmonisation.

6. Treatment. No intervention has classically been shown to prevent progression to dementia, and **cholinesterase inhibitors are not licensed for MCI**. Management therefore centres on: **an informed discussion of diagnosis and prognosis with arrangements for ongoing review and support**; **treating contributory conditions** — depression with **citalopram, sertraline or mirtazapine** (minimal cognitive side effects), hypothyroidism, B12 deficiency, sleep apnoea and alcohol excess; **withdrawing anticholinergic and other cognition-impairing drugs**; **controlling vascular risk factors, especially hypertension, which may favourably influence the course of both degenerative and vascular cognitive impairment**; and **physical activity and multicomponent exercise**, the intervention with the most promising evidence (**FINGER** positive for multidomain intervention; **MAPT** largely negative). Add **cognitive training, driving and safety assessment, and advance planning**.

Latest Update — 1 Point

7. MCI due to AD has become a treatable, biomarker-defined diagnosis. Lecanemab (FDA full approval July 2023, CLARITY-AD) and **donanemab (FDA approval July 2024, TRAILBLAZER-ALZ 2)** are licensed specifically for **MCI due to AD or mild AD dementia with confirmed amyloid pathology**, requiring **APOE genotyping and serial MRI for amyloid-related imaging abnormalities (ARIA)**, with the **EMA restricting use to non-APOE ε4 homozygotes**. Confirmation of amyloid is now possible by blood: the **FDA cleared the Lumipulse G pTau217/Aβ1-42 plasma ratio in May 2025** for symptomatic adults aged 55 and over, and the **Alzheimer's Association Clinical Practice Guideline (2025)** endorses blood biomarkers in patients with objective cognitive impairment including MCI — as a **triaging test at ≥90% sensitivity and ≥75% specificity**, or **as a substitute for amyloid PET or CSF at ≥90% for both**. **US POINTER (2025)** confirmed benefit from structured multidomain lifestyle intervention in at-risk older adults.

Outcome — Learn These Numbers

Outcome	Figure
Prevalence of cognitive impairment without dementia, age 71+	**Just over 20%**
Annual conversion of MCI/CIND to dementia	**15–20% per year**
Remainder	A proportion **remain stable**, and some **revert to normal** — especially where depression, drugs or sleep disorder were contributory
Biomarker effect	**Biomarker-positive MCI converts far faster**; biomarker-negative MCI often remains stable
Depression-related cognitive impairment	Older patients with reversible cognitive impairment during depression are at **high risk of dementia within a few years** — follow closely

Current Practice Update

Area	Textbook	Current recommendation	Exam approach
Treatment	"No intervention has been shown to prevent progression"	**Anti-amyloid antibodies licensed at the MCI stage** — slowing, not prevention	State the textbook line, then add the caveat that slowing ≠ prevention
Amyloid confirmation	PET or CSF, limited availability	**FDA-cleared plasma p-tau217/Aβ42 ratio (2025)**	This is the single most quotable update
Lifestyle intervention	FINGER positive, MAPT largely negative	**US POINTER (2025) positive**	Quote all three trials for balance

Extra Recall Box — Recurring Topic

Mnemonic for the four core criteria — "CIPN": Concern about cognition · **I**mpairment objectively demonstrated · **P**reserved independence in function · **N**ot demented

Numerical anchors: >20% of those 71+ have CIND · **15–20%** annual conversion · biomarker thresholds **≥90/≥75** for triage, **≥90/≥90** to replace PET or CSF

Examiner traps: 1. Saying MCI has **defined cognitive thresholds** — it does not. 2. Saying **ChEIs are indicated** — they are not licensed for MCI. 3. Forgetting that MCI can **revert to normal**, especially with depression or drugs. 4. Failing to exclude depression, drugs, B12 deficiency and thyroid disease before labelling MCI. 5. Missing the functional criterion — **any significant ADL impairment makes it dementia, not MCI**.

15-second answer skeleton: Define with the three components → four NIA-AA core criteria (**CIPN**) → evaluation: informant history, cognitive and functional testing, the three screening questions, named blood panel and imaging → biomarkers and likelihood tiers → treatment: no licensed drug classically, treat contributors, deprescribe, control vascular risk, exercise and multidomain lifestyle → outcome: **20% prevalence, 15–20% annual conversion, some revert** → anti-amyloid antibodies and plasma p-tau217 as the update.

Exam One-Liners

- MCI = **subjective complaint + objective impairment + preserved ADLs + not demented**.
- **No thresholds are specified** for the degree of impairment.
- **Just over 20%** of people aged 71+ have cognitive impairment without dementia.
- **15–20% convert to dementia per year.**
- **ChEIs are not licensed for MCI.**
- **Exercise and multidomain intervention** carry the best evidence (**FINGER**, **US POINTER**).
- **Anti-amyloid antibodies are licensed at the MCI stage** when amyloid is confirmed.
- **Plasma p-tau217/Aβ42 ratio FDA-cleared (2025)** for symptomatic adults 55+.
- Reversible cognitive impairment during depression → **high risk of later dementia**.

QUESTION 27

Vascular dementia.

7-Mark Answer

Textbook Core — 6 Points

1. Definition and concept. Vascular dementia (VaD) is **dementia resulting from cerebrovascular disease**, having evolved from the older concept of **arteriosclerotic dementia**. The current and preferred umbrella term is **vascular cognitive impairment (VCI)**, which spans the full spectrum from **vascular MCI (Va-MCI) to VaD**, and includes **mixed vascular and AD pathology**. **About 5% of people over 65 have VCI**, and **in patients under 74 VCI may be the single most common cause of cognitive impairment**. The inclusion of **VCI-no dementia** emphasises the **preventable nature of VCI and the importance of early diagnosis**; the asymptomatic susceptible host with risk factors is termed the **"brain at risk"**.

2. Classification — seven subtypes. Multi-infarct (cortical) dementia from large cortical infarcts; **small-vessel (subcortical) dementia** from lacunes and white or deep grey matter lesions; **strategic infarct dementia** from a unilateral or bilateral infarct in a strategic area; **hypoperfusion dementia**; **haemorrhagic dementia**; **Alzheimer's disease with cerebrovascular disease**

(mixed); and **hereditary dementia** such as **CADASIL, due to NOTCH3 mutation on chromosome 19**. **Subcortical ischaemic vascular disease (SIVD)** — the **lacunar state and Binswanger's disease** — is the commonest subtype (**82.2% small-vessel versus 25.9% large-vessel** in one study).

3. Clinical features by vessel size. Large-vessel disease produces cortical infarcts in strategic locations giving a **cortical dementia** — **frontal lobe** (aphasia, apraxia, disinhibition, apathy), **hippocampus and basal forebrain** (amnesia), **gyrus angularis** (construction problems), **parietal lobe** (alexia, agraphia). **Small-vessel disease** affects **thalamus, caudate nucleus and internal capsule**, disrupting **frontal–subcortical or thalamocortical circuits** to give a **subcortical dementia** — **executive dysfunction, attention deficit, apathy and personality change** with **comparatively milder memory deficit** and slowed processing speed. **"Lower-half parkinsonism"** produces **hesitation, marche à petit pas and diminished step height**, and **the triad of dementia, urinary incontinence and gait disturbance is more often produced by small-vessel VaD than by normal-pressure hydrocephalus**.

4. Course. Classically **abrupt onset with stepwise deterioration and a fluctuating course with plateaus**, but in practice **60% have a slow, less abrupt onset and 80% show slow progression** with or without stepwise decline — so a gradual course does not exclude VaD.

5. Diagnosis. NINDS-AIREN criteria for probable VaD require **(i) dementia; (ii) cerebrovascular disease — focal neurological signs consistent with stroke plus neuroimaging evidence of clinically relevant vascular lesions; and (iii) a temporal relationship — onset of dementia within three months of a recognised stroke, abrupt deterioration, or fluctuating/stepwise progression**. Supportive clinical features are **early gait disturbance, unsteadiness with frequent unprovoked falls, early urinary frequency and urgency unexplained by urological disease, pseudobulbar palsy, and personality and mood change with abulia, depression, emotional incontinence, psychomotor retardation and abnormal executive function**. Imaging severity criteria include **white matter lesions affecting more than 25% of white matter**. These criteria **exclude about 90% of AD but have only modest sensitivity (50–70%)** and **misclassify mixed dementia as VaD (54% for ADDTC, 29% for NINDS-AIREN)**. MRI shows **basal ganglionic and thalamic hyperintense foci, thromboembolic infarcts, confluent white matter and irregular periventricular hyperintensities (leukoencephalopathy)**; **SPECT shows multiple focal deficits**; and **cerebral microbleed number independently predicts cognitive impairment and dementia severity**.

6. Management — four-pronged. (i) Symptomatic treatment for cognition: three donepezil and two galantamine studies show **modest improvement of about two points on the VADAS-Cog**, with inconsistent effects on activities of daily living, behaviour and global function; two memantine studies show **modest cognitive benefit but no global benefit**. **There is no recommendation to prescribe cholinesterase inhibitors for pure VaD, but they should be used in mixed dementia once both AD and vascular components are demonstrated. (ii) Management of neuropsychiatric disturbance — depression is common with stroke and SIVD and is eminently treatable, so an antidepressant course is justified where suspected**. **(iii) Stroke prevention** — control of vascular risk factors, especially **hypertension**, remains **the most effective strategy**; **aspirin's effect on cognition and prognosis remains unclear**, and **antithrombotics should be used with caution when lobar microbleeds are seen on MRI** because of amyloid angiopathy. **Neither pravastatin (PROSPER) nor atorvastatin (LEADe) improved cognition. (iv) Management of stroke-related disability** — spasticity, parkinsonism and incontinence; **levodopa can help apraxic gait in SIVD** when the vascular burden lies in the basal ganglia or substantia nigra. **Physical activity and multicomponent exercise combining aerobic and strength training** have the most promising preventive evidence.

Latest Update — 1 Point

7. The field has moved from "vascular dementia" to biologically and clinically harmonised vascular cognitive impairment. The **VASCOG statement** and the harmonised **VICCCS** criteria have superseded NINDS-AIREN and ADDTC, aligning with **DSM-5 major and mild vascular neurocognitive disorder**, and **STRIVE-2 (2023)** standardised the neuroimaging reporting of small-vessel disease (**lacunes, white matter hyperintensities, perivascular spaces, microbleeds, cortical superficial siderosis, incidental infarcts**). Therapeutically, **intensive blood pressure lowering remains the only intervention with randomised evidence for cognitive benefit** — **SPRINT MIND** reduced incident MCI and the combined MCI-plus-dementia outcome, and its extended follow-up supports sustained benefit — reinforced by the **Lancet Commission 2024**, which added **high midlife LDL cholesterol (population attributable fraction 7%)** to hypertension, diabetes, smoking, obesity and physical inactivity among the modifiable determinants of vascular brain injury.

Current Practice Update

Area	Textbook	Current recommendation	Exam approach
Nomenclature	Vascular dementia; NINDS-AIREN and ADDTC criteria	**Vascular cognitive impairment (VCI)**; **VASCOG/VICCCS** criteria aligned to DSM-5	Use VCI as the umbrella term and NINDS-AIREN for the operational criteria
Imaging reporting	Descriptive leukoencephalopathy and infarcts	**STRIVE-2 (2023)** standardised small-vessel disease terminology	Name the six STRIVE features
Prevention	Vascular risk factor control, no randomised cognitive endpoint quoted	**SPRINT MIND** — intensive BP lowering reduced incident MCI	Best single trial to quote for VaD prevention
Symptomatic drugs	ChEIs not recommended for pure VaD; use in mixed dementia	**Unchanged**	State plainly; it is a common marking point

Exam One-Liners

- **VCI** is the preferred umbrella term; **~5% of those over 65** have VCI; **under 74 it may be the commonest cause of cognitive impairment**.
- **Seven subtypes**; **SIVD (lacunar state and Binswanger's) is commonest**.
- **CADASIL = NOTCH3 mutation, chromosome 19.**
- **Large vessel → cortical dementia; small vessel → subcortical dementia.**

- NINDS-AIREN needs **dementia + cerebrovascular disease + temporal relationship (within 3 months of stroke, abrupt deterioration, or stepwise course)**.
- White matter lesions must affect **>25% of white matter**.
- Criteria **exclude 90% of AD but have only 50–70% sensitivity** for VaD.
- **60% have slow onset, 80% slow progression** — stepwise decline is not obligatory.
- **The gait–incontinence–dementia triad is commoner in small-vessel VaD than in NPH.**
- **ChEIs are not recommended in pure VaD**; use in **mixed dementia**; benefit ≈ **2 points on VADAS-Cog**.
- **PROSPER and LEADe negative** for statins and cognition; **SPRINT MIND positive** for intensive BP lowering.
- **Levodopa may help apraxic gait** in SIVD.

QUESTION 28

Differentiating features of delirium, depression and dementia.

7-Mark Answer

Textbook Core — 6 Points

1. The three screening questions. Any older person presenting with confusion should be assessed with three questions that separate the three D's: **(i) Has the onset of abnormalities been acute — over a few hours or a few days?** (delirium); **(ii) Are there physical factors — medical illness, sensory deprivation, drugs — that may contribute?** (delirium or reversible dementia); **(iii) Are psychological factors — depression and/or psychosis — contributing to or complicating the impairment?** (depression). **These questions focus on identifying treatable conditions which, when diagnosed and treated, might result in substantially improved cognitive function.**

2. Delirium. An **acute or subacute alteration in mental status**, with **prevalence of about 15% on admission in hospitalised geriatric patients and incidence up to one-third**; it may **persist for days or weeks** and so is common in post-acute care. Key features are **disturbance of consciousness**, **change in cognition not better accounted for by dementia**, **development over hours to days with fluctuation through the day**, and **evidence of a causative medical condition, substance intoxication, side effect or withdrawal**. **The disturbances of consciousness and attention, with sudden onset and fluctuating cognitive status, are the major features that distinguish delirium from other causes of impaired cognitive function.** There is **difficulty sustaining attention, sensory misperception (illusions), fragmented or disordered stream of thought, psychomotor disturbance (restlessness, picking at bedclothes, or sluggishness and drowsiness) and emotional disturbance**; **neurological signs other than asterixis are uncommon**.

3. Dementia. A **clinical syndrome of sustained loss of intellectual function and memory sufficient to cause dysfunction in daily living**, with **a gradually progressing course over months to years** and **no disturbance of consciousness**. **Loss of functional ability due to impaired cognition is the feature that distinguishes dementia from MCI.**

4. Depression ("depressive pseudodementia"). Refers to patients with **reversible or partially reversible impairment of cognitive function caused by depression**. **Depression may coexist with dementia in more than one-third of outpatients with dementia and in an even greater proportion in nursing homes**, and the relationship is complex: **many patients with early dementia become depressed, and depression may be diagnosed in many patients before they become demented**. Helpful clinical characteristics are **prominent complaints of memory loss, patchy and inconsistent cognitive deficits on examination, and frequent "don't know" answers**.

5. Tools and testing. Delirium is screened with the **Confusion Assessment Method (CAM)**, which requires **acute onset and fluctuating course, AND inattention, AND either disorganised thinking OR altered level of consciousness**; **inattention is tested by months of the year backward, days of the week backward, or digit span backward**, and **disorganised thinking by orientation to time and place and observation of illogical flow of ideas**. Depression is quantified with the **Geriatric Depression Scale**, or the **Cornell Scale for Depression in Dementia** where dementia coexists. **Detailed neuropsychological testing, performed by someone skilled in these tools, is helpful in differentiating depression and dementia**, and **where uncertainty persists after full assessment, a careful trial of an antidepressant is justified** to facilitate diagnosis and improve function and quality of life.

6. Why the distinction matters, and the pitfalls. Differentiating delirium from dementia is important because the latter is not immediately life-threatening, and inappropriately labelling a delirious patient as demented may delay diagnosis of serious and treatable underlying medical conditions. It is not possible to diagnose dementia while delirium is present in a patient with previously normal or unknown cognitive function — **the diagnosis of dementia must await treatment of all potentially reversible causes of delirium**. Conversely, **attributing symptoms common in dementia — disorientation, agitation — to the dementia itself leads to under-diagnosis of delirium and to missing a serious underlying illness**. The practical strategy when both are possible is to **focus on the core diagnostic symptoms of abnormal attention and awareness and altered level of consciousness or alertness, rather than on the behavioural symptoms** — especially useful when no reliable informant is available. **Sundowning** — increased confusion at night, especially in pre-existing dementia — is probably related to **sensory deprivation in unfamiliar surroundings**, and such patients **may actually meet the criteria for delirium**.

Latest Update — 1 Point

7. Delirium superimposed on dementia (DSD) is now a defined clinical entity in its own right, present in a large proportion of hospitalised people with dementia and associated with **prolonged length of stay, accelerated long-term cognitive decline and poor outcomes**. Detection has been simplified by **ultra-brief validated tools — the 4AT, the 3D-CAM, the brief CAM (bCAM) and the Ultra-Brief 2-item screener** — which are practical at the bedside and in emergency departments; the **NICE delirium guidance (updated 2023)** and Indian practice both favour the **4AT** because it requires no special training and can be completed in under two minutes even in an uncooperative patient. Alongside this, the **"depression–dementia continuum"** is now better characterised: **late-life depression with cognitive impairment carries a substantially raised risk of subsequent dementia**, so such patients require **structured cognitive follow-up rather than reassurance**, and **plasma p-tau217** can help distinguish depression-related cognitive impairment from underlying Alzheimer pathology in specialist settings.

Master Differentiation Table

Feature	Delirium	Dementia	Depression
Onset	**Acute — hours to days**	**Insidious — months to years**	Subacute — weeks to months, often datable
Course	**Fluctuating through the day**, worse at night	Slowly progressive, stable through the day	Diurnal variation, typically **worse in the morning**
Consciousness	**Reduced clarity of awareness — the defining feature**	**Clear until terminal stages**	Clear
Attention	**Impaired — cardinal feature**	Normal early, impaired late	Poor concentration but attention preserved on testing
Orientation	Impaired, fluctuating	Impaired as disease advances	Usually intact
Memory complaint	Not volunteered	**Concealed or minimised**; informant reports it	**Prominently complained of**
Cognitive deficits	Global, fluctuating	Consistent, progressive	**Patchy and inconsistent**
Answers on testing	Wrong, inconsistent, distractible	**Near-miss or confabulated** answers, effortful	**Frequent "don't know"**, poor effort
Psychomotor activity	**Hyperactive, hypoactive or mixed**; picking at bedclothes	Usually normal until late	**Retardation** or agitation
Perception	**Illusions and hallucinations common** (visual)	Hallucinations late (early in DLB)	Uncommon; mood-congruent if psychotic
Thought	**Fragmented, disordered, illogical**	Impoverished	Themes of guilt, worthlessness, hopelessness
Sleep–wake cycle	**Markedly disrupted, reversed**	Fragmented, sundowning	**Early morning waking**
Neurological signs	**Uncommon except asterixis**	Late focal or extrapyramidal signs	Absent
Reversibility	**Usually reversible** if the cause is treated	Largely irreversible (<4% reversible)	**Reversible with treatment**
Screening tool	**CAM, 4AT, 3D-CAM**	MMSE, MoCA, informant history	**GDS, Cornell Scale**

Current Practice Update

Area	Textbook	Current recommendation	Exam approach
Delirium screening	CAM (4 criteria)	**4AT and 3D-CAM** for ultra-brief bedside and emergency use	Name CAM first, then the 4AT as the practical update
Delirium with dementia	Recognised as difficult	**DSD is a defined entity** with worse outcomes; focus on **attention and level of consciousness**, not behaviour	Use the phrase "delirium superimposed on dementia"
Depression with cognitive impairment	High risk of later dementia; follow closely	**Confirmed**; **plasma p-tau217** can identify underlying AD pathology	Mention structured follow-up, not reassurance

Exam One-Liners

- The three discriminators: **onset, attention and level of consciousness**.
- **Delirium: acute, fluctuating, inattentive, clouded consciousness.**
- **Dementia: insidious, progressive, clear consciousness.**
- **Depression: prominent memory complaints, patchy deficits, "don't know" answers.**
- Delirium prevalence **~15% on admission**, incidence **up to one-third**.
- **CAM = acute onset and fluctuation + inattention + (disorganised thinking OR altered consciousness).**
- **Neurological signs other than asterixis are uncommon in delirium.**
- **You cannot diagnose dementia while delirium is present.**
- Depression coexists with dementia in **over one-third** of outpatients.
- **Sundowning patients may actually meet criteria for delirium.**
- When both are possible, judge by **attention and alertness, not by behaviour**.

CONSOLIDATED NIGHT-BEFORE MASTER SHEET

Questions 21–28 on One Page

#	Topic	Marks	The single fact most likely to earn the mark
21	BPSD — types, aetiology, investigation, management, drugs	15 + 7	**90–98%** develop at least one NPS; **DICE**; **brexpiprazole 2023 and dextromethorphan–bupropion 2026** now licensed for agitation
22	Non-pharmacological management of behavioural disturbance	7	Caregiver-directed interventions: **23 trials, ~3300 dyads, effect size 0.34 — greater than antipsychotics**
23	Addressing caregiver stress	7	**Zarit Burden Interview**; caregiver counselling **prevents caregiver depression**; treat the **dyad**
24	Innovations to ease caregiver burden	7	**ICT delivery of proven interventions**; **telemedicine prevents psychiatric admissions from LTC homes**
25	Reversible dementias	7	**<4% truly reversible**; **treatment does not guarantee reversal**; **PENS 2025 — gait improves, cognition does not**
26	Mild cognitive impairment	7	**>20% of those 71+ have CIND**; **15–20% convert per year**; **anti-amyloid antibodies now licensed at MCI stage**
27	Vascular dementia	7	**VCI is the umbrella term**; **ChEIs not recommended for pure VaD, use in mixed**; **SPRINT MIND** for prevention
28	Delirium vs depression vs dementia	7	Judge by **onset, attention and level of consciousness**; **you cannot diagnose dementia during delirium**

The Twelve Numbers to Carry Into the Hall

Fact	Number
Dementia patients developing at least one NPS	**90–98%**
Caregiver intervention effect size on behavioural symptoms	**0.34**
Brexpiprazole CMAI effect size	**~0.35**
AXS-05 relapse rate versus placebo (ACCORD-2)	**8.4% vs 28.6%**
Truly reversible dementias	**<4%**
Depression coexisting with dementia in outpatients	**>1/3**
Cognitive impairment without dementia, age 71+	**>20%**
MCI annual conversion to dementia	**15–20%**
VCI prevalence over age 65	**~5%**
White matter lesion threshold, NINDS-AIREN	**>25% of white matter**
Delirium prevalence on admission / incidence in hospital	**~15% / up to one-third**
PENS trial gait velocity gain (iNPH shunting)	**0.21 m/s; 80% vs 24% improved**

Indian Policy Sentences to Drop Into Any of These Answers

- **LASI-DAD:** dementia prevalence **7.4%** in adults 60+, about **8.8 million cases**, projected **16.9 million by 2036**; caregiving data from **4196 informants (2025)** confirm measurable caregiver stress and mental-health impact
- **Over 90% of dementia care in India is family-provided**, with scarce respite and long-term care infrastructure — the case for **digital and community-based caregiver support**
- **NPHCE** — Regional Geriatric Centres, district geriatric units and CHC/PHC geriatric clinics: the platform for cognitive and caregiver screening
- **National and District Mental Health Programme** — the practical route for BPSD management and caregiver depression
- **AB-PMJAY and the Ayushman Vay Vandana Card** — cover for all citizens aged 70 and above regardless of income
- **Maintenance and Welfare of Parents and Senior Citizens Act, 2007** — legal remedy where caregiving breaks down
- **Rights of Persons with Disabilities Act, 2016** — disability certification including chronic neurological conditions and mental illness
- **Rashtriya Vayoshri Yojana / AVYAY** — assistive devices and senior citizen welfare

- **Elder Line 14567** — national toll-free helpline
- **ARDSI** — caregiver training, day care, support groups, Dementia India reports
- **WHO ICOPE (2nd edition)** — cognitive decline as one of the six domains of intrinsic capacity screened in primary care

End of compilation — Questions 21 to 28.

Parkinsonism, Movement Disorders & the Ageing Brain

MD Geriatric Medicine — Strict Mark-Wise Answers (Q53–Q60)

Primary sources: Pathy's Principles and Practice of Geriatric Medicine (6th ed.) and Essentials of Clinical Geriatrics (7th ed.)
Latest updates from guidelines and peer-reviewed evidence to August 2026 | Indian context integrated

Contents

53.	**15 marks**	Motor and non-motor symptoms of Parkinson's disease; treatment modalities and side effect profile, with special reference to the geriatric population RECURS
54.	**7 marks**	Principles of therapy in a 65-year-old with newly diagnosed PD, predominant rigidity and minimal tremor
55.	**7 marks**	Levodopa RECURS
56.	**7 marks**	Drug-induced parkinsonism
57.	**15 marks**	Multiple system atrophy — clinical features, types, diagnosis, prognosis and treatment RECURS
58.	**7 marks**	Essential tremor RECURS
59.	**7 marks**	Movement disorders in the elderly
60.	**15 marks**	Ageing changes in the central nervous system including the brain, and the degenerative diseases of the brain

Format used in this compilation

- **15-mark essay = exactly 12 points** — 10 textbook core points (1–10) plus 2 latest updates (11–12).
- **7-mark note = exactly 7 points** — 6 textbook core points (1–6) plus 1 latest update (point 7).
- Tables, algorithms and panels are **revision aids, not extra points** — they compress content you would otherwise write as prose.
- A **Current Practice Update** appears only where the textbook meaningfully differs from current recommendations.
- Topics flagged RECURS carry a green **Extra Points to Remember** panel.

Q53. Enumerate the motor and non-motor symptoms of Parkinson's disease. What treatment modalities are available and what is the side effect profile of the drugs used, with special reference to the geriatric population? RECURS

15-Mark Answer | 10 Textbook Points + 2 Latest Updates = 12 Points

Source: Pathy's 6th ed., Chapter 53 (Tables 53.3, 53.4); Essentials of Clinical Geriatrics 7th ed., Chapter 10 (Table 10-6).

Textbook Core — 10 Points

1. **Definition.** Parkinson's disease is the **second most common neurodegenerative disease**, caused by **loss of substantia nigra neurons with striatal dopamine depletion** and **α-synuclein Lewy bodies**. Prevalence is **about 1% over age 65**.
2. **Cardinal motor symptoms. Bradykinesia** (obligatory), **4–6 Hz rest tremor**, **rigidity** and **postural instability**. Onset is **asymmetric or unilateral** and becomes bilateral as the disease progresses.
3. **Other motor symptoms.** Difficulty in moving, writing and walking; **flexed posture of limbs and trunk**; **reduced step length and height with reduced arm swing**; **falls**; expressionless face; freezing; dysarthria and dysphagia in advanced disease.
4. **Non-motor — neuropsychiatric and cognitive. Bradyphrenia** (slowness of mental function); **depression in about 50%**; anxiety, mainly **generalised anxiety disorder and obsessive–compulsive disorder**; visual hallucinations and delusions; **PD-related dementia only after several years** — cognitive impairment is **never a sign of early PD**.
5. **Non-motor — autonomic.** Dominated by **orthostatic hypotension** (fall of **20 mmHg systolic or 10 mmHg diastolic within three minutes of standing**, often worsened by antiparkinsonian drugs) and **constipation in about 50%**. Also sweating, drooling, sexual dysfunction, urinary frequency, urgency and dysuria, and **delayed gastric emptying in at least 70%**.
6. **Non-motor — other. Hyposmia**, insomnia, REM sleep behaviour disorder, pain and fatigue. In advanced disease, insomnia, pain, sweats, dysphagia, dysarthria, drooling and urinary dysfunction cluster together.
7. **Levodopa — the mainstay in the elderly.** It is the **main and often the only treatment for PD in older people**, given with a peripheral decarboxylase inhibitor as **Madopar** (levodopa + benserazide) or **Sinemet** (carbidopa + levodopa). Effective for **tremor, bradykinesia and rigidity**. Start low, increase very gradually; usual early control on **300–600 mg/day** in 3–4 doses **30 minutes before meals**; dispersible form for **early-morning akinesia**.
8. **Side effect profile of levodopa.** Nausea, vomiting, anorexia, **orthostatic hypotension**; sedation, nightmares, vivid dreams, **hallucinations, delusions and delirium** (dose-related, especially in the old); and after several years **motor fluctuations** (delayed on, wearing-off, on–off) with **dyskinesias** — chorea, athetosis and dystonia.
9. **Other drug classes and their geriatric restrictions. Dopamine agonists are not used in older people with PD** and **anticholinergics are never used**. COMT inhibitors, amantadine and MAO-B inhibitors are adjuncts (see table).

10. **Non-drug modalities and non-motor treatment. Bilateral deep brain stimulation of the STN or GPi** for cognitively intact patients with refractory motor complications; **rehabilitation started early and timed to the peak effect of levodopa**; speech therapy for dysarthria, drooling and swallowing; home support, caregiver education and psychological support. Non-motor symptoms are treated only **after adjusting levodopa** — venlafaxine for depression, clozapine or pimavanserin for psychosis, rivastigmine for dementia and apathy, droxidopa or midodrine for orthostatic hypotension, macrogol for constipation, domperidone for nausea.

Side Effect Profile of Antiparkinsonian Drugs (revision table)

Drug	Dose	Side effects	Geriatric comment
Carbidopa–levodopa	300–600 mg/day levodopa, 3–4 doses	Nausea, vomiting, orthostatic hypotension, dyskinesia, vivid dreams, hallucinations, delirium	**Drug of choice; start low, go slow**
Dopamine agonists (pramipexole 0.5–1.5 mg tid, ropinirole 3–8 mg tid, bromocriptine)	As listed	Hallucinations, somnolence, nausea, orthostatic hypotension, syncope, behavioural change, impulse-control disorder	**Not used in older people with PD**
Anticholinergics (trihexyphenidyl 2–20 mg/day, benztropine)	As listed	Dry mouth, constipation, urinary retention, blurred vision, glaucoma exacerbation, tachycardia, **confusion and delirium**	**Never used in the elderly**
Amantadine	100–300 mg/day; 200–400 mg/day for dyskinesia	**Delirium**, hallucinations, livedo reticularis, ankle oedema	**Renally excreted — reduce dose in renal impairment**
Selegiline / Rasagiline	10 mg/day / 1 mg/day	Selegiline: nausea, confusion, agitation, insomnia. Rasagiline: balance difficulty, anorexia, weight loss, depressive symptoms	Rasagiline needs **no dietary restriction**
Entacapone / Tolcapone	200 mg with each levodopa dose / 100–200 mg tid	Nausea, dyskinesia, orthostatic hypotension, **diarrhoea**; tolcapone — **liver dysfunction**	Useful for motor fluctuations in the old
Clozapine (for psychosis)	12.5 mg start → 25–50 mg	**Neutropenia**, sedation, hypotension	**Stop permanently if neutrophils <1500/mm^3**

Latest Updates — 2 Points

11. **New levodopa delivery systems (2024–25). Crexont** (extended-release carbidopa/levodopa capsules) was FDA-approved in **August 2024**, **Vyalev** (foslevodopa/foscarbidopa) — the **first 24-hour continuous subcutaneous infusion** — in **October 2024**, and **Onapgo** (subcutaneous apomorphine infusion) in **February 2025**. **Tavapadon**, a novel **D1/D5 partial agonist**, gave an extra **1.1 hours of daily "on" time** in Phase 3 TEMPO-3 and is under FDA review.
12. **Adaptive deep brain stimulation (February 2025).** The FDA approved Medtronic **BrainSense Adaptive DBS** on the basis of the **ADAPT-PD** randomised crossover trial — the first closed-loop system, which titrates stimulation in real time to sensed **beta-band local field potentials** for patients with motor fluctuations. Contemporary data also show DBS outcomes in patients **over 70 are comparable to younger patients**, so age alone is no longer a contraindication.

Current Practice Update

Aspect	Textbook	Current recommendation
DBS in the elderly	"Not currently used in elderly people with PD"	**Age alone is not a bar**; adaptive closed-loop DBS approved February 2025
Levodopa safety	Nausea, hypotension, neuropsychiatric effects, motor complications	Add **vitamin B6 deficiency and seizures** — FDA label warning for all carbidopa/levodopa products, **March 2026**
Neuroprotection	MAO-B inhibitors "proposed for neuroprotective effects"	No disease-modifying agent exists; **Exenatide-PD3 was negative** (Lancet 2025). MAO-B inhibitors are used for **symptomatic benefit only**

Exam approach: state the textbook position first, then add the current position in one sentence — examiners reward awareness of the change, not contradiction of the textbook.

Extra Points to Remember — this topic recurs

- **Enumeration mnemonic for non-motor symptoms — "SAD CUPS":** **S**leep (insomnia, RBD), **A**utonomic (orthostatic hypotension, constipation, bladder, sexual), **D**epression and anxiety; **C**ognitive (bradyphrenia, dementia), **U**rinary, **P**ain and fatigue, **S**mell loss.
- **The single highest-yield sentence in this answer:** "Levodopa does not improve non-motor symptoms, posture or gait."
- **Two drugs to name as contraindicated: metoclopramide** (use domperidone instead) and **typical neuroleptics**.
- If the question weights "geriatric population", spend two of your ten points on the **two prohibitions** (agonists, anticholinergics) and on **orthostatic hypotension monitoring at every visit** — that is what distinguishes a geriatrics answer from a neurology answer.

Examiner's Tip — lines that earn marks

- The word "enumerate" means the examiner wants **lists** — write motor and non-motor symptoms as two clearly headed lists before you begin treatment.
- Give the three numbers: **depression ~50%, constipation ~50%, delayed gastric emptying ≥70%**.
- Reproduce the side effect table — a drug-by-drug table converts directly into marks and saves writing time.
- Close with: "**In the elderly, levodopa is the main and often the only treatment; dopamine agonists are not used and anticholinergics are never used.**"

Q54. What are the principles of therapy and treatment in a 65-year-old patient recently diagnosed with Parkinson's disease who has predominant rigidity and minimal tremor?

7-Mark Answer | 6 Textbook Points + 1 Latest Update = 7 Points

Source: Pathy's 6th ed., Chapter 53 (Tables 53.2, 53.3, 53.4; Box 53.1); Essentials of Clinical Geriatrics 7th ed.

Textbook Core — 6 Points

1. **Confirm the diagnosis before treating.** Apply the **Queen Square Brain Bank criteria**. An **akinetic-rigid presentation with minimal tremor** must prompt exclusion of other causes of extrapyramidal syndrome, which account for **20–40% of late-onset parkinsonism** — drug-induced parkinsonism (take an exhaustive neuroleptic history), vascular parkinsonism, PSP, MSA and normal pressure hydrocephalus. **Ioflupane SPECT** is normal in drug-induced parkinsonism and abnormal in PD.
2. **Levodopa is the drug of choice.** It is **effective in tremor, bradykinesia and rigidity**, and is the **main and often the only treatment for PD in the elderly**. Give it as **Madopar or Sinemet** with a peripheral decarboxylase inhibitor. Since rigidity and bradykinesia dominate here, levodopa targets exactly the symptoms present.
3. **Start low and titrate slowly.** Begin at a low dose and increase **very gradually** until adequate effect; most patients are controlled in the first years on **300–600 mg/day** in **3–4 divided doses given 30 minutes before meals**. A good response supports the diagnosis; a **negative response to large doses is an exclusion criterion for PD**.
4. **Avoid the two prohibited classes. Anticholinergics are never used in older people with PD** — and are in any case indicated for tremor, which is minimal in this patient. **Dopamine agonists are not used in older people** because adverse effects are numerous and can be severe.
5. **Anticipate and monitor adverse effects.** Warn about nausea (treat with **domperidone**, never metoclopramide), and check for **orthostatic hypotension at every visit** — a fall of 20 mmHg systolic or 10 mmHg diastolic within three minutes, frequently worsened by antiparkinsonian drugs. Watch for sedation, vivid dreams and hallucinations. Counsel that **motor fluctuations and dyskinesia appear after several years**.
6. **Start non-pharmacological therapy at once and think as a geriatrician. Functional rehabilitation should be started early as it helps preserve motor function**, with sessions **timed to the peak effect of levodopa**, combining joint flexibility, walking, posture, balance and turning exercises against the flexed attitude. Add **combined aerobic, strengthening, balance and stretching exercise**. Screen for non-motor symptoms (depression, constipation), review polypharmacy for dopamine-blocking drugs, assess falls risk, and provide **patient and caregiver education and psychological support**.

Latest Update — 1 Point

7. **Levodopa should be started early at the lowest effective dose, not withheld.** No agent has proven disease modification — the Phase 3 **Exenatide-PD3** trial (*Lancet* 2025) showed **no slowing of progression**, and MAO-B inhibitors are no longer claimed to be neuroprotective. Contemporary evidence confirms levodopa is **not toxic to nigral neurons** and that delaying it does not alter disease course, so "saving levodopa for later" is obsolete — particularly in a 65-year-old with disabling rigidity. Newer formulations (**Crexont**, August 2024) are available if wearing-off develops later.

Current Practice Update

Aspect	Textbook	Current recommendation
Timing of levodopa	Implicit — first-line in the elderly	**Explicitly start early at lowest effective dose**; no benefit from delaying
Neuroprotection	Selegiline/rasagiline "proposed for neuroprotective effects in early PD"	Prescribed for **symptomatic benefit only**; no proven disease modification

Examiner's Tip — lines that earn marks

- The examiner has deliberately written "**predominant rigidity and minimal tremor**" — your first point must show you recognise this as a **red flag for atypical parkinsonism**, not just a variant of PD.
- Second, use it to justify **not** prescribing an anticholinergic: "anticholinergics are used for tremor, which is minimal here, and are never used in the elderly."
- Write the dose precisely: "**levodopa–carbidopa started low and titrated to 300–600 mg/day of levodopa in 3–4 doses, 30 minutes before meals.**"
- End on the geriatric note: **early rehabilitation timed to peak levodopa effect, falls assessment, and caregiver education.**

Q55. Levodopa RECURS

7-Mark Answer | 6 Textbook Points + 1 Latest Update = 7 Points

Source: Pathy's 6th ed., Chapters 53 and 30; Essentials of Clinical Geriatrics 7th ed.

Textbook Core — 6 Points

1. **Mechanism.** Levodopa is the **precursor of dopamine** and, unlike dopamine, **crosses the blood-brain barrier**. It is combined with a **peripheral dopa-decarboxylase inhibitor** to lessen systemic effects — **Madopar** (levodopa + benserazide) and **Sinemet** (carbidopa + levodopa).
2. **Indication.** It is the **main and often the only treatment for Parkinson's disease in the elderly**, and is **effective in tremor, bradykinesia and rigidity**. Critically, it **does not improve non-motor symptoms, posture or gait**.
3. **Dose and administration. Start at low doses and increase very gradually.** Most patients are controlled in the first years on **300–600 mg/day**, given **three or four times daily, 30 minutes before meals**. The **dispersible form** reaches peak plasma concentration faster and is used for **early-morning akinesia**.
4. **Adverse effects. Peripheral** — nausea, vomiting, anorexia and hypotension. **Neuropsychiatric** — sedation, nightmares, vivid dreams, hallucinations, delusions and delirium, all **dose-related** and common in older patients.
5. **Long-term complications.** After several years, **motor fluctuations** appear — delayed effect of levodopa, wearing-off between doses, and the **on–off phenomenon** — together with **involuntary movements: chorea, athetosis and dystonia**. These require **revision of the timing of administration**. **Gastric emptying is delayed in at least 70%** of patients, and levodopa retained in the stomach is metabolised to dopamine and lost, causing erratic absorption that itself contributes to the on–off phenomenon.
6. **Diagnostic role.** In the **levodopa challenge**, a fasting patient is given **150–250 mg with a decarboxylase inhibitor** and tremor, rigidity and akinesia are assessed **every 30 minutes for two to three hours**. This indicates **dopaminergic responsiveness and is not a formal diagnostic test**. An **excellent 70–100% response sustained for five years or more** is a supportive criterion for PD; a **negative response to large doses is an exclusion criterion**.

Latest Update — 1 Point

7. **New formulations and a new safety warning. Crexont** (extended-release carbidopa/levodopa capsules) was FDA-approved in **August 2024** for wearing-off, and **Vyalev** (foslevodopa/foscarbidopa) in **October 2024** as the **first 24-hour continuous subcutaneous infusion**, which bypasses the gastrointestinal tract and solves the erratic gastric absorption problem **without surgery**. In **March 2026 the FDA required a new label warning for all carbidopa/levodopa products regarding vitamin B6 deficiency and seizures**.

Extra Points to Remember — this topic recurs

- **Drugs that must not be combined: metoclopramide is contraindicated** in parkinsonian patients because of its striatal dopamine receptor effects — use **domperidone**, which does not cross the blood–brain barrier (with ECG monitoring for QT prolongation).
- **Protein interaction:** dietary protein competes with levodopa for absorption — this is the reason for pre-meal dosing, and in Indian practice a high-protein evening meal (dal, paneer, milk) can blunt the last dose of the day.
- **Never stop levodopa abruptly** — abrupt withdrawal can precipitate a **neuroleptic malignant-like syndrome** with rigidity, fever and raised creatine kinase. This matters when a patient is kept nil by mouth perioperatively; use a dispersible preparation via nasogastric tube or a rotigotine patch instead.
- **Drugs that prolong or modify levodopa action:** COMT inhibitors (entacapone, tolcapone), the fixed combination **Stalevo**, and **amantadine 200–400 mg/day** for peak-dose dyskinesia.
- **The one-line definition to open with:** "Levodopa is the metabolic precursor of dopamine which, unlike dopamine, crosses the blood–brain barrier, and is given with a peripheral decarboxylase inhibitor."

Examiner's Tip — lines that earn marks

- Structure a drug note as **Mechanism → Indication → Dose → Adverse effects → Long-term complications → Precautions** — exactly six points.
- Quote the numbers: **300–600 mg/day; 3–4 doses; 30 minutes before meals; challenge 150–250 mg; ≥70% delayed gastric emptying.**
- Do not forget the negative: "**Levodopa does not improve non-motor symptoms, posture or gait.**"

Q56. Drug-induced parkinsonism

7-Mark Answer | 6 Textbook Points + 1 Latest Update = 7 Points

Source: Pathy's 6th ed., Chapters 53 (Table 53.2) and 54 (Tables 54.3, 54.4); Essentials of Clinical Geriatrics 7th ed., Chapters 10 and 14.

Textbook Core — 6 Points

1. **Definition and importance.** Drug-induced parkinsonism is an extrapyramidal syndrome defined by **exposure to neuroleptics or other drugs before the onset of parkinsonism**. It is listed among the causes **frequently observed as very late-onset parkinsonism**, and **many elderly patients, especially in long-term care institutions, have undiagnosed but treatable drug-induced parkinsonism** resulting from the extrapyramidal side effects of antipsychotics.
2. **Causative drugs. Antipsychotics** — haloperidol, fluphenazine, olanzapine; **antiemetics** — metoclopramide and prochlorperazine; and other agents implicated in drug-induced movement disorders including **calcium channel blockers, lithium, valproate, carbamazepine, phenytoin, amiodarone, antidepressants and antihistamines**.
3. **Clinical features. Dyskinesia is frequent and tremor is absent** — the reverse of idiopathic PD. The picture is usually more symmetrical, and **ioflupane (DaTSCAN) SPECT shows a normal pattern**, which distinguishes it from PD and other degenerative parkinsonian syndromes.
4. **Course after withdrawal.** When the offending drug is stopped, signs often alleviate but **very slowly, over periods as long as several weeks or months**. In **about 20% of patients some extrapyramidal signs persist**, which suggests these people had **PD or another neurodegenerative parkinsonism precipitated (unmasked) by the drug challenge**. **Tardive dyskinesia is often permanent** in many patients even after stopping the drug.
5. **Management.** There is **no satisfactory treatment** for drug-induced parkinsonism — **stopping the responsible drug is the best option**, and in elderly patients this is often possible. If severe psychosis demands treatment, **switch from a classical neuroleptic to a low-dose atypical antipsychotic**. **Anticholinergic drugs, used in younger psychiatric patients on neuroleptics, must be avoided in the elderly** because they often induce delirium and other adverse effects. For established tardive dyskinesia, options are slow down-titration of the offending agent, switching to **clozapine or quetiapine**, and specific pharmacotherapy with **valbenazine or deutetrabenazine**.
6. **Prevention. Prevention of drug-induced parkinsonism is a crucial issue in geriatric care** and is achieved by **avoiding the prescription of neuroleptics in elderly patients, especially those with dementia — a condition for which they are ineffective** for behavioural complications. Untreated parkinsonian patients eventually become immobile and develop **flexion contractures, pressure sores, malnutrition and aspiration pneumonia**.

Latest Update — 1 Point

7. **Deprescribing is now formalised.** The **AGS Beers Criteria 2023 update** (*JAGS* 2023) lists **antipsychotics and metoclopramide as potentially inappropriate medications in older adults**, with antipsychotics carrying a boxed warning for **increased mortality in dementia**, and specifically advises avoiding metoclopramide unless for gastroparesis and limiting it to under 12 weeks. Where signs persist after withdrawal, current practice adds **DaTSCAN** and, in research settings, **α-synuclein seed amplification assay** to identify the subgroup in whom the drug has unmasked underlying Parkinson's disease.

Current Practice Update

Aspect	Textbook	Current recommendation
Prevention	"Avoid prescribing neuroleptics in elderly patients, especially those with dementia"	Formalised in the **AGS Beers Criteria 2023**; antipsychotics and metoclopramide are explicitly potentially inappropriate in older adults
Persisting signs after withdrawal	20% persist, "suggests underlying neurodegeneration"	Confirm with **DaTSCAN**; α-synuclein SAA can identify unmasked PD in research settings

Examiner's Tip — lines that earn marks

- Two contrasts win marks: "**dyskinesia is frequent and tremor is absent**" and "**ioflupane SPECT is normal, unlike in PD**."
- Quote the two figures: recovery takes **weeks to months**, and **about 20% have persisting signs** indicating unmasked neurodegenerative disease.
- State the prohibition explicitly: "**anticholinergics should be avoided in the elderly because they often induce delirium**" — this is a common trap, since anticholinergics are standard in younger psychiatric patients.
- Close with the geriatric public-health line: "**Prevention is a crucial issue in geriatric care — avoid neuroleptics in older people with dementia, in whom they are ineffective.**"

Q57. Multiple system atrophy — clinical features, types, diagnosis, prognosis and treatment RECURS

15-Mark Answer | 10 Textbook Points + 2 Latest Updates = 12 Points

*Source: Pathy's 6th ed., Chapter 32 (Hypotension) and Chapter 53 (Table 53.2). Note: Pathy's covers MSA only briefly; the detailed MDS classification, MRI signs and prognostic figures are **not covered adequately in the uploaded textbook** and are supplied from current evidence, clearly separated below.*

Textbook Core — 10 Points

1. **Definition.** Multiple system atrophy is a **sporadic, progressive, adult-onset disorder characterised by autonomic dysfunction, parkinsonism and ataxia in any combination**.
2. **Types (textbook nomenclature).** Subtypes include **striatonigral degeneration** (parkinsonian predominant), **sporadic olivopontocerebellar atrophy** (cerebellar predominant) and **Shy–Drager syndrome** (autonomic predominant).
3. **Autonomic features and their timing — the key diagnostic clue. Orthostatic hypotension, impotence, urinary incontinence or retention is typically seen before the onset of motor symptoms, or within two years after the onset of motor symptoms.** Table 53.2 emphasises **severe dysautonomia early in the course of parkinsonism** — orthostatic hypotension, urinary disorder, sexual dysfunction and gastrointestinal symptoms.
4. **Motor features. Parkinsonism with a poor or unsustained motor response to levodopa therapy** — this poor levodopa response is the single most useful clinical discriminator from idiopathic PD.
5. **Additional neurological signs.** There may be **cerebellar or corticospinal (pyramidal) signs on physical examination**. In Table 53.2 the syndrome is defined by severe dysautonomia **plus a pyramidal or cerebellar syndrome**.
6. **Orthostatic hypotension — definition and measurement.** A fall in **systolic BP of 20 mmHg or diastolic BP of 10 mmHg within three minutes of standing**. **Delayed orthostatic hypotension** occurs after six minutes, and some patients require **standing for 10 minutes** before the fall appears. **Asymptomatic orthostatic hypotension is more common than the symptomatic form**, and even when asymptomatic it predicts falls, cerebrovascular and cardiovascular disease and all-cause mortality.
7. **Context among neurogenic causes of orthostatic hypotension.** The commonest neurogenic causes are **pure autonomic failure (PAF), Parkinson's disease with autonomic failure, and MSA**. PAF is idiopathic and sporadic with widespread autonomic failure and **reduced supine plasma noradrenaline**; **some PAF patients later manifest MSA**. In all three there is **decreased sympathetic and parasympathetic innervation of cardiac and extra-cardiac autonomic receptors**. Orthostatic hypotension affects **30–40% of PD patients**, against a pooled prevalence of **22% in community-dwelling older adults**.
8. **Epidemiology and place among the Parkinson-plus syndromes.** MSA is listed among the causes **very rarely observed as very late-onset parkinsonism** — important, because in a very old patient with parkinsonism, drug-induced, vascular, PSP and Lewy body causes are far more likely.
9. **Diagnosis.** Diagnosis is **clinical**, resting on the triad of **early severe dysautonomia + levodopa-unresponsive parkinsonism + cerebellar or pyramidal signs**. Response to levodopa can be tested formally by the **levodopa challenge** (150–250 mg fasting, assessed every 30 minutes for two to three hours). Ioflupane SPECT is abnormal but **does not separate MSA from PD**. The differential includes **PSP, corticobasal degeneration, dementia with Lewy bodies, vascular parkinsonism and normal pressure hydrocephalus**.
10. **Treatment — symptomatic only.** Give an **adequate trial of levodopa** although the response is poor or unsustained. Treat **orthostatic hypotension** with **droxidopa, midodrine, fludrocortisone and elastic venous compression**, together with physical manoeuvres that reduce postural hypotension, adequate salt and fluid, head-up tilt at night, and **withdrawal of drugs that worsen orthostasis** — alpha blockers, nitrates, diuretics, calcium channel blockers, tricyclics and antiparkinsonian drugs. Manage bladder dysfunction (solifenacin has low brain penetration; catheterisation for retention), constipation, drooling with botulinum toxin, and dysphagia and dysarthria with **speech and swallow therapy**. Provide falls prevention, rehabilitation and caregiver support. **Deep brain stimulation is not indicated.**

Latest Updates — 2 Points

11. **MDS 2022 diagnostic criteria and imaging biomarkers.** The **Movement Disorder Society criteria for MSA** (Wenning et al., *Mov Disord* 2022) replaced the 2008 consensus and define **four levels of certainty — neuropathologically established, clinically established, clinically probable, and the research category of**

possible prodromal MSA. Nomenclature is now **MSA-P (parkinsonian) and MSA-C (cerebellar)**. MRI markers are formally incorporated: the **"hot cross bun" sign** in the pons (seen in around **75% of MSA-C**) and the **hyperintense putaminal rim** with putaminal atrophy in MSA-P. Brain-bank validation confirmed better early sensitivity, and a 2026 registry re-evaluation found the 2022 criteria gave **94% specificity for clinically established MSA** versus 71% for clinically probable.

12. **Prognosis and biomarkers.** Prevalence is about **3.4–4.9 per 100,000**, with mean onset near **55–60 years**; **median survival from symptom onset is 6–10 years** (natural-history cohorts report 8.9–9.8 years), and **severe symptomatic autonomic failure at diagnosis and early dysphagia predict shorter survival**. **α-Synuclein seed amplification assay** in CSF and skin, and **fibrillar phosphorylated α-synuclein in Schwann cells** as an MSA-specific hallmark, are emerging diagnostic tools, and **neurofilament light chain (NfL)** is elevated in MSA and predicts mortality (*Lancet Neurology* 2024; *npj Parkinson's Disease* 2026). There is still **no disease-modifying therapy**; agents such as TEV-56286 are in Phase 2.

Current Practice Update

Aspect	Textbook	Current recommendation
Classification	Striatonigral degeneration, sporadic olivopontocerebellar atrophy, Shy–Drager syndrome	**MSA-P and MSA-C**; the eponym Shy–Drager is obsolete because all MSA has dysautonomia
Diagnostic criteria	Clinical description only	**MDS 2022 criteria** with four certainty levels, including **possible prodromal MSA**
Imaging	Not specified for MSA	**Hot cross bun sign** (pons, MSA-C) and **putaminal rim sign** with putaminal atrophy (MSA-P) are formal supportive features
Biomarkers	Reduced supine noradrenaline in PAF	Add **α-synuclein SAA (CSF/skin)**, Schwann-cell phospho-α-synuclein and **NfL**
Prognosis	Not stated	**Median survival 6–10 years**; worse with early severe autonomic failure and dysphagia

Exam approach: write the textbook triad and subtypes first, then add "current MDS 2022 criteria classify MSA as MSA-P and MSA-C with four levels of diagnostic certainty" — one sentence secures the update mark.

Extra Points to Remember — this topic recurs

- **The defining timing rule to quote:** "Orthostatic hypotension, impotence and urinary symptoms appear **before, or within two years of**, the onset of motor symptoms." This is what separates MSA from PD, where autonomic failure appears late.
- **Red flags for MSA in a parkinsonian patient (mnemonic "RAPID"):** **R**apid progression; **A**utonomic failure early and severe; **P**yramidal or cerebellar signs; **I**nspiratory stridor and disproportionate antecollis; **D**ysphagia and dysarthria early, with poor levodopa response.
- **Two clinical signs examiners like: nocturnal inspiratory stridor** (from vocal cord abductor paralysis — a cause of sudden death, may need CPAP or tracheostomy) and **disproportionate antecollis**.
- **Supine hypertension is the therapeutic trap:** most MSA patients with orthostatic hypotension also have supine hypertension. Never give midodrine or fludrocortisone within a few hours of lying down; raise the head of the bed by 30–40° and avoid daytime recumbency.
- **Non-pharmacological measures first for orthostatic hypotension:** physical counter-manoeuvres (leg crossing, squatting, tensing), abdominal binder and compression stockings, small frequent meals to limit postprandial hypotension, adequate salt and 2–2.5 L fluid, and slow rising.
- **Neuropathology one-liner:** MSA is an **α-synucleinopathy** characterised by **glial cytoplasmic inclusions (Papp-Lantos bodies) in oligodendrocytes**, in contrast to the neuronal Lewy bodies of PD.

Examiner's Tip — lines that earn marks

- Open with the textbook definition verbatim: "**a sporadic, progressive, adult-onset disorder characterised by autonomic dysfunction, parkinsonism and ataxia in any combination.**"
- Give the orthostatic hypotension definition with numbers — **20 mmHg systolic or 10 mmHg diastolic within three minutes** — and mention **delayed orthostatic hypotension after six minutes**, which most candidates omit.
- Answer the four parts of the question in order, with clear headings: **Clinical features → Types → Diagnosis → Prognosis → Treatment**. The question names them, so the examiner is marking against them.
- Name the two MRI signs: **hot cross bun sign** and **putaminal rim sign**.
- Prognosis must have a number: **median survival 6–10 years**, worse with early severe autonomic failure.

Q58. Essential tremor RECURS

7-Mark Answer | 6 Textbook Points + 1 Latest Update = 7 Points

Source: Pathy's 6th ed., Chapter 54 (Tables 54.3, 54.4) and Chapter 53.

Textbook Core — 6 Points

1. **Definition and epidemiology.** Essential tremor is the **most common tremor disorder and the most common movement disorder**, with prevalence **up to 3% in the general population and up to 5% in adults over 60**, rising to as high as **21.7% in adults over 95**.
2. **Pathophysiology.** Tremor arises from dysfunction in networks involving the **inferior olive, cerebellum, thalamus, basal ganglia and cortex**. The **basal ganglia network triggers the onset** of tremor while the **cerebellar network — predominantly affected in essential tremor — modulates its amplitude**.
3. **Clinical presentation.** A **bilateral action-induced (postural and kinetic) tremor** of the upper limbs, which may involve **head and voice**. It may be asymmetric but is **usually not unilateral**. An **intention tremor and a kinetic tremor of greater amplitude than the postural tremor** make ET more likely; **rest tremor is not typical** and suggests parkinsonism. A **family history of tremor** is common; **ethanol may improve** and **caffeine and smoking may worsen** the tremor.
4. **Differential diagnosis. Parkinsonian tremor** (rest tremor with rigidity, bradykinesia, postural instability, hypomimia); **dystonic tremor** (isolated head tremor, unilateral tremor, abnormal posturing or thumb flexion, torticollis, geste antagoniste, arrhythmic and directional head tremor); **cerebellar tremor** (intention tremor with dysmetria, scanning speech, truncal ataxia); **functional tremor** (variability, distractibility, entrainment, suggestibility); **drug-induced tremor** — caffeine, ethanol, nicotine, beta-agonists, tricyclics, lithium, SSRIs, neuroleptics, valproate, amiodarone, ciclosporin, tacrolimus, metoclopramide; and **hyperthyroidism, orthostatic tremor and late-onset Wilson's disease**. The **spiral test** is a useful bedside discriminator — ET spirals have **one clear axis**, dystonic tremor spirals have **multiple or no clear axes**.
5. **Investigation.** In most elderly patients **no further workup is required**, as clinical assessment is sufficient. **Liver function tests and thyroid studies** can at times be helpful; **EMG is very beneficial for orthostatic tremor**; **DaTSCAN is normal in essential tremor** and abnormal in parkinsonism; MRI with and without contrast is indicated if cerebellar tremor is suspected. **Misdiagnosis is frequent** and leads to inadequate therapy.
6. **Treatment.** There is **no curative therapy, and the indication for symptomatic therapy depends solely on the functional and social impairment the patient experiences**. **First line: propranolol and primidone**, which give a **30–70% reduction in tremor amplitude** but frequently have to be discontinued for side effects — **bradycardia with propranolol; nausea, dizziness and drowsiness with primidone**. **Second line: topiramate, gabapentin and benzodiazepines. Surgery** — deep brain stimulation of the **ventral intermediate (Vim) nucleus of the thalamus**, gamma knife surgery or **focused ultrasound thalamotomy** — gives greater amplitude reduction but is reserved for **severe medication-refractory tremor**.

Latest Update — 1 Point

7. **Staged bilateral MR-guided focused ultrasound thalamotomy is now approved.** The FDA approved **staged bilateral MRgFUS thalamotomy** for essential tremor, with the **second side treated at least nine months after the first**; the pivotal open-label trial was published by Kaplitt and colleagues in ***JAMA Neurology* 2024**. A 2025 *Movement Disorders* analysis found efficacy **comparable to bilateral DBS**, with two target "sweet spots" (dorsal Vim–Vop border and ventral Vim/posterior subthalamic area), while prospective series confirm **gait and balance impairment as the principal adverse effects** — a significant consideration for falls risk in older patients.

Extra Points to Remember — this topic recurs

- **Modern classification:** the MDS consensus uses **two axes** (clinical characteristics and aetiology) and defines "**essential tremor plus**" — isolated tremor with additional soft signs such as mild dystonic posturing, impaired tandem gait or questionable rest tremor. Pathy's uses the phrase "ET or ET plus" without defining it.
- **Practical prescribing in the elderly:** propranolol is contraindicated in asthma, COPD, bradycardia and decompensated heart failure and may mask hypoglycaemia in diabetes; **primidone is preferred in these patients**, started at 12.5–25 mg at night to avoid the notorious acute first-dose reaction of nausea, vertigo and ataxia.
- **Why the distinction from PD matters:** misdiagnosing ET as PD leads to years of unnecessary levodopa; misdiagnosing PD as ET delays effective treatment. **DaTSCAN settles it** — normal in ET.
- **Indian context:** propranolol and primidone are inexpensive and universally available; MRgFUS exists in only a handful of Indian tertiary centres and is largely out-of-pocket, so **DBS and stereotactic thalamotomy remain the practical surgical options**. Check **serum ceruloplasmin, 24-hour urinary copper and slit-lamp examination** in any atypical or younger tremor, as Wilson's disease is relatively more common in India.
- **The sentence that defines the geriatric approach:** "Treat the disability, not the tremor amplitude."

Examiner's Tip — lines that earn marks

- Open with the epidemiology triple: **3% general population, 5% over 60, 21.7% over 95.**
- The one-line contrast: "**rest tremor in Parkinson's disease, action tremor in essential tremor; DaTSCAN is abnormal in the former and normal in the latter.**"
- Name the **spiral test** — a bedside test always scores.
- Quote the treatment principle verbatim and give the **30–70% amplitude reduction** figure for propranolol and primidone.

Q59. Movement disorders in the elderly

7-Mark Answer | 6 Textbook Points + 1 Latest Update = 7 Points

Source: Pathy's 6th ed., Chapter 54 (Tables 54.1–54.4) and Chapters 44 and 53.

Textbook Core — 6 Points

1. **Classification. The most common movement disorders in the elderly are the parkinsonian syndromes.** The non-parkinsonian movement disorders affecting older people are **myoclonus, tremor, dystonia, tardive dyskinesia, chorea, functional movement disorders, and restless legs syndrome with periodic limb movements** during sleep and while awake.
2. **Phenomenology (Table 54.1). Myoclonus** — sudden brief muscle jerks (positive) or sudden cessation of muscle activity (negative), "lightning brief", irregular and non-stereotyped. **Tremor** — rhythmic oscillation around a body part. **Dystonia** — sustained or intermittent contractions causing abnormal movements or postures. **Chorea** — irregular movements flitting from one body part to another with a dance-like appearance. **Tardive dyskinesia** — stereotyped masticatory movements with lip and tongue protrusion. **Restless legs syndrome** — an urge to move relieved by walking or stretching.
3. **Commonest conditions and age relationship. Essential tremor** is the most common, affecting up to **5% of adults over 60** and up to **21.7% over 95**. Parkinsonism prevalence also rises with age, and **mild extrapyramidal signs, ageing-related tremor and paratonia (gegenhalten)** may be found in healthy older people as part of neurological ageing.
4. **Drug-induced movement disorders are a major geriatric issue** because of polypharmacy (Table 54.3): **myoclonus** — gabapentin, pregabalin, phenytoin, valproate, penicillins, cephalosporins, quinolones, SSRIs, lithium, opioids, amiodarone; **tremor** — SSRIs, tricyclics, lithium, beta-agonists, valproate, amiodarone, ciclosporin, tacrolimus, metoclopramide; **dystonia and tardive dyskinesia** — haloperidol, olanzapine, metoclopramide, prochlorperazine, phenytoin, carbamazepine; **chorea** — levodopa, dopamine agonists, neuroleptics, phenytoin, lamotrigine, lithium, oestrogens; **restless legs** — olanzapine, quetiapine, mirtazapine, topiramate. **The medication list must be scrutinised in every case and offending drugs stopped where possible.**
5. **Diagnosis.** Most are **clinical diagnoses** based on history and examination. Investigations are selective: metabolic and liver profile and thyroid studies; **brain and spine imaging**, particularly where symptoms are focal or cerebellar signs are present; **CSF studies** in inflammatory conditions; **EEG and EMG** to classify myoclonus; **EMG for orthostatic tremor**; and **DaTSCAN** to separate parkinsonism from essential tremor.
6. **Treatment (Table 54.4). Reverse the cause first** — stop the offending drug, correct metabolic derangement. **Myoclonus** — valproate, piracetam or levetiracetam for cortical myoclonus; botulinum toxin or GABAergic drugs for segmental and peripheral myoclonus. **Tremor** — propranolol and primidone, then topiramate, gabapentin or benzodiazepines, with DBS, gamma knife or focused ultrasound for refractory cases. **Dystonia** — physiotherapy, botulinum toxin injections, anticholinergic and GABAergic drugs, tetrabenazine for tardive dystonia, DBS for refractory cases. **Tardive dyskinesia** — slow down-titration of the offending agent, switch to clozapine or quetiapine, and **valbenazine or deutetrabenazine**. **Chorea** — tetrabenazine, deutetrabenazine or valbenazine, or neuroleptics. **Restless legs syndrome** — iron replacement if indicated, then **α2δ ligands (gabapentin, pregabalin) or dopaminergic drugs** first line and opioids second line. **Functional movement disorders** — convey the diagnosis as a genuine neurological condition with potential for recovery, with physiotherapy for motor retraining, CBT and treatment of comorbid anxiety and depression.

Latest Update — 1 Point

7. **Restless legs syndrome — a complete reversal of first-line therapy.** The **AASM clinical practice guideline (Winkelman et al., *Journal of Clinical Sleep Medicine*, January 2025)** now gives a **strong recommendation for α2δ ligands — gabapentin, gabapentin enacarbil and pregabalin — and for intravenous ferric carboxymaltose**, while **dopamine agonists have been moved to "conditional against" and are no longer recommended as initial therapy** because of **augmentation**, which affects roughly **7–10% of patients per year**. Cabergoline carries a strong recommendation against use at any dose. Iron should be assessed in every patient, with **serum ferritin targets above 75–100 µg/L and transferrin saturation above 20%**, regardless of haemoglobin. This is a complete inversion of the 2012 guideline and is directly relevant to older patients, in whom augmentation and impulse-control disorders are especially damaging.

Current Practice Update

Aspect	Textbook	Current recommendation
First-line therapy for restless legs syndrome	"α2δ ligands and dopaminergic medications" — both offered; dopamine agonists officially approved and widely used	**Gabapentinoids and IV ferric carboxymaltose are first line; dopamine agonists are recommended against** (AASM 2025)
Rationale	Augmentation noted — 76% of dopaminergic-treated patients need dose increase or develop augmentation	Same rationale, now translated into a formal **strong recommendation against** dopamine agonists

Exam approach: state the textbook option pair, then add "the 2025 AASM guideline now recommends gabapentinoids and IV iron first line and advises against dopamine agonists because of augmentation."

Examiner's Tip — lines that earn marks

- Open with the classification sentence: "**The most common movement disorders in the elderly are the parkinsonian syndromes**," then list the non-parkinsonian six.
- Define each phenomenologically in **one line each** — the examiner is testing whether you can describe an abnormal movement precisely.
- Devote a full point to **drug-induced movement disorders**; in a geriatrics paper this is the highest-yield element of the answer.
- Use the mnemonic **URGE** for restless legs syndrome: **U**rge to move the legs, worse at **R**est, **G**ets better with movement, occurs in the **E**vening or night.

Q60. Describe the ageing changes in the central nervous system including the brain, and the degenerative diseases of the brain

15-Mark Answer | 10 Textbook Points + 2 Latest Updates = 12 Points

Source: Pathy's 6th ed., Chapter 2 (Physiology of ageing), Chapter 6 (Ageing of the brain, Table 6.2) and Chapter 44 (Neurological signs of ageing, Table 44.1); Essentials of Clinical Geriatrics 7th ed., Chapter 6 (Table 6-7).

Textbook Core — 10 Points

1. **Cellular changes.** With age **some neurons are lost and others shrink**, and neurons attempt to compensate by **dendritic proliferation**. Support structures such as **neuroglia increase**. Unlike other systems, **new neurons cannot grow to replace lost neurons**, but the sheer number of CNS neurons means losses are not recognised until a threshold is crossed. **Lipofuscin accumulates** in nerve cells.
2. **Structural and volume changes.** Imaging shows **selective, not generalised, atrophy**. Global brain atrophy increases from **0.2% per year at ages 30–50 to 0.3–0.5% per year at ages 70–80**. **Grey matter loss is greater in frontal and parietal than in temporal and occipital cortices**, while **temporal lobe and hippocampal volumes decline continuously with accelerating loss after age 70**.
3. **Regional neuronal loss.** There is **relative preservation of cortical and hippocampal neuronal populations**, but **subcortical structures — the basal forebrain, locus ceruleus and substantia nigra — do show neuronal loss**. Plasticity persists through **dendritic sprouting**, though this compensatory ability declines with age.
4. **Mechanisms of brain ageing. Mitochondrial dysfunction** with oxidative damage to mitochondrial DNA; **oxidative damage** from increased reactive oxygen species and reduced antioxidant defence; **loss of proteostasis** with impaired autophagic and proteasomal degradation; **dysregulation of calcium homeostasis** causing calcium-mediated excitotoxicity; **impaired adaptive stress responses** (reduced BDNF and NGF signalling); **neuroinflammation** from microglia and astrocytes; **impaired DNA repair**; **impaired neurogenesis** in the subventricular zone and hippocampal dentate gyrus; and **dysregulated energy metabolism** with neuronal insulin resistance.
5. **Neurotransmitter and network changes. Impaired inhibitory GABA signalling** causes excitatory imbalance, hyperexcitability and excitotoxicity, and **dysregulation of acetylcholine, dopamine and serotonin** is linked with neurodegeneration and impaired brain function. There is a **progressive reduction of synaptic plasticity with ageing**, similar to that seen in Alzheimer-like and Parkinson-like conditions, underlying cognitive and motor impairment.
6. **Neuropathology of the normal ageing brain.** Both **neurofibrillary pathology (tangles and neuropil threads)** and **senile neuritic plaques** — the hallmarks of the Alzheimer brain — are found in **cognitively normal older individuals**. Neurofibrillary pathology follows a **stereotyped hierarchical pattern, appearing first in the transentorhinal cortex** (Braak staging), and spread to hippocampal and association cortex parallels the appearance of cognitive decline. **Senile plaques have a broader, more variable distribution, and a significant burden may coexist with normal cognition.** Similarly, **α-synuclein-immunopositive Lewy bodies and Lewy neurites** are found in ageing but asymptomatic brains.
7. **Vascular changes.** Vascular change becomes more frequent in the ageing brain, manifested as **leukoaraiosis — small-vessel ischaemic change in white matter — and silent infarcts**, possibly related to **elevated blood pressure and cholesterol in midlife**. Age-related vascular changes also reduce cerebral perfusion and impair endothelial function.
8. **Cognitive ageing.** Typical cognitive ageing involves losses in **processing speed, cognitive flexibility, and the efficiency of working memory and sustained attention**, so it takes longer or more trials to learn new information. **Vocabulary, access to remotely learned information including semantic networks, and retention of well-encoded new information are spared** — therefore **impairment in these latter domains is a sensitive indicator of pathology rather than physiology**. Memory decline in healthy ageing is largely secondary to reduced processing speed.
9. **Clinical signs of the ageing nervous system (Tables 6.2 and 44.1). Sensory** — impaired distal vibration sense (about one-third of healthy people over 60), reduced pain perception and joint position sense. **Reflexes — loss of ankle jerks in almost one-third of healthy people by age 80** while knee, biceps and triceps jerks are maintained, and emergence of **primitive reflexes** (palmomental, snout, sustained glabellar, grasp). **Cranial nerves** — diminished olfaction after 60, presbyopia, senile miosis, **reduced upward conjugate gaze and convergence**, increased saccadic latency, presbycusis. **Motor** — loss of muscle bulk and strength, **mild increase in tone with paratonia (gegenhalten)**, mild parkinsonism, ageing-related tremor. **Gait, posture and balance** — reduced walking speed and stride length, reduced tandem ability, stooped posture, reduced ability to stand on one leg with increased postural sway.

10. **Degenerative diseases of the brain (Essentials Table 6-7). Degenerative CNS diseases** — Alzheimer's disease, dementia associated with Lewy bodies, Parkinson's disease, Pick disease (frontotemporal degeneration), Huntington's disease and progressive supranuclear palsy. **Vascular dementias** — multi-infarct dementia, Binswanger disease, arteritis, and anoxic damage. **Traumatic** — craniocerebral injury including dementia pugilistica. **Infective** — HIV-associated dementia, opportunistic infections, Creutzfeldt–Jakob disease, progressive multifocal leukoencephalopathy, post-encephalitic dementia. Alzheimer's disease affects **6–8% of those over 65** with prevalence **doubling every five years**, reaching nearly **30% over 85**. **The crucial clinical principle is that neurodegenerative conditions share pathophysiological processes, neuropathological modifications and phenotypic manifestations with the physiological ageing process**, so the key skill is to juxtapose the signs of ageing against those of incipient neurological disease.

Ageing versus disease — the discriminating features

Domain	Normal ageing	Suggests disease
Memory	Slower learning; retrieval helped by cueing; preserved semantic and well-encoded memory	Impaired **recognition** and loss of well-encoded information; no benefit from cueing
Gait	Reduced speed and stride length; stooped posture	Freezing, festination, early falls, wide-based ataxia, step-wise decline
Tone	Mild increase; paratonia	True lead-pipe rigidity with bradykinesia
Reflexes	Absent ankle jerks; primitive reflexes	Loss of knee, biceps or triceps jerks; extensor plantars
Eye movements	Reduced **upgaze** and convergence	Reduced **downgaze** — suggests progressive supranuclear palsy
Function	Instrumental and basic ADLs preserved	Loss of independence in ADLs

Latest Updates — 2 Points

11. **Neurodegenerative disease is now defined biologically, not clinically.** The **2024 revised Alzheimer's Association criteria** (Jack et al., *Alzheimer's & Dementia* 2024) define Alzheimer's disease by **core biomarkers of amyloid and tau — including blood-based p-tau217** — rather than by syndrome, and **anti-amyloid monoclonal antibodies (lecanemab, donanemab)** are now approved for early symptomatic disease. In parallel, the **NSD-ISS and SynNeurGe frameworks** (*Lancet Neurology* 2024) redefine Parkinson's disease and dementia with Lewy bodies as **neuronal α-synuclein disease** using **α-synuclein seed amplification assay**. The practical consequence is that the boundary between "brain ageing" and "early degenerative disease" is increasingly drawn by **biomarkers rather than symptoms**.
12. **Much of late-life cognitive decline is preventable.** The **2024 Lancet Commission on dementia prevention, intervention and care** identified **14 modifiable risk factors accounting for about 45% of dementia cases worldwide**, adding **untreated vision loss and high LDL cholesterol** to the previous twelve (low education, hearing loss, hypertension, smoking, obesity, depression, physical inactivity, diabetes, excess alcohol, traumatic brain injury, air pollution, social isolation). This reframes the ageing brain as a **modifiable** rather than purely degenerative target, and is highly relevant to India, where hypertension, diabetes, low education, hearing loss, cataract-related visual impairment and air pollution are all highly prevalent.

Current Practice Update

Aspect	Textbook	Current recommendation
Diagnosis of degenerative disease	Clinical syndromes with pathology confirmed at autopsy	**Biomarker-based biological definitions** — plasma p-tau217 for Alzheimer's, α-synuclein SAA for synucleinopathies
Treatment of Alzheimer's disease	Cholinesterase inhibitors and memantine (symptomatic)	**Anti-amyloid antibodies (lecanemab, donanemab)** for early symptomatic disease with confirmed amyloid, with ARIA monitoring; not yet routine in India
Prevention	Risk factors mentioned in passing	**14 modifiable risk factors, ~45% of dementia potentially preventable** (Lancet Commission 2024)

Examiner's Tip — lines that earn marks

- The question has **two halves** — allocate roughly seven points to ageing changes and three to degenerative diseases, and head them separately.
- Give the atrophy numbers: "**0.2% per year at 30–50, rising to 0.3–0.5% per year at 70–80**."
- The sentence that shows genuine understanding: "**Plaques and tangles occur in cognitively normal older people; a significant plaque burden may be associated with normal cognition.**"
- Reproduce the ageing-versus-disease table — it directly answers the unstated question the examiner is really asking.
- Two reflex facts always score: **one-third of healthy people over 60 have impaired distal vibration sense, and almost one-third have lost ankle jerks by 80, while knee and upper limb jerks are maintained.**
- Close with the Chapter 44 principle: "**The key skill is to juxtapose the signs of ageing with those of incipient neurological disease.**"

Rapid Revision Grid — Q53 to Q60

Numbers to memorise

- **Parkinson's disease:** prevalence ~1% over 65; levodopa 300–600 mg/day, 3–4 doses, 30 min pre-meal; challenge dose 150–250 mg; rest tremor 4–6 Hz; depression ~50%; constipation ~50%; delayed gastric emptying ≥70%; orthostatic hypotension in 30–40% of PD patients.
- **Orthostatic hypotension:** ≥20 mmHg systolic or ≥10 mmHg diastolic within 3 minutes; delayed form after 6 minutes; pooled prevalence 22% in community-dwelling older adults, 23.9% in long-term care.
- **Drug-induced parkinsonism:** recovery over weeks to months; ~20% have persisting signs; DaTSCAN normal.
- **Multiple system atrophy:** prevalence 3.4–4.9/100,000; autonomic features before or within 2 years of motor onset; median survival 6–10 years; hot cross bun sign in ~75% of MSA-C.
- **Essential tremor:** 3% general population, 5% over 60, 21.7% over 95; propranolol and primidone reduce amplitude by 30–70%; MRgFUS second side at ≥9 months.
- **Restless legs syndrome:** augmentation 7–10% per year on dopamine agonists; ferritin target >75–100 µg/L, transferrin saturation >20%.
- **Ageing brain:** atrophy 0.2%/year at 30–50 → 0.3–0.5%/year at 70–80; impaired distal vibration sense in one-third over 60; ankle jerks lost in one-third by 80; Alzheimer's 6–8% over 65, ~30% over 85, prevalence doubling every 5 years.
- **Clozapine:** discontinue permanently if neutrophils <1500/mm^3.

Guidelines, trials and approvals to cite (2023–2026)

Source	One-line message
AGS Beers Criteria 2023	Antipsychotics and metoclopramide are potentially inappropriate in older adults — the formal basis for preventing drug-induced parkinsonism
NSD-ISS and SynNeurGe, Lancet Neurol 2024	Parkinson's disease and DLB redefined biologically as neuronal α-synuclein disease using CSF α-synuclein seed amplification assay
Revised Alzheimer's Association criteria, 2024	Alzheimer's disease defined by core biomarkers including blood p-tau217, not by clinical syndrome
Lancet Commission on dementia, 2024	14 modifiable risk factors account for about 45% of dementia; vision loss and high LDL cholesterol newly added
Crexont (Aug 2024), **Vyalev** (Oct 2024), **Onapgo** (Feb 2025)	Extended-release oral levodopa, 24-hour subcutaneous levodopa prodrug infusion, and subcutaneous apomorphine infusion for motor fluctuations
Kaplitt et al., JAMA Neurol 2024	Staged bilateral focused ultrasound thalamotomy is effective in essential tremor, second side at ≥9 months
ADAPT-PD → FDA approval Feb 2025	Adaptive closed-loop DBS driven by beta-band local field potentials for PD with motor fluctuations
Exenatide-PD3, Lancet 2025	Weekly exenatide did not slow Parkinson's disease progression — no disease-modifying therapy exists (Expression of Concern issued June 2026)
AASM RLS guideline, J Clin Sleep Med Jan 2025	Gabapentinoids and IV ferric carboxymaltose are first line; dopamine agonists are recommended against because of augmentation
MDS MSA criteria 2022, validated 2023–2026	Four certainty levels including possible prodromal MSA; 94% specificity for clinically established MSA
FDA labelling change, March 2026	Vitamin B6 deficiency and seizure warning added to all carbidopa/levodopa products

Drugs of choice

Situation	Drug of choice
Parkinson's disease in the elderly	**Levodopa** with a peripheral decarboxylase inhibitor
Early-morning akinesia	**Dispersible levodopa** (dispersible Madopar)
Peak-dose dyskinesia	**Amantadine** 200–400 mg/day (renal dose adjustment)
Wearing-off / end-of-dose fluctuation	**COMT inhibitor** (entacapone) or Stalevo
Levodopa-induced nausea	**Domperidone** — never metoclopramide
Psychosis in Parkinson's disease	**Clozapine** or **pimavanserin**
Depression in Parkinson's disease	**Venlafaxine**
Dementia or apathy in Parkinson's disease	**Rivastigmine**
Neurogenic orthostatic hypotension (PD, MSA)	**Droxidopa**; alternatives midodrine, fludrocortisone, compression
Essential tremor	**Propranolol** or **primidone**
Refractory essential tremor	**Vim DBS** or **focused ultrasound thalamotomy**
Restless legs syndrome	**Gabapentin / pregabalin** plus iron repletion (2025 AASM)
Tardive dyskinesia	**Valbenazine** or **deutetrabenazine**
Cortical myoclonus	**Levetiracetam**, valproate or piracetam
Focal dystonia	**Botulinum toxin** injection

MD GERIATRIC MEDICINE

Consolidated Examination Compendium

29 Questions · Eight Subject Parts

Infection in Old Age · Tuberculosis · Vaccine-Preventable Disease
Tropical Infection · Urinary and Gastrointestinal Infection
Rheumatology and Vasculitis · Metabolic Bone Disease
Pressure Injury and Prescribing Safety

Primary sources: Pathy's Principles and Practice of Geriatric Medicine, 6th edition
Kane's Essentials of Clinical Geriatrics
Current evidence integrated to 2023 – present · Indian context throughout
Answer format: 15-mark = 10 + 2 | 7-mark = 6 + 1 | 6-mark = 5 + 1

Table of Contents

PART I — INFECTION IN OLD AGE — PRINCIPLES, DIAGNOSIS AND STEWARDSHIP			
1	Fever of unknown origin — definition, evaluation and management of prolonged fever in the elderly 15 MARKS	15 marks	**p. 4**
2	Types of shock; aetiopathogenesis, diagnosis and treatment of septic shock 15 MARKSASKED ×2	15 marks · asked ×2	**p. 6**
3	Procalcitonin 7 MARKS	7 marks	**p. 9**
4	Infection control and antimicrobial stewardship in hospitals 7 MARKS	7 marks	**p. 10**
PART II — TUBERCULOSIS			
5	DOTS; treatment, dose and regimens of MDR and XDR tuberculosis; drug-resistant TB 7 MARKSASKED ×4	7 marks · asked ×4	**p. 11**
6	GeneXpert test 7 MARKS	7 marks	**p. 13**
PART III — VIRAL AND VACCINE-PREVENTABLE INFECTION			
7	Herpes zoster and post-herpetic neuralgia 15 MARKSRECURRING	15 marks · recurring	**p. 14**
8	COVID-19 in the older adult — presentation and management 7 MARKS	7 marks	**p. 16**
9	Prevention and management of influenza 7 MARKS	7 marks	**p. 17**
10	Vaccinations in elderly patients 7 MARKS	7 marks	**p. 18**
11	Ebola virus — clinical features, spread, treatment and prevention 7 MARKS	7 marks	**p. 20**
PART IV — TROPICAL AND VECTOR-BORNE INFECTION			
12	Leptospirosis and dengue in the older adult 7 MARKS	7 marks	**p. 21**
13	Chikungunya — clinical presentation, differential diagnosis, laboratory tests and management 7 MARKS	7 marks	**p. 23**
14	Scrub typhus 7 MARKS	7 marks	**p. 24**
15	Cerebral malaria 6 MARKS	6 marks	**p. 25**
PART V — URINARY AND GASTROINTESTINAL INFECTION			
16	Asymptomatic bacteriuria in the older adult 7 MARKSRECURRING	7 marks · recurring	**p. 26**
17	Catheter-associated urinary tract infection (CAUTI) 7 MARKS	7 marks	**p. 28**
18	Clinical presentations and diagnosis of acute pyelonephritis 7 MARKS	7 marks	**p. 29**
19	Management of recurrent urinary tract infection in an older female 7 MARKS	7 marks	**p. 30**
20	Diagnosis and management of Clostridioides difficile 7 MARKS	7 marks	**p. 32**
PART VI — RHEUMATOLOGY AND SYSTEMIC VASCULITIS			
21	Polymyalgia rheumatica 7 MARKSRECURRING	7 marks · recurring	**p. 34**
22	Systemic vasculitides in the elderly 15 MARKS	15 marks	**p. 36**
23	Late / elderly-onset rheumatoid arthritis — how it differs from young-onset disease, and the relevant investigations 15 MARKSRECURRING	15 marks · recurring	**p. 38**
24	Psoriatic arthropathy 7 MARKS	7 marks	**p. 42**
25	Sjögren's syndrome 7 MARKS	7 marks	**p. 43**
PART VII — METABOLIC BONE DISEASE			
26	Osteoporosis, DEXA and FRAX 15 MARKSRECURRING	15 marks · recurring	**p. 44**
27	Paget's disease of bone — epidemiology, aetiology, pathophysiology, clinical manifestations and management 15 MARKSRECURRING	15 marks · recurring	**p. 47**
PART VIII — PRESSURE INJURY AND PRESCRIBING SAFETY			
28	Pressure ulcers — staging and management 15 MARKSRECURRING	15 marks · recurring	**p. 51**
29	Beers criteria, STOPP/START and anticholinergic burden 15 MARKSRECURRING	15 marks · recurring	**p. 54**
APPENDIX — CONSOLIDATED NIGHT-BEFORE MASTER SHEETS			

HOW THIS COMPENDIUM IS ORGANISED

This volume consolidates three separate question sets into a single continuously numbered sequence and regroups them by subject rather than by the order in which they were originally set. **All 29 questions are distinct.** Several address the same organ system or disease from different angles — four separate urinary questions, four tropical febrile illnesses, two metabolic bone questions — and these have been placed adjacent to one another and cross-referenced rather than merged, because each asks something the others do not. Where content genuinely overlaps (giant cell arteritis, vaccination, stewardship, the metabolic bone laboratory table), the fuller treatment is kept in one place and signposted from the others; the map below shows where to look.

Topic overlap map — questions to revise together

Theme running across several questions	Questions to read together
Bacteriuria, catheters and urinary infection	**Q16** asymptomatic bacteriuria · **Q17** CAUTI · **Q18** pyelonephritis · **Q19** recurrent UTI. Q16 sets the rule ("no symptom, no culture, no cure") that the other three depend on.
Giant cell arteritis	**Q21** polymyalgia rheumatica (the association, 16–21%) · **Q22** systemic vasculitides (full GCA work-up and treatment) · **Q1** fever of unknown origin (GCA is the commonest identifiable cause in over-65s).
Vaccination	**Q10** is the consolidated schedule; disease-specific detail sits in **Q7** zoster (RZV), **Q8** COVID-19, **Q9** influenza (high-dose/adjuvanted).
Antimicrobial stewardship	**Q4** is the framework; applied in **Q3** procalcitonin-guided stopping, **Q16** not treating bacteriuria, **Q17** catheter bundles, **Q20** *C. difficile*, **Q18–19** resistance-driven empirical choice.
Acute undifferentiated febrile illness in India	**Q12** dengue and leptospirosis · **Q13** chikungunya · **Q14** scrub typhus · **Q15** cerebral malaria · **Q2** septic shock (all five can present as shock).
Inflammatory arthritis of later life	**Q21** PMR · **Q23** late-onset RA · **Q24** psoriatic arthropathy · **Q25** Sjögren's · **Q13** post-chikungunya arthritis. The three-way PMR / LORA / YORA table in Q23 is the discriminator.
Metabolic bone laboratory differential	**Q26** osteoporosis and **Q27** Paget's share the calcium / phosphate / alkaline phosphatase / PTH table reproduced in Q27.
Corticosteroid-sparing therapy	**Q21** sarilumab in PMR · **Q22** tocilizumab and upadacitinib in GCA, avacopan in AAV · **Q24** IL-17/IL-23 inhibitors · **Q23** and **Q29** the harms that make steroid-sparing necessary.
Drugs to avoid in older adults	**Q29** is the systematic answer (Beers, STOPP/START, anticholinergic burden); specific instances appear in **Q7** amitriptyline, **Q24** JAK inhibitors and NSAIDs, **Q19** nitrofurantoin, **Q4** fluoroquinolones.

CONVENTIONS USED THROUGHOUT

Every answer follows the same four-block architecture: **Textbook Core** (6 points for a 7-mark note, 10 for a 15-mark essay), **Latest Update** (1 point for 7 marks, 2 for 15 marks, all from 2023 onwards and cited inline), a **Current Practice Update** table wherever the textbook position and current evidence genuinely diverge, and **Exam One-Liners** for rapid recall. Questions flagged RECURRING or ASKED ×n carry additional scaffolding — a mnemonic anchor, numerical anchors, named examiner traps, a 15-second answer skeleton and an extra recall box. Where a 7-mark note is also examinable at 15 marks, a boxed **expansion note** lists exactly what further content to add. Topics not adequately covered by the two primary textbooks are explicitly flagged at the head of the answer, with the textbook fragments that do exist quoted and marked, and guideline-derived content kept separate. All clinical and laboratory values use **Indian conventional units**.

PART I

Infection in Old Age — Principles, Diagnosis and Stewardship

The cross-cutting material: how infection presents differently in older adults, how to work up prolonged fever, how to recognise and resuscitate septic shock, the role of biomarkers, and the stewardship framework that governs every antibiotic decision in the rest of this book.

Question 1. Fever of unknown origin — definition, evaluation and management of prolonged fever in the elderly 15 MARKS

ANSWER

Textbook Core – 10 Points

1. **Classical definition.** Petersdorf and Beeson (1961), modified by Durack and Street (1991): a temperature **>38.3 °C (101 °F) on several occasions**, of **more than 3 weeks' duration**, remaining **undiagnosed after appropriate investigation** — three days of inpatient work-up, three outpatient visits, or one week of intelligent and invasive ambulatory investigation.
2. **The four categories (Durack and Street). Classic** FUO; **nosocomial** FUO (onset in hospital, not incubating on admission); **neutropenic / immune-deficient** FUO (neutrophils <500/mm³); and **HIV-associated** FUO. Each has a different differential and a different work-up — say which you are addressing.
3. **Why the definition must be adapted in old age.** Kane's: the **febrile response may be blunted or absent** in older people with bacterial infection, baseline body temperature is lower, and nursing-home residents may fail to show a diurnal rise. Fever in a long-term care resident is therefore redefined as **a single oral temperature above 37.8 °C (100 °F), repeated oral temperatures above 37.2 °C (99 °F), or a rise of more than 1.1 °C (2 °F) above the patient's baseline**. Applying the 38.3 °C threshold to a frail older patient will miss disease. **A poor febrile response is itself a negative prognostic factor.**
4. **The causes are distributed differently in the elderly — the key examinable fact.** Approximate shares in over-65s: **infection 25–35%**, **connective tissue and inflammatory disease 25–30%** (far higher than in the young, and **giant cell arteritis with polymyalgia rheumatica is the single commonest identifiable cause**), **malignancy 15–20%**, **miscellaneous including drug fever 10%**, and **undiagnosed 10–20%** (lower than in the young). The governing principle is that **FUO in an older person is usually an atypical presentation of a common disease, not a rare disease**.
5. **Infective causes to hunt for. Tuberculosis**, especially **miliary and extrapulmonary** — Kane's rule: "suspect tuberculosis when a patient is inexplicably failing". **Infective endocarditis** — nearly **50% of cases occur in older adults**, on degenerative valves and prostheses, and blood cultures may be negative if antibiotics were given. Also **intra-abdominal, hepatobiliary and pelvic abscess**, **complicated urinary infection and prostatic abscess**, **vertebral osteomyelitis and discitis**, **infected prosthetic joints and devices**, dental and sinus sepsis, and — in India — **enteric fever, brucellosis, melioidosis, kala-azar, scrub typhus and malaria**.
6. **Non-infective causes. Inflammatory**: giant cell arteritis and polymyalgia rheumatica, adult-onset Still's disease, rheumatoid arthritis, ANCA-associated vasculitis, polyarteritis nodosa, sarcoidosis. **Malignant**: **lymphoma is commonest**, then renal cell carcinoma, hepatocellular carcinoma, myelodysplastic syndrome, leukaemia, atrial myxoma and metastatic disease. **Miscellaneous**: **drug fever** — very common with polypharmacy (antibiotics, allopurinol, anticonvulsants, antiarrhythmics, heparin); **venous thromboembolism**; subacute thyroiditis and apathetic thyrotoxicosis; gout and pseudogout; resolving haematoma; adrenal insufficiency; and factitious fever.
7. **Step 1 — history and repeated examination.** The highest-yield "investigation" is a **meticulous, repeated daily physical examination**. Take a full **drug history including over-the-counter and AYUSH preparations and every recently started agent**, plus travel, animal and rural exposure, dental work, prosthetic implants, prior TB, transfusions and immunosuppression. On examination, look specifically at: **temporal arteries**, a **new or changing murmur**, all lymph node groups, liver and spleen, skin, nails and splinter haemorrhages, joints, thyroid, prostate and pelvis, rectum, fundi (Roth spots) and every prosthesis or device site.
8. **Step 2 — first-line investigations.** CBC with differential and **peripheral blood film**, **ESR and CRP**, renal and liver function, electrolytes, LDH, creatine kinase, urinalysis and urine culture, **three sets of blood cultures taken off antibiotics**, chest radiograph, sputum for AFB and **GeneXpert**, HIV test, TSH, ANA and rheumatoid factor, serum and urine protein electrophoresis, and — in India — a **blood film and rapid test for malaria** plus dengue and scrub typhus serology in season.
9. **Step 3 — second-line and targeted investigations. CT of chest, abdomen and pelvis**; **echocardiography** (transthoracic, then transoesophageal if endocarditis is suspected); ultrasound of abdomen and of any tender area; **temporal artery ultrasound or biopsy in anyone over 50 with an ESR above 50**; **bone marrow aspiration and trephine biopsy with culture** (high yield for miliary TB, lymphoma and myelodysplasia); and **FDG-PET/CT**. Then **tissue is the answer** — lymph node, liver or bone biopsy sent for histology, culture and TB PCR; lumbar puncture if there are any CNS features; joint aspiration if any joint is involved.
10. **Management principles. Avoid empirical "blind" antibiotics.** They obscure cultures, cause *C. difficile* and resistance, and are themselves a common cause of drug fever. Empirical therapy is justified only for **haemodynamic instability or sepsis, neutropenia, or a strong suspicion of a dangerous treatable condition**. **The great**

exception is giant cell arteritis — start high-dose corticosteroids immediately on suspicion, without waiting for the biopsy, to prevent blindness. A trial of anti-tuberculous therapy may be justified in a high-burden setting when miliary TB is strongly suspected and the patient is deteriorating, but only after a determined attempt at tissue diagnosis. **Stop or substitute every non-essential drug** — drug fever settles within 48–72 hours. Throughout, provide geriatric care in parallel: hydration and nutrition, delirium and pressure-injury prevention, early mobilisation, medication review, and an explicit discussion of goals of care. **Knowing when to stop investigating a very frail patient, and to observe instead, is itself a legitimate management decision** — a specific diagnosis is eventually reached in 70–90% of older patients, and undiagnosed FUO that settles spontaneously has a good prognosis.

Latest Updates (2023 – present)

11. **FDG-PET/CT is now the recommended second-line imaging modality in FUO**, with a diagnostic yield of roughly **50–60%** and the highest utility when CRP or ESR are raised. Current EANM/SNMMI and European guidance place it **before invasive biopsy** in the algorithm, and it is particularly valuable in older adults because it detects **large-vessel vasculitis, occult lymphoma and vertebral osteomyelitis** — the three commonest occult causes in this age group — and it directs the biopsy when one is needed.
12. **Molecular diagnostics and a reframed terminology. Metagenomic next-generation sequencing of plasma cell-free DNA** and targeted NGS can now identify pathogens in **culture-negative endocarditis** and deep-seated infection, though cost and false-positive signals limit routine use. Separately, current practice increasingly uses the term **"inflammation of unknown origin" (IUO)** for the raised-CRP, no-fever presentation — an important reframing for geriatrics, because many older patients with occult giant cell arteritis, lymphoma or endocarditis **never mount a fever at all** and would be missed by a temperature-based definition.

Current Practice Update

Item	Textbook / classical position	Current recommendation	Exam approach
Fever threshold	>38.3 °C for >3 weeks	In frail/LTC older adults, use the lower thresholds (single oral >37.8 °C, repeated >37.2 °C, or a rise of 1.1 °C)	Give the classical definition, then the geriatric modification
Imaging sequence	CT then invasive biopsy	**FDG-PET/CT before biopsy**; use it to direct the biopsy	Name PET/CT and its ~50–60% yield
Empirical antibiotics	Common practice	**Avoid** unless septic, neutropenic or a specific strong suspicion	State the exception for GCA steroids
Terminology	Fever of unknown origin	Add **inflammation of unknown origin (IUO)** for afebrile raised-CRP presentations	A high-value closing line for a geriatrics paper

Exam One-Liners

- Classical definition — **>38.3 °C, >3 weeks, undiagnosed after appropriate work-up**.
- Four categories — **classic, nosocomial, neutropenic, HIV-associated**.
- Commonest identifiable cause in over-65s — **giant cell arteritis / PMR**.
- Commonest malignancy — **lymphoma**; commonest infection — **tuberculosis**.
- Highest-yield "test" — **repeated physical examination and the drug chart**.
- Best single imaging test — **FDG-PET/CT**.
- The one condition to treat empirically — **giant cell arteritis**.
- Undiagnosed FUO that settles spontaneously — **good prognosis**.

Question 2. Types of shock; aetiopathogenesis, diagnosis and treatment of septic shock

15 MARKS ASKED ×2

ANSWER

Textbook Core – 10 Points

1. **Definition of shock.** Acute circulatory failure causing **inadequate tissue oxygen delivery relative to demand**, leading to cellular dysoxia, anaerobic metabolism, lactate accumulation and progressive organ dysfunction. **Hypotension is a late sign** — compensated shock with a normal blood pressure but raised lactate is common, and in a chronically hypertensive older patient a "normal" pressure may already represent profound shock. Clinically: tachycardia, tachypnoea, cool or mottled peripheries, prolonged capillary refill, oliguria and altered mentation.
2. **The four types of shock.**

Type	Causes	Cardiac output	SVR	Filling pressures (CVP/PCWP)
Hypovolaemic	Haemorrhage, GI losses, burns, dehydration — the elderly are highly susceptible (reduced total body water, blunted thirst, diuretics)	Low	High	Low
Cardiogenic	Myocardial infarction, arrhythmia, acute valvular failure, myocarditis, cardiomyopathy, drug toxicity	Low	High	High
Obstructive	Massive pulmonary embolism, tension pneumothorax, cardiac tamponade, severe pulmonary hypertension	Low	High	High
Distributive	**Septic** (commonest), anaphylactic, neurogenic, adrenal crisis, acute liver failure	**High** (or normal)	**Low**	Low or normal

   Mixed patterns are the rule in older patients — septic shock with a hypovolaemic and a cardiogenic component is the commonest single combination.
3. **Definitions of sepsis and septic shock (Sepsis-3, 2016). Sepsis = life-threatening organ dysfunction caused by a dysregulated host response to infection**, operationalised as an acute rise in the **SOFA score of 2 or more points**. **Septic shock** = sepsis with **persisting hypotension requiring vasopressors to maintain a MAP ≥65 mmHg AND a serum lactate >2 mmol/L despite adequate volume resuscitation** — a subset with **hospital mortality exceeding 40%**. **qSOFA** (respiratory rate ≥22, altered mentation, systolic BP ≤100 mmHg) is a bedside *prompt*, not a diagnostic tool; NEWS2 performs better for screening.
4. **Aetiology in the older adult.** Commonest sources: **respiratory (pneumonia), urinary, intra-abdominal, skin and soft tissue including pressure injury, and device-related**. Kane's makes the key microbiological point that **Gram-negative bacilli increase in frequency in every infection category in older adults** — *E. coli*, *Klebsiella*, *Proteus*, *Enterobacter* and *Pseudomonas* — alongside *S. pneumoniae*, *S. aureus*, *Enterococcus*, *Listeria* and *Candida*. Predisposing factors: immunosenescence, malnutrition, diabetes, indwelling devices, institutionalisation, and recent hospitalisation or antibiotics (which raise the risk of multidrug-resistant organisms).
5. **Pathogenesis — six linked mechanisms.** Pathogen-associated molecular patterns bind pattern-recognition (Toll-like) receptors → NF-κB activation → a cytokine storm of **TNF-α, IL-1 and IL-6**, producing: **(i) vasodilatation** through inducible nitric oxide synthase and loss of vascular tone — the distributive component; **(ii) endothelial injury with glycocalyx shedding** → capillary leak, interstitial oedema and **relative hypovolaemia**; **(iii) immunothrombosis** — tissue factor expression, microvascular thrombi and, at the extreme, DIC; **(iv) septic cardiomyopathy** — direct cytokine-mediated myocardial depression; **(v) mitochondrial dysfunction and cytopathic hypoxia**, so cells cannot use the oxygen that is delivered; and **(vi) a later immunoparalysis phase** with lymphocyte apoptosis and monocyte deactivation, which explains secondary infection and the late deaths that are so characteristic of older survivors.
6. **Presentation in the elderly — atypical and easily missed.** Kane's: **fever may be blunted or absent**, and the **absence of leucocytosis does not exclude bacterial infection**; **hypothermia in an older patient should promptly be treated as sepsis unless proven otherwise**. **Delirium, falls, functional decline, incontinence, anorexia and unexplained tachypnoea** may be the only features. Kane's list of the signs that should raise suspicion of infection in a long-term care resident — decline in functional status, increasing confusion, incontinence, falling, deteriorating mobility, reduced food intake, or failure to cooperate with staff — is worth reproducing verbatim.
7. **Diagnosis and investigations. At least two sets of blood cultures before antibiotics** where this does not delay them, plus site-directed cultures (urine, sputum, wound, CSF, drain fluid). **Serum lactate, repeated** to guide resuscitation. CBC, renal and liver function, coagulation screen with fibrinogen and D-dimer, arterial blood gas, glucose, **procalcitonin** and CRP, ECG and troponin, chest radiograph, and focused ultrasound or CT to identify the source. **Echocardiography** to assess cardiac function and volume status. Calculate the **SOFA score** and document a **Clinical Frailty Scale** score.
8. **Treatment — the first hour.** Oxygen to target saturation; obtain cultures and lactate; **broad-spectrum intravenous antibiotics within 1 hour** for septic shock, chosen from the **local antibiogram** and covering Gram-

negatives; **fluid resuscitation with at least 30 mL/kg of balanced crystalloid over the first 3 hours**; and **vasopressors if hypotension persists**. **Critical geriatric modification:** in an older patient with heart failure, valvular disease or chronic kidney disease, give fluid in **250–500 mL aliquots with reassessment after each** rather than an unmonitored bolus — over-resuscitation causes pulmonary oedema and is a common iatrogenic death. **Source control within 6–12 hours** is non-negotiable: drain the abscess, remove the infected line or urinary catheter, debride the necrotic tissue, decompress the obstructed urinary tract.

9. **Vasopressors and adjunctive therapy. Noradrenaline is first-line**, and may be started peripherally to avoid delay while central access is obtained. **Add vasopressin** when the noradrenaline requirement is escalating (usually around 0.25–0.5 µg/kg/min) rather than escalating noradrenaline indefinitely; **add adrenaline** if the MAP remains inadequate. Use **dobutamine or adrenaline** for septic cardiomyopathy with persistent hypoperfusion despite adequate filling. Give **intravenous hydrocortisone 200 mg/day** for shock requiring ongoing vasopressors. Supportive care: lung-protective ventilation if ARDS develops, VTE prophylaxis, stress-ulcer prophylaxis, glucose target **140–180 mg/dL**, early enteral nutrition, renal replacement therapy for the usual indications, and **daily antibiotic review with de-escalation** as cultures return.
10. **Geriatric-specific management and outcome.** Age is an independent predictor of mortality, and survivors face a **"post-sepsis syndrome"** of long-term functional and cognitive decline, sarcopenia, readmission and late death — so recovery, not just survival, is the outcome that matters. Therefore: **renal dose-adjust every drug** and monitor aminoglycoside and vancomycin levels; avoid nephrotoxins; screen for and prevent **delirium**; mobilise early and start rehabilitation in the ICU; prevent pressure injury; attend to nutrition; and hold an **early, explicit discussion of goals of care and ceilings of treatment** with the patient and family — **frailty and comorbidity, not chronological age, should inform escalation decisions**.

Latest Updates (2023 – present)

11. The **Surviving Sepsis Campaign 2026 international guidelines** confirm crystalloids as first-line fluid, **balanced crystalloids in preference to 0.9% saline**, **at least 30 mL/kg in the first 3 hours with frequent ongoing reassessment** to avoid both under- and over-resuscitation, and **crystalloids alone rather than routine supplemental albumin**. Noradrenaline remains first-line with vasopressin added on escalation. Two changes are directly geriatric: an **initial MAP target of 60–65 mmHg rather than a higher target in adults aged 65 and over** (permissive hypotension, from the 65 trial), and the suggestion to use **capillary refill time as an adjunct to lactate** for guiding resuscitation — a bedside measure available in any Indian hospital.
12. **Antibiotic timing has been formally stratified rather than made universally one-hour.** Current guidance recommends antimicrobials **within 1 hour for definite septic shock or high-probability sepsis**, but permits **up to 3 hours where sepsis is only possible and there is no shock**, allowing rapid assessment for a non-infectious mimic. This matters most in older adults, in whom pulmonary embolism, myocardial infarction, gastrointestinal bleeding, adrenal crisis, drug reactions and hypothermia routinely mimic sepsis. Counterbalancing this, the stewardship agenda now emphasises **de-escalation and procalcitonin-guided discontinuation** to shorten courses.

MNEMONIC ANCHOR

Types of shock = **"HCOD"** — **H**ypovolaemic · **C**ardiogenic · **O**bstructive · **D**istributive.
The septic shock bundle = **"CLAF-V-S"** — **C**ultures · **L**actate · **A**ntibiotics within 1 hour · **F**luids (balanced crystalloid) · **V**asopressor (noradrenaline) · **S**ource control.

NUMERICAL ANCHORS

Sepsis-3: SOFA rise **≥2** · septic shock MAP **<65 mmHg on vasopressors** + lactate **>2 mmol/L** · mortality **>40%** · qSOFA: RR ≥22, altered mentation, SBP ≤100 · fluids **30 mL/kg in 3 hours** (aliquots of 250–500 mL in the elderly) · antibiotics **<1 hour** in shock, up to 3 hours without shock · source control **6–12 hours** · vasopressin added at noradrenaline **0.25–0.5 µg/kg/min** · hydrocortisone **200 mg/day** · glucose **140–180 mg/dL** · MAP target **60–65 mmHg if ≥65 years**.

NAMED EXAMINER TRAPS

(a) Hypothermia in an older patient is sepsis until proved otherwise — and carries a worse prognosis than fever. **(b)** A "normal" blood pressure in a chronic hypertensive may be shock — ask what the patient's usual pressure is. **(c) qSOFA is a prompt, not a diagnostic criterion**, and it performs poorly in the elderly because altered mentation is so common. **(d)** Do **not** give an unmonitored 30 mL/kg bolus to a frail patient with heart failure — aliquot and reassess. **(e) No antibiotic can substitute for source control** — the infected catheter or undrained abscess will defeat any regimen. **(f)** Absence of leucocytosis does not exclude bacterial infection. **(g)** Steroids are for **vasopressor-dependent shock**, not for sepsis without shock.

15-SECOND ANSWER SKELETON

Define shock and note that hypotension is late → the four types in a table with CO/SVR/filling pressures → Sepsis-3 definitions of sepsis and septic shock with the numbers → aetiology and the Gram-negative shift in the elderly → the six pathogenic mechanisms → atypical presentation (hypothermia, delirium, falls) → investigations including lactate, cultures, SOFA, frailty score → the first-hour bundle with the geriatric fluid modification → source control → noradrenaline → vasopressin → adrenaline → hydrocortisone → post-sepsis syndrome and goals of care → close with SSC 2026 (balanced crystalloid, MAP 60–65 in ≥65s, stratified antibiotic timing).

EXTRA RECALL BOX — THE INDIAN ANGLE AND THE MIMICS

Indian context. Empirical therapy must follow the **local hospital antibiogram**, not a textbook: carbapenem-resistant *Klebsiella* and *Acinetobacter* and ESBL *E. coli* are widespread, and *Candida auris* has emerged as a nosocomial bloodstream pathogen. **NAP-AMR 2.0 (2025–2029)** makes infection prevention and stewardship programmes mandatory in every facility. In an Indian older adult with fever and shock, always also consider **severe falciparum malaria, scrub typhus, leptospirosis, dengue and disseminated tuberculosis** — all of which can present exactly as septic shock and several of which have specific, life-saving treatment.

The mimics that must be excluded before committing to "sepsis": pulmonary embolism, myocardial infarction and cardiogenic shock, gastrointestinal haemorrhage, acute adrenal insufficiency, thyroid storm and myxoedema coma, anaphylaxis, drug reactions including neuroleptic malignant and serotonin syndromes, pancreatitis, mesenteric ischaemia, and heat stroke.

Exam One-Liners

- Four types — **hypovolaemic, cardiogenic, obstructive, distributive**.
- Distributive shock alone has a **high cardiac output with low SVR**.
- Sepsis-3 — **life-threatening organ dysfunction from a dysregulated host response**; SOFA rise ≥2.
- Septic shock — **vasopressor-dependent MAP <65 plus lactate >2**; mortality >40%.
- First-line vasopressor — **noradrenaline**; second — **vasopressin**.
- Fluid of choice — **balanced crystalloid**.
- MAP target in adults ≥65 years — **60–65 mmHg**.
- Steroid — **hydrocortisone 200 mg/day for vasopressor-dependent shock**.
- The intervention no drug replaces — **source control**.

Question 3. Procalcitonin 7 MARKS

ANSWER

Textbook Core – 6 Points

1. **What it is.** A **116-amino-acid precursor peptide of calcitonin**, encoded by the **CALC-1 gene**. In health it is produced only by thyroid C cells and is completely cleaved to calcitonin, so circulating levels are negligible (**<0.05 ng/mL**). During **bacterial infection**, bacterial endotoxin and the cytokines IL-1β, TNF-α and IL-6 induce **ubiquitous extrathyroidal CALC-1 expression** in liver, lung, kidney, intestine and adipose tissue, releasing large amounts of **intact, uncleaved procalcitonin** into the blood.
2. **Kinetics — why it is more useful than CRP.** It **rises within 2–4 hours**, **peaks at 12–24 hours**, has a half-life of about **22–35 hours**, and **falls rapidly once the infection is controlled**. CRP by contrast peaks only at 48 hours and falls slowly. This makes procalcitonin usable for **early decisions and for serial trend interpretation**.
3. **The discriminating principle.** Procalcitonin synthesis is **suppressed by interferon-γ**, which is released in viral infection — so **it stays low in viral illness and rises in bacterial illness**. This is the single concept the examiner wants. It is therefore more **specific for bacterial infection** than CRP, ESR or the white cell count, and its magnitude correlates with the extent of infection and with organ dysfunction, giving it prognostic value.
4. **Interpretation thresholds.** In suspected lower respiratory tract infection: **<0.1 ng/mL** — antibiotics strongly discouraged; **0.1–0.25** — discouraged; **0.25–0.5** — encouraged; **>0.5 ng/mL** — strongly encouraged. In the critically ill, **>2 ng/mL** suggests a high probability of severe sepsis or septic shock, and **>10 ng/mL** suggests overwhelming bacterial sepsis with organ failure. For monitoring, **failure of the level to fall by 80%, or to below 0.5 ng/mL, by day 4** predicts a poor outcome.
5. **Clinical uses. (i) Antimicrobial stewardship** — procalcitonin-guided algorithms reduce antibiotic initiation and duration in lower respiratory tract infection and in ICU sepsis without increasing mortality. **(ii) Differential diagnosis** — helping separate bacterial pneumonia or bacteraemia from a COPD exacerbation, decompensated heart failure, viral illness or a non-infective inflammatory state. **(iii) Prognostication and monitoring** of response to treatment. **(iv) Particular value in the older adult**, in whom fever, leucocytosis and symptoms are all unreliable, and in whom CRP and ESR are often non-specifically raised by comorbidity.
6. **Limitations — the marks are here. False positives**: major surgery and trauma, burns, cardiac arrest and resuscitation, prolonged cardiogenic shock, severe pancreatitis, malaria, heat stroke, **medullary thyroid carcinoma and small-cell lung cancer**, **end-stage renal disease and dialysis**, and treatment with anti-thymocyte globulin or OKT3. **False negatives**: very early presentation (<6 hours), and **localised or walled-off infection** — abscess, empyema, **infective endocarditis**, osteomyelitis and septic arthritis — as well as intracellular and atypical organisms. It must therefore **never be used alone** to start or stop antibiotics; it supplements, but does not replace, clinical judgement and culture.

Latest Update (2023 – present)

7. Recent randomised evidence — including the **ADAPT-Sepsis** and **PRONTO** trials and the **NICE 2023 diagnostics guidance** — has clarified exactly where procalcitonin adds value. It **safely shortens antibiotic duration** in ICU sepsis and in suspected lower respiratory tract infection, but it **does not reduce mortality** and does **not** reliably reduce antibiotic *initiation* in the emergency department. Current guidance therefore supports its use for **guiding discontinuation and de-escalation rather than for deciding whether to start**. In Indian public hospitals, where cost limits routine availability, this is the rational deployment — reserve it for **de-escalation decisions in the ICU** rather than as a screening test.

Exam One-Liners

- Nature — **precursor of calcitonin**, CALC-1 gene, normally <0.05 ng/mL.
- Rises in **2–4 h**, peaks at **12–24 h**, half-life **~24 h**.
- Key principle — **suppressed by interferon-γ, so low in viral infection**.
- Antibiotic threshold in LRTI — **>0.5 ng/mL strongly encourages**, **<0.1 strongly discourages**.
- Classic false positives — **surgery, trauma, burns, dialysis, medullary thyroid carcinoma**.
- Classic false negatives — **endocarditis, abscess, empyema, osteomyelitis**.
- Best proven role — **shortening antibiotic duration**, not starting antibiotics.

Question 4. Infection control and antimicrobial stewardship in hospitals 7 MARKS

ANSWER

Textbook Core — 6 Points

1. **Definitions and why they matter in geriatrics. Infection prevention and control (IPC)** is the set of measures that prevent transmission of pathogens within healthcare settings. **Antimicrobial stewardship (AMS)** is a coordinated programme to optimise the **choice, dose, route and duration** of antimicrobials so as to improve outcomes, reduce toxicity and cost, and limit resistance. Older inpatients are the highest-risk group because of **atypical presentation** (fever and leucocytosis may be absent), high device use, prolonged stay and heavy antibiotic exposure.
2. **Core IPC measures. Hand hygiene (WHO 5 moments) is the single most effective intervention**; **standard precautions** plus **transmission-based precautions** (contact, droplet, airborne); appropriate PPE; environmental cleaning and disinfection; safe injection and sharps practice; **isolation or cohorting** of MDRO carriers and *C. difficile*; biomedical waste management; and **device care bundles** for CAUTI, CLABSI, ventilator-associated pneumonia and surgical site infection.
3. **Surveillance.** Continuous surveillance of **device-associated infection rates**, construction of a **hospital antibiogram** to guide empirical therapy, outbreak detection, and MDRO screening. In India, the **ICMR Antimicrobial Resistance Surveillance Network (AMRSN)** and NCDC's HAI surveillance network provide national resistance data.
4. **Core elements of an AMS programme (CDC / ICMR). Leadership commitment** (budget and staff); **accountability** — a named physician lead; **pharmacy expertise**; **action** — formulary restriction and pre-authorisation, **prospective audit and feedback**, an **antibiotic timeout at 48–72 hours**, de-escalation on culture results, **IV-to-oral switch** and defined duration limits; **tracking** (DDD per 100 bed-days, days of therapy); **reporting**; and **education**.
5. **Practical prescribing rules.** Send cultures **before** the first antibiotic dose; choose empirical therapy from the **local antibiogram**, not from memory; apply the **WHO AWaRe classification** — **Access** preferred, **Watch** restricted, **Reserve** for last-line use; use the **shortest effective duration**; and **adjust every dose for renal function**, with therapeutic drug monitoring mandatory for **aminoglycosides and vancomycin** in older adults.
6. **Geriatric-specific stewardship issues.** Do not treat **asymptomatic bacteriuria** or colonisation; avoid **fluoroquinolones** where possible (delirium, tendon rupture, QT prolongation, aortic aneurysm, *C. difficile* — flagged in the Beers criteria); watch for antibiotic-related **drug interactions** (warfarin, statins, antiarrhythmics); recognise antibiotics as a leading precipitant of ***C. difficile* infection** and of delirium; and remember that **vaccination** (influenza, pneumococcal, COVID-19, herpes zoster) is itself a stewardship intervention because it prevents antibiotic-requiring illness.

Latest Update (2023 – present)

7. India launched the **National Action Plan on Antimicrobial Resistance 2.0 (NAP-AMR 2.0, 2025–2029)** on **18 November 2025**, during World AMR Awareness Week. Key changes from NAP-AMR 1.0 (2017–21): **IPC programmes are now mandatory in all healthcare facilities**; **environmental surveillance of antibiotic residues** in wastewater from hospitals, farms and pharmaceutical units is mandatory; the **ICMR AMRSN is being expanded** with genomic surveillance; and **governance is elevated to a standalone pillar**, with 20+ ministries required to submit time-bound, budgeted action plans. It follows the **One Health** approach and is aligned with the **UNGA 2024 political declaration target of a 10% reduction in AMR-related deaths by 2030**.

If this appears as a 15-mark question, add: the burden of AMR (Lancet GRAM: 1.27 million deaths directly attributable globally in 2019; ~2.97 lakh AMR-attributable deaths in India); Indian resistance patterns (carbapenem-resistant *Klebsiella* and *Acinetobacter*, ESBL *E. coli*, *Candida auris*); a full CAUTI/CLABSI/VAP bundle; the Schedule H1 regulation of antibiotic sales; NABH accreditation requirements for hospital infection control committees; AMS outcome metrics and reported Indian results (reduction in ceftriaxone and piperacillin-tazobactam DDDs after AMSP introduction); and barriers to AMS in Indian secondary-care hospitals.

Exam One-Liners

- Single most effective IPC measure — **hand hygiene**.
- AWaRe — **Access, Watch, Reserve**.
- Stewardship intervention with best evidence — **prospective audit and feedback**.
- Antibiotic timeout — **48–72 hours**.
- Indian surveillance network — **ICMR AMRSN**.
- Current national policy — **NAP-AMR 2.0 (2025–2029)**.

PART II

Tuberculosis

Programmatic management and drug-resistant disease, followed by the molecular diagnostic that underpins it.

Question 5. DOTS; treatment, dose and regimens of MDR and XDR tuberculosis; drug-resistant TB

7 MARKS ASKED ×4

ANSWER

Textbook Core – 6 Points

1. **Definitions of drug resistance (WHO 2021 revision). Mono-resistant** — resistance to one first-line drug. **Poly-resistant** — resistance to more than one first-line drug other than to isoniazid and rifampicin together. **MDR-TB** — resistance to at least **isoniazid AND rifampicin**. **RR-TB** — rifampicin resistance with or without other resistance; managed exactly as MDR-TB. **Pre-XDR-TB** — MDR/RR-TB plus resistance to **any fluoroquinolone**. **XDR-TB** — MDR/RR-TB plus fluoroquinolone resistance plus resistance to **at least one Group A drug (bedaquiline or linezolid)**.
2. **DOTS — the five components.** (i) **Sustained political and financial commitment**; (ii) **case detection by quality-assured bacteriology**; (iii) **standardised short-course chemotherapy given under direct observation with patient support**; (iv) an **uninterrupted, quality-assured drug supply and management system**; and (v) **standardised recording, reporting, monitoring and impact measurement**. **DOTS-Plus** extended the framework to programmatic management of drug-resistant TB. In India, RNTCP became the **National TB Elimination Programme (NTEP)** in 2020, with the **Ni-kshay** digital notification portal, **Ni-kshay Poshan Yojana** direct-benefit nutritional support (raised to **₹1000 per month in 2024**), the **Pradhan Mantri TB Mukt Bharat Abhiyaan** with **Ni-kshay Mitra** community adoption, and a national elimination target ahead of the global 2030 goal.
3. **Drug-susceptible TB (for contrast). 2HRZE / 4HR** — two months of isoniazid, rifampicin, pyrazinamide and ethambutol, then four months of isoniazid and rifampicin, given **daily as weight-band fixed-dose combinations** (NTEP moved from thrice-weekly to daily dosing in 2017). Doses: **H 5 mg/kg, R 10 mg/kg, Z 25 mg/kg, E 15 mg/kg**, with **pyridoxine 10 mg daily** — essential in older adults to prevent isoniazid neuropathy.
4. **Current MDR/RR-TB regimens — the core of the answer.**

Regimen	Composition	Use
BPaLM — 6 months (preferred)	**B**edaquiline + **Pa**retomanid + **L**inezolid 600 mg + **M**oxifloxacin	MDR/RR-TB, age ≥14 y. Drop moxifloxacin → **BPaL** if fluoroquinolone-resistant (pre-XDR). **Not** for pregnancy/breastfeeding (no pretomanid safety data)
BDLLfxC — 6 months	**B**edaquiline + **D**elamanid + **L**inezolid 600 mg + **L**evo**flox**acin + **C**lofazimine	Alternative 6-month regimen; usable in **pregnancy, breastfeeding and children**. Becomes **BDLC** if fluoroquinolone-resistant. Requires no prior exposure >1 month to bedaquiline, delamanid or linezolid
9-month all-oral	Bedaquiline-containing (BPaLM or BDLLfxZ)	**Fluoroquinolone-susceptible only** — must not be used in pre-XDR-TB
Longer individualised 18–20 months	Built from Groups A → B → C	When short regimens are unsuitable, and **mandatory for CNS, osteoarticular and disseminated multi-organ TB** — all short regimens are contraindicated in these

5. **Drug groups and key doses. Group A (include all three)** — levofloxacin or moxifloxacin, **bedaquiline**, **linezolid**. **Group B (add both)** — clofazimine, cycloserine or terizidone. **Group C (add to complete)** — ethambutol, delamanid, pyrazinamide, imipenem-cilastatin or meropenem, amikacin, ethionamide, para-aminosalicylic acid. Doses to quote: **bedaquiline 400 mg daily for 2 weeks then 200 mg three times a week for 22 weeks**; **pretomanid 200 mg daily**; **linezolid 600 mg daily**; **moxifloxacin 400 mg daily**; **levofloxacin 750–1000 mg daily**; **delamanid 100 mg twice daily**; **clofazimine 100 mg daily**.
6. **Monitoring and geriatric considerations.** Monthly **sputum smear and culture** for conversion; monthly weight; baseline and serial **ECG for QT interval** (bedaquiline, moxifloxacin, clofazimine and delamanid all prolong QT — correct potassium and magnesium first); **monthly CBC, vision and peripheral nerve assessment for linezolid** (myelosuppression, optic and peripheral neuropathy — the commonest reason for dose reduction to 300 mg or interruption); liver and renal function; TSH (ethionamide, PAS); psychiatric review (cycloserine — psychosis, depression, seizures); and audiometry if an aminoglycoside is unavoidable — **injectable aminoglycosides should be avoided altogether in older adults** because of ototoxicity and nephrotoxicity. In the elderly also: **dose-adjust for the age-related fall in GFR**, review interactions with cardiac and psychotropic drugs, screen for and treat diabetes and malnutrition, and provide family-supported or video-observed treatment rather than daily clinic attendance.

Latest Update (2023 – present)

7. The **WHO Consolidated Guidelines on Tuberculosis, Module 4 (2025 update)** now prioritise **all-oral 6-month**

regimens — BPaLM and BDLLfxC — as the standard of care for MDR/RR-TB, with 9-month all-oral regimens reserved for fluoroquinolone-susceptible disease, and **CNS, osteoarticular and disseminated TB listed as contraindications to all short regimens**. Uptake has accelerated sharply: **34,256 people worldwide started a 6-month regimen in 2024, up from 5,653 in 2023**, across 97 countries. WHO also **strongly recommended 6 months of levofloxacin (6-Lfx) as preventive treatment for household contacts of MDR/RR-TB** in September 2024. India, which carries about **27% of the global rifampicin-resistant TB burden**, has rolled out BPaLM nationally under NTEP, with pooled Indian real-world **treatment success of about 89.7%**, the main toxicities being linezolid-related **anaemia (13.4%)** and **peripheral neuropathy (8.7%)**.

MNEMONIC ANCHOR

Group A = "B-L-Q" — **B**edaquiline, **L**inezolid, **Q**uinolone (all three always included).
Group B = "2 C's" — **C**lofazimine, **C**ycloserine.
Regimens = "6 – 6 – 9 – 18" — BPaLM 6 months · BDLLfxC 6 months · all-oral 9 months (FQ-sensitive only) · longer individualised 18–20 months.

NUMERICAL ANCHORS

DOTS has **5 components** · drug-susceptible regimen **2HRZE/4HR** · bedaquiline **400 mg × 2 weeks → 200 mg thrice weekly × 22 weeks** · linezolid **600 mg daily** · pretomanid **200 mg daily** · moxifloxacin **400 mg daily** · delamanid **100 mg BD** · clofazimine **100 mg daily** · Ni-kshay Poshan Yojana **₹1000/month** · India ≈ **27%** of global RR-TB · Indian BPaLM success **≈90%** · 6-Lfx preventive therapy for MDR contacts.

NAMED EXAMINER TRAPS

(a) RR-TB is managed as MDR-TB — rifampicin resistance alone is enough. **(b)** The **2021 XDR definition changed** — it is no longer "second-line injectable resistance" but **resistance to a Group A drug (bedaquiline or linezolid)**. **(c)** The **9-month regimen is only for fluoroquinolone-susceptible disease** — using it in pre-XDR is a serious error. **(d) Short regimens are contraindicated in CNS, osteoarticular and disseminated TB. (e) Avoid injectable aminoglycosides in older adults. (f)** Always add **pyridoxine** with isoniazid in the elderly. **(g)** Get a **baseline ECG** before starting — three of the four drugs in BPaLM prolong QT.

15-SECOND ANSWER SKELETON

Definitions of resistance (mono → poly → MDR → RR → pre-XDR → XDR with the 2021 change) → the 5 components of DOTS and the Indian NTEP framework → drug-susceptible regimen for contrast → the four current regimens in a table → Groups A/B/C with doses → monitoring (culture, ECG, CBC, vision, nerves) and geriatric caveats → WHO 2025 Module 4 and the Indian BPaLM roll-out.

EXTRA RECALL BOX — TB IN THE OLDER ADULT (THIS IS WHAT MAKES IT A GERIATRICS ANSWER)

Kane's makes three points that must appear: **(i)** older people of all groups are **especially at risk**, having lived through a period of higher TB incidence, having probably never received isoniazid preventive therapy, and having predisposing factors — physiological change, malnutrition and underlying disease — that lead to **reactivation**; they are also at increased risk of **primary infection**, particularly in long-term care facilities. **(ii) TB screening programmes should be implemented in long-term care facilities**, with isoniazid 300 mg/day for 9–12 months for skin-test converters who have an abnormal chest film, diabetes, end-stage renal disease, haematological malignancy, steroid or immunosuppressive therapy, or malnutrition. **(iii)** The single most quotable line: **"A useful rule in geriatric care is to suspect tuberculosis when a patient is inexplicably failing."** Add that **tuberculin skin testing may be falsely negative because of anergy** in older people, so an IGRA or a two-step test is preferred.

Exam One-Liners

- MDR-TB — resistance to **isoniazid + rifampicin**.
- Pre-XDR — MDR/RR + **fluoroquinolone** resistance.
- XDR (2021) — pre-XDR + resistance to **bedaquiline or linezolid**.
- Preferred regimen — **BPaLM for 6 months**; drop the M if FQ-resistant.
- Regimen usable in pregnancy — **BDLLfxC**.
- Group A drugs — **quinolone, bedaquiline, linezolid**.
- Commonest limiting toxicity — **linezolid** (marrow, optic and peripheral nerve).
- Mandatory baseline test — **ECG for QT**.
- MDR contact prophylaxis — **6 months levofloxacin**.

Question 6. GeneXpert test 7 MARKS

ANSWER

Source note: Not covered in the uploaded textbooks; content is from WHO consolidated guidelines on rapid diagnostics and Indian NTEP policy, and is presented as current evidence.

Textbook Core – 6 Points

1. **What it is. Xpert MTB/RIF** is an automated, **cartridge-based, closed-system, real-time PCR** (nucleic acid amplification test) run on the GeneXpert platform. In a single step it **simultaneously detects *Mycobacterium tuberculosis* complex DNA and rifampicin resistance**, the latter by using five molecular beacon probes to detect mutations in the **81-base-pair "hot-spot" (core) region of the *rpoB* gene**, which harbours >95% of rifampicin-resistance mutations. **Result in about 2 hours**, with minimal training and biosafety requirements — hence its Indian programme name, **CBNAAT** (cartridge-based nucleic acid amplification test).
2. **Xpert MTB/RIF Ultra.** The improved-generation assay adds two **multi-copy amplification targets (IS6110 and IS1081)**, a larger reaction chamber and **melting-curve analysis** for rifampicin resistance. This lowers the limit of detection from about **114 to 16 colony-forming units/mL**, giving markedly better sensitivity in **smear-negative, paucibacillary, HIV-associated, paediatric and extrapulmonary disease**. Ultra reports an additional semi-quantitative **"trace"** category, which is treated as positive in high-risk groups but may require clinical correlation elsewhere.
3. **Performance.** Against culture, pooled **sensitivity ≈85% and specificity ≈98%** for pulmonary TB; sensitivity is about **98% in smear-positive but only ≈67% in smear-negative** disease (Ultra improves this). For **rifampicin resistance, sensitivity ≈95% and specificity ≈98%**. It therefore vastly outperforms Ziehl–Neelsen smear microscopy, which needs about 10,000 bacilli/mL.
4. **Indications.** WHO recommends Xpert MTB/RIF or Ultra as the **initial diagnostic test in all people with signs or symptoms of TB**, replacing smear microscopy. It is particularly indicated in **presumptive MDR-TB, HIV-associated TB, paediatric TB, and extrapulmonary specimens** — and it is the **preferred initial test on cerebrospinal fluid for TB meningitis** (where a rapid answer changes outcome), as well as on lymph node aspirate, pleural and other body fluids, gastric aspirate and tissue.
5. **Limitations — the marks are here.** It **cannot distinguish live from dead bacilli**, so it must **never be used for treatment monitoring or as a test of cure** (remains positive for months). It **does not detect isoniazid or fluoroquinolone resistance** — that needs a line probe assay, Xpert MTB/XDR or sequencing. **False rifampicin resistance** can arise from silent or borderline *rpoB* mutations, so a positive result in a low-prevalence setting should be confirmed by a second method. Sensitivity is **lower in pleural fluid and in paucibacillary extrapulmonary disease**, so a negative result never excludes TB. Practical constraints: stable electricity, ambient temperature control, annual module calibration, and cartridge cost.
6. **Indian context.** Under NTEP, **universal drug susceptibility testing** mandates a rapid molecular test with rifampicin resistance detection for **every** diagnosed TB patient, and CBNAAT machines have been placed down to district and sub-district level. India also uses the indigenous **Truenat MTB / MTB Plus and MTB-RIF Dx** — a chip-based, battery-operated, portable real-time PCR system (Molbio, Goa), **WHO-endorsed in 2020**, which can be deployed at primary health centre level without air conditioning and has brought molecular diagnosis to peripheral settings.

Latest Update (2023 – present)

7. The WHO rapid-diagnostics menu has broadened well beyond Xpert alone: **targeted next-generation sequencing (tNGS) directly on sputum** was recommended in 2023 for comprehensive drug-resistance profiling in one test; **low-complexity automated assays (Xpert MTB/XDR)** now detect **isoniazid, fluoroquinolone, ethionamide and amikacin resistance** as a reflex test after a positive Xpert MTB/RIF; and **computer-aided detection (CAD) software on digital chest radiographs** is recommended for TB triage in people aged 15 and over. **Tongue-swab sampling** with Xpert Ultra — which removes the need for sputum production, a real advantage in frail and cognitively impaired older adults — is under active evaluation for programmatic use.

Exam One-Liners

- Principle — **cartridge-based real-time PCR**; target the **81-bp *rpoB* hot-spot**.
- Detects — **M. tuberculosis complex + rifampicin resistance**, in **2 hours**.
- Ultra adds — **IS6110 and IS1081**; limit of detection **16 CFU/mL**; reports **"trace"**.
- Best extrapulmonary use — **CSF in TB meningitis**.
- Never use for — **treatment monitoring or test of cure**.
- Does not detect — **isoniazid or fluoroquinolone resistance**.
- Indian portable alternative — **Truenat**.

PART III

Viral and Vaccine-Preventable Infection

The four vaccine-preventable illnesses that dominate morbidity in older adults, followed by the consolidated immunisation schedule and a high-consequence viral haemorrhagic fever.

Question 7. Herpes zoster and post-herpetic neuralgia 15 MARKS RECURRING

ANSWER

Textbook Core — 10 Points

1. **Definition.** Herpes zoster (shingles) is **reactivation of latent varicella-zoster virus (VZV)** from the **dorsal root ganglion**, where it lies dormant after childhood chickenpox. Reactivation occurs because of **immunosenescence**. **Post-herpetic neuralgia (PHN)** is severe shooting, burning or stabbing pain persisting **beyond 3 months** after the rash.
2. **Epidemiology and risk.** Incidence rises considerably after **age 50**, with a **50% lifetime risk by 85 years**. Incidence is **11.8 per 1000 per year in those over 60** versus 1.2–3.4 per 1000 per year at all ages. **Increasing age is the strongest risk factor for PHN**; others are severe rash, severe acute pain and prodromal pain.
3. **Clinical features.** A **prodrome of neuropathic pain** — intense burning or aching in the dermatome — precedes the rash. Then a **unilateral, dermatomal cluster of vesicles on an erythematous base**, which **crusts over in 7–10 days**, after which the patient is no longer contagious.
4. **Important clinical variants. Herpes zoster ophthalmicus** (V1 division; **Hutchinson's sign** = vesicles on the nose tip indicating nasociliary involvement — urgent ophthalmology referral); **Ramsay Hunt syndrome** (facial palsy with vesicles in the ear canal); **disseminated zoster** in the immunosuppressed.
5. **Complications. PHN is the commonest.** Also **zoster encephalitis** — most common in advanced age, immunosuppression and disseminated cutaneous zoster; **contralateral hemiplegia** occurring weeks after zoster ophthalmicus (VZV granulomatous arteritis) accounts for up to **one-third** of CNS zoster; post-herpetic itch; secondary bacterial infection; myelitis; ocular complications.
6. **Diagnosis.** Usually **clinical**. **VZV DNA PCR** from vesicle fluid confirms. In suspected CNS disease, CSF shows **lymphocytic pleocytosis with raised protein** (also seen in up to 40% of zoster patients *without* CNS involvement); **CSF VZV PCR specificity >95%, sensitivity 80–95%**; viral culture is rarely helpful.
7. **Antiviral therapy.** Start ideally **within 72 hours** of symptom onset for maximum effect. **Aciclovir 800 mg five times daily**, or **valaciclovir 1 g three times daily**, or famciclovir — for **7 days**. Early therapy prevents new vesicles and reduces pain intensity and duration, viral shedding and **PHN incidence**. **Reduce dose for renal impairment** — GFR falls ~30% with normal ageing. IV aciclovir 30 mg/kg/day in divided doses for CNS disease.
8. **Acute pain management.** Paracetamol and, where safe, an NSAID; **moderate-to-severe pain may require opioids**. Corticosteroids or gabapentin may be added if pain persists — but **corticosteroids have NOT been shown to reduce the incidence of PHN in the elderly**.
9. **Treatment of established PHN.** Approved options: **topical 5% lidocaine patch, gabapentin and pregabalin**. **Nortriptyline** and opioids are also of benefit; **combination therapy** is often necessary. Avoid **amitriptyline** in older adults — strongly anticholinergic (dry mouth, retention, constipation, delirium, tachycardia, blurred vision, falls). Gabapentinoids need renal dose reduction and cause sedation and ataxia.
10. **Prevention (textbook position). Zostavax**, a **live attenuated** vaccine, is recommended for immunocompetent adults **≥60 years** (permitted from 50). It reduces the incidence of **herpes zoster by 51%** and of **PHN by 66%**, and also reduces ophthalmic zoster and zoster hospitalisations.

Latest Updates (2023 – present)

11. **Recombinant zoster vaccine (RZV, Shingrix) has replaced the live vaccine.** Two IM doses 2–6 months apart, efficacy **>90%**, now the **preferred vaccine from age 50** and — because it is **non-live** — the only zoster vaccine usable in the **immunocompromised**. Live Zostavax was discontinued in the USA (2020) and many other countries.
12. **Zoster vaccination and dementia risk.** *Nature* 2025 (Eyting et al., Wales natural experiment) found roughly a **20% relative reduction in dementia** over 7 years after zoster vaccination; *Nature Medicine* 2024 (Taquet et al.) showed RZV was associated with lower dementia risk than the live vaccine — **17% more diagnosis-free time (≈164 extra days)**. Large cohort data (Kaiser Permanente, Medicare) are concordant. This is now a strong additional argument for offering RZV to all older adults.

Current Practice Update

Item	Textbook	Current recommendation	Exam approach
Vaccine	Live attenuated Zostavax, age ≥60	Recombinant RZV, 2 doses, age ≥50, including immunocompromised	Quote Zostavax figures (51% / 66%), then name RZV as the current standard
Steroids	May be added if pain persists	Do not use to prevent PHN; use only for selected acute severe pain	State explicitly that steroids do not prevent PHN

PHN first-line drug	Lidocaine patch, gabapentin, TCA	Gabapentinoid or lidocaine patch first; TCAs avoided in frail elderly (Beers)	Name the drug and the geriatric caveat together
Wider benefit	Not discussed	Zoster vaccination associated with reduced dementia incidence	Use as the closing "latest update" line

MNEMONIC ANCHOR

"72 – 7 – 3" → antiviral within **72 hours** · give for **7 days** · call it **PHN after 3 months**.

NUMERICAL ANCHORS

50% lifetime risk by age 85 · incidence 11.8/1000/yr over 60 · crusting at 7–10 days · antiviral window 72 h · aciclovir 800 mg × 5/day · valaciclovir 1 g TDS · PHN threshold 3 months · Zostavax ↓HZ 51%, ↓PHN 66% · RZV efficacy >90% · dementia risk ↓ ~20%.

NAMED EXAMINER TRAPS

(a) PHN is defined at **3 months**, not 1 month. **(b)** Corticosteroids do **not** reduce PHN incidence. **(c)** RZV is **non-live** — therefore safe in immunosuppression, unlike Zostavax. **(d) Hutchinson's sign** means nasociliary involvement — same-day ophthalmology. **(e)** Do not start amitriptyline in a frail older adult for PHN. **(f)** Vesicles are infectious until crusted — isolate from non-immune contacts and pregnant staff.

15-SECOND ANSWER SKELETON

Define (VZV reactivation, dorsal root ganglion, immunosenescence) → epidemiology (50% by 85) → prodromal pain then dermatomal vesicles → variants (ophthalmicus, Ramsay Hunt) → complications (PHN, encephalitis, contralateral hemiplegia) → PCR → antiviral <72 h × 7 days, renal dose → acute pain ladder → PHN: gabapentinoid / lidocaine patch / nortriptyline → prevention with RZV → dementia signal.

EXTRA RECALL BOX

Zoster ophthalmicus followed weeks later by contralateral hemiplegia = VZV granulomatous arteritis — a classic viva question; angiography shows unilateral arteritis or thrombosis and CT/MR shows infarction. **Zoster sine herpete** = dermatomal pain with no rash, diagnosed by PCR/serology. **Zoster is a marker of immunosuppression** — in an older Indian patient, consider undiagnosed diabetes, malignancy, HIV and steroid use.

Exam One-Liners

- Site of latency — **dorsal root ganglion**.
- Antiviral window — **72 hours**.
- Drug of choice — **aciclovir / valaciclovir**, renally dose-adjusted.
- PHN definition — pain **>3 months** after rash.
- PHN drugs — **gabapentin, pregabalin, lidocaine 5% patch, nortriptyline**.
- Vaccine of choice today — **recombinant zoster vaccine, 2 doses, ≥50 years**.
- Most feared eye sign — **Hutchinson's sign**.

Question 8. COVID-19 in the older adult — presentation and management 7 MARKS

ANSWER

Textbook Core — 6 Points

1. **Why older adults are the highest-risk group. Immunosenescence and inflammaging**, multimorbidity, reduced pulmonary and cardiac reserve, and **frailty** make **age the single strongest predictor of severe disease and death**. Residents of long-term care facilities are at particular risk because of close cohabitation and shared staff. The great majority of COVID-19 hospitalisations occur in those **≥65 years**, with the highest rates over 75.
2. **Atypical presentation. Delirium or acute confusion may be the sole presenting feature.** Also falls, functional decline, anorexia, generalised weakness, unexplained tachypnoea and **"silent hypoxia"** — significant desaturation without breathlessness. **Fever may be absent** because of the blunted febrile response of ageing; anosmia is reported less often than in younger patients.
3. **Assessment. Pulse oximetry** is essential (target SpO_2 94–98%, or 88–92% if at risk of hypercapnia); respiratory rate; CBC (lymphopenia), CRP, renal and liver function, blood glucose, D-dimer, ECG and chest imaging; **RT-PCR or rapid antigen test**. Document a **Clinical Frailty Scale** score and comorbidity — used **alongside, never instead of**, clinical assessment when making escalation decisions. Screen for delirium with the **4AT**.
4. **Mild-to-moderate disease in a high-risk older adult.** Start an antiviral **within 5 days of symptom onset**. **Nirmatrelvir/ritonavir** is first line — dose-reduce if eGFR 30–60 mL/min, **avoid if eGFR <30**, and check for **CYP3A4 interactions**, which are the major geriatric pitfall (statins, calcium-channel blockers, amiodarone, DOACs, tacrolimus, some antipsychotics — many need temporary suspension or dose reduction). Alternatives: **remdesivir** (3-day IV course) or **molnupiravir**.
5. **Severe disease (hypoxia).** Controlled oxygen, awake proning if tolerated, **dexamethasone 6 mg daily for up to 10 days** (only if hypoxic), plus **tocilizumab or baricitinib** in rapidly progressive inflammatory disease; escalate to HFNO/NIV/ventilation according to agreed goals of care. Give **LMWH thromboprophylaxis** unless contraindicated. Antibiotics only when bacterial co-infection is genuinely suspected. Monitor for hyperglycaemia and steroid-induced delirium.
6. **The geriatric care bundle. Delirium prevention** (reorientation, hearing aids and spectacles, hydration, early mobilisation, sleep hygiene, minimising sedatives and restraints); nutrition and swallow assessment; **pressure-ulcer prevention**; medication review; **early rehabilitation** for deconditioning and post-COVID functional decline; and explicit **advance care planning and goals-of-care discussion** with the patient and family. Prevention: **updated COVID-19, influenza and pneumococcal vaccination**, ventilation, and IPC in long-term care.

Latest Update (2023 – present)

7. The **2025 IDSA guideline update** continues to **strongly recommend nirmatrelvir/ritonavir** for high-risk adults with mild-to-moderate COVID-19. However, the **PANORAMIC and CanTreatCOVID platform trials (NEJM 2026)**, conducted in a largely **vaccinated** higher-risk community population, found **faster symptom recovery but no significant reduction in hospitalisation or death** — so the absolute benefit is now understood to be greatest in the **unvaccinated, immunocompromised and oldest-old**. CDC 2025–2026 guidance recommends COVID-19 vaccination for adults **≥65 years through individual-based (shared clinical) decision-making**, and CDC MMWR 2026 data show only about **31% of eligible outpatients ≥65 years receive an antiviral within 7 days** — under-treatment, not over-treatment, is the current practice gap.

If this appears as a 15-mark question, add: pathophysiology (ACE2, cytokine response, endothelial injury, immunothrombosis); a severity classification table (mild / moderate / severe / critical with SpO_2 and respiratory rate cut-offs); the Indian Ministry of Health clinical management protocol and its home-isolation criteria; specific management of long COVID / post-COVID syndrome in older adults (fatigue, breathlessness, cognitive impairment, deconditioning, and the role of pulmonary rehabilitation); the ethical framework for triage and ceilings of care in frail older adults; and lessons for outbreak management in Indian old-age homes.

Exam One-Liners

- Commonest atypical presentation — **delirium**.
- Most dangerous missed sign — **silent hypoxia**.
- Antiviral window — **within 5 days**.
- Biggest practical problem with nirmatrelvir/ritonavir — **CYP3A4 drug interactions**.
- Steroids — only if **hypoxic**; dexamethasone 6 mg × up to 10 days.
- Frailty score — used **with**, not instead of, clinical judgement.

Question 9. Prevention and management of influenza 7 MARKS

ANSWER

Textbook Core – 6 Points

1. **Why it matters in older adults.** Influenza A and B cause annual epidemics, and **the great majority of influenza deaths occur in those aged over 65** — through **primary viral pneumonia**, **secondary bacterial pneumonia** (*S. pneumoniae*, *S. aureus*, *H. influenzae*), and **decompensation of heart failure, COPD, diabetes and cerebrovascular disease**. The risk of **myocardial infarction and stroke rises sharply in the week following influenza**. Long-term care facilities are the classic setting for explosive outbreaks with high mortality.
2. **Presentation in the elderly.** Frequently **atypical**: fever may be absent or low-grade, and the illness may present as **delirium, functional decline, falls, anorexia, generalised weakness or an exacerbation of a chronic disease** rather than the classical abrupt fever, myalgia, headache and dry cough. Kane's directs that **at the onset of a suspected respiratory viral outbreak in a facility, a nasopharyngeal wash or swab should be obtained for virus isolation and rapid diagnostic testing for influenza A and other common viruses**.
3. **Diagnosis. RT-PCR (or a multiplex respiratory panel) on a nasopharyngeal swab is the test of choice** — sensitive, specific, and able to distinguish influenza A from B and to subtype. **Rapid antigen tests are quick but insensitive**, so a negative result during an outbreak does not exclude influenza and should not delay treatment. Add pulse oximetry, chest radiograph if hypoxic or if pneumonia is suspected, and CBC, renal function and CRP.
4. **Vaccination — the cornerstone. Annual inactivated influenza vaccine for everyone aged 65 and over**, for **residents and staff** of long-term care facilities, and for household contacts. Kane's records that over ten seasons, influenza vaccination in community-dwelling older people was associated with a **hazard ratio of 0.75 for hospitalisation for pneumonia or influenza and 0.52 for death**. Because immunosenescence blunts the antibody response, **high-dose, adjuvanted or recombinant formulations are preferentially recommended over standard-dose vaccine from age 65**. The **live attenuated (nasal) vaccine is contraindicated in older adults**. Timing in India: before the monsoon in most of the country, with a second winter peak in the north; the Southern Hemisphere formulation is generally appropriate.
5. **Antiviral treatment.** Start **as early as possible and within 48 hours of symptom onset** for maximum benefit — but **treat any hospitalised, severe or high-risk patient regardless of how long symptoms have been present**. **Oseltamivir 75 mg twice daily for 5 days** is first-line, and the frequently missed geriatric point is that the dose must be **reduced to 30 mg twice daily if creatinine clearance is 30–60 mL/min**, with further reduction below that. Alternatives: **inhaled zanamivir** (avoid in asthma and COPD), intravenous **peramivir**, and **baloxavir marboxil** as a single oral dose. Add antibiotics only where bacterial co-infection is genuinely suspected. Use paracetamol for fever; **avoid aspirin**.
6. **Outbreak control in long-term care.** Kane's protocol: give **oseltamivir or zanamivir within 24–48 hours of an outbreak of suspected influenza**, to **residents and staff**, and continue **throughout the outbreak** — in practice, oseltamivir 75 mg once daily as chemoprophylaxis for at least 14 days and until 7 days after the last case. Alongside this: **annual vaccination of residents and staff**, isolation and cohorting of cases, restriction of new admissions and visitors, exclusion of symptomatic staff from work, hand hygiene, droplet precautions with masks, and environmental cleaning.

Latest Update (2023 – present)

7. Current WHO and national guidance now emphasise **co-administration of influenza vaccine with COVID-19 and RSV vaccines at the same visit** for older adults, since separate visits reduce uptake; and **ACIP preferentially recommends the high-dose, adjuvanted or recombinant influenza vaccine for all adults aged 65 and over** rather than standard-dose. The Indian problem is structural: **influenza vaccine is not part of the Universal Immunisation Programme** and is available only through the private sector or selected state initiatives, so out-of-pocket cost and lack of physician recommendation are the main barriers. The strongest practical levers in Indian practice are therefore **vaccinating healthcare workers and long-term care staff**, and **opportunistic vaccination at every comprehensive geriatric assessment**.

Exam One-Liners

- Most influenza deaths occur in those **over 65**.
- Test of choice — **RT-PCR on nasopharyngeal swab**.
- Vaccine of choice from 65 — **high-dose, adjuvanted or recombinant**; live nasal vaccine contraindicated.
- Kane's vaccination figures — **HR 0.75 for hospitalisation, 0.52 for death**.
- Antiviral — **oseltamivir 75 mg BD × 5 days**; **30 mg BD if CrCl 30–60**.
- Treat within **48 hours**, but treat any hospitalised or severe case regardless of timing.
- Outbreak in a facility — **chemoprophylaxis for residents and staff within 24–48 hours**.

Question 10. Vaccinations in elderly patients 7 MARKS

ANSWER

Textbook Core – 6 Points

1. **Rationale. Immunosenescence** — thymic involution, reduction in naive T cells, impaired B-cell affinity maturation and class switching — together with **inflammaging**, both increases susceptibility to infection and **blunts the response to vaccines**, which is why older adults need higher-potency formulations. Vaccine-preventable infection in older adults causes hospitalisation, delirium, functional decline and death, and drives a large share of antibiotic use, so **vaccination is also an antimicrobial stewardship intervention**. Vaccination status should be recorded and reviewed at **every comprehensive geriatric assessment**.
2. **Influenza. Annually for everyone aged 65 and over**, and for **residents and staff of long-term care facilities**. **High-dose, adjuvanted or recombinant formulations are preferred over standard-dose** from age 65. The **live attenuated nasal vaccine is contraindicated** in this group.
3. **Pneumococcal.** A single dose of a **pneumococcal conjugate vaccine — PCV20 or PCV21 — for all PCV-naïve adults**. If **PCV15** is used it must be followed by **PPSV23 at least one year later** (or after 8 weeks if the patient is immunocompromised, or has a cerebrospinal fluid leak or a cochlear implant). Pathy's states that all adults aged 65 and over should be vaccinated, and that adults with **asplenia, CSF leak, cochlear implants or immunosuppressing conditions** should also be vaccinated regardless of age.
4. **Herpes zoster. Two doses of the recombinant zoster vaccine (RZV, Shingrix), 2–6 months apart, from age 50**, including in the **immunocompromised** — it is non-live, with efficacy above **90%**. It has replaced the live Zostavax, which was recommended from age 60 and reduced herpes zoster by **51%** and post-herpetic neuralgia by **66%**.
5. **Tetanus, diphtheria and pertussis. Td booster every 10 years**, and every 5 years for a tetanus-prone wound, with **one dose given as Tdap** to cover pertussis, since pertussis immunity wanes. Kane's makes the important point that **many older people never received primary vaccination** and therefore require **tetanus toxoid plus 250–500 units of tetanus immune globulin**, with the series completed by a second injection at 4–6 weeks and a third at 6–12 months.
6. **COVID-19, RSV and situational vaccines.** An **updated COVID-19 vaccine for adults aged 65 and over**, now through individual/shared clinical decision-making. **RSV vaccine — a single dose for all adults aged 75 and over, and for those aged 50–74 with high-risk conditions, frailty, or residence in a long-term care facility**; it is currently a **one-time dose, not annual**. Situational: **hepatitis B** in diabetes and dialysis; **rabies** post-exposure prophylaxis; **typhoid, hepatitis A and yellow fever** for travel; and **meningococcal vaccine for Hajj and Umrah pilgrims** — a common and examinable Indian indication. **Live vaccines are contraindicated in significant immunosuppression.**

Latest Update (2023 – present)

7. Three changes dominate current adult immunisation practice. **(i)** ACIP **lowered the age-based pneumococcal conjugate recommendation from 65 to 50 years** (October 2024, effective January 2025), and added **PCV21 (CAPVAXIVE)** alongside PCV20, PCV21 offering broader serotype coverage and superior immunogenicity for 10 of its 11 unique serotypes. **(ii)** ACIP **lowered the risk-based RSV vaccination age from 60 to 50 years** (April 2025), retaining the universal recommendation at 75. **(iii)** COVID-19 vaccination for those aged 65 and over moved from a universal recommendation to **individual-based (shared clinical) decision-making** for the 2025–26 season. In addition, evidence that **recombinant zoster vaccination is associated with roughly a 20% lower incidence of dementia** (*Nature*, 2025) provides a powerful further argument for uptake in this population.

Quick-Reference Schedule

Vaccine	Who and when	Key points
Influenza	Annually, all ≥65; LTC residents and staff	**High-dose / adjuvanted / recombinant** preferred; live nasal contraindicated
Pneumococcal	Single PCV20 or PCV21 from age 50 if PCV-naïve	If PCV15 used → **PPSV23 after ≥1 year** (8 weeks if immunocompromised)
Herpes zoster	**RZV × 2 doses**, 2–6 months apart, from age 50	Non-live → safe in immunosuppression; efficacy >90%
Td / Tdap	Booster every 10 years; one dose as Tdap	Every 5 years for tetanus-prone wounds; many elderly never primed
COVID-19	Updated vaccine, ≥65 years	Now **shared clinical decision-making** (2025–26)
RSV	All ≥75; ages 50–74 if high risk or frail or in LTC	**One-time dose**, not annual

INDIAN CONTEXT — WHAT TO WRITE FOR THE INDIAN EXAMINER

India has **no national adult immunisation programme**: the Universal Immunisation Programme covers children and pregnant women only, so influenza, pneumococcal, zoster and RSV vaccines are almost entirely **out-of-pocket in the private sector**. The **Association of Physicians of India (API) and Geriatric Society of India** have issued adult immunisation recommendations, and **NPHCE** geriatric clinics are the natural delivery platform. Practical answer points: prioritise **influenza and pneumococcal** as the two with the strongest cost-benefit case; **vaccinate caregivers and long-term care staff**; use **every hospital contact opportunistically**; counsel on cost transparently; and remember that **tetanus toxoid coverage is genuinely poor in older rural Indians**, making tetanus-prone wound management a real and examinable issue.

Exam One-Liners

- Underlying reason for altered schedules — **immunosenescence**.
- Influenza — **annual, high-dose/adjuvanted/recombinant from 65**.
- Pneumococcal — **single PCV20 or PCV21 from age 50**.
- Zoster — **2 doses of recombinant vaccine from age 50**, safe in immunosuppression.
- RSV — **all ≥75; 50–74 if high risk**; one-time dose.
- Contraindicated in immunosuppression — **all live vaccines**.
- India — **no adult immunisation programme**; delivery through NPHCE geriatric clinics.

Question 11. Ebola virus — clinical features, spread, treatment and prevention 7 MARKS

ANSWER

Source note: Not covered in the uploaded textbooks; content is from WHO and current outbreak-response literature.

Textbook Core – 6 Points

1. **Aetiology.** Ebolavirus is an enveloped, single-stranded, negative-sense RNA **filovirus** (family Filoviridae). Six species are recognised, four of which cause human disease: ***Zaire ebolavirus*** — much the most virulent, with case fatality of **25–90%** — *Sudan*, *Bundibugyo* and *Taï Forest* ebolavirus. The natural reservoir is believed to be **fruit bats**, with index cases arising from contact with infected bats or non-human primates (bushmeat). **Incubation 2–21 days.**
2. **Spread.** Human-to-human transmission is by **direct contact with blood or body fluids** — vomit, stool, urine, saliva, sweat, semen, breast milk — of a **symptomatic** or **dead** person, entering through broken skin or mucous membranes, or through contact with contaminated surfaces, linen and needles. **It is not airborne.** The classic outbreak amplifiers are **traditional burial practices** involving washing of the body, and **nosocomial transmission to healthcare workers** where PPE is inadequate. Critically, the virus **persists in immune-privileged sites — semen for 18 months or more**, ocular fluid, CNS and breast milk — causing sexual transmission, late relapse, and new outbreaks seeded from survivors.
3. **Clinical features.** Abrupt onset of **fever, profound weakness, myalgia, headache and sore throat** (the "dry" phase), followed at days 3–10 by the **"wet" phase — high-volume vomiting and profuse watery diarrhoea** causing massive fluid and electrolyte loss, hypovolaemic shock, acute kidney injury and hepatitis. **Haemorrhage occurs in only about 20%** (petechiae, mucosal and gastrointestinal bleeding) — **death at days 6–16 is usually from hypovolaemic and septic shock with severe electrolyte derangement, not from bleeding**, which is the point examiners look for. Late features: encephalopathy, seizures, DIC and multi-organ failure. Survivors develop **post-Ebola syndrome** — arthralgia, **uveitis which may cause blindness**, hearing loss and cognitive difficulty.
4. **Diagnosis. RT-PCR on whole blood is the confirmatory test**; it may be **negative in the first 3 days**, so a symptomatic patient must be retested at 72 hours. Antigen-detection rapid tests are used for triage, and ELISA detects IgM and IgG. Supportive findings: early leucopenia then neutrophilia, **thrombocytopenia**, markedly **raised transaminases with AST greater than ALT**, rising creatinine, and profound **hypokalaemia and hyponatraemia**. All specimens are **category A infectious substances** requiring high-level biosafety handling and specialised transport. In an endemic region the differential includes **malaria — test every patient** — typhoid, Lassa fever, meningococcaemia and dengue.
5. **Treatment.** The foundation, which by itself substantially reduces mortality, is **aggressive supportive care**: high-volume oral and intravenous **fluid and electrolyte replacement** with particular attention to **potassium, magnesium and calcium**, antiemetics, nutritional support, analgesia, correction of hypoglycaemia, and **empirical antimalarials and antibiotics** for co-infection, with organ support as needed. **Specific therapy for *Zaire ebolavirus*:** two monoclonal antibody products are approved on the basis of the **PALM trial — Inmazeb (atoltivimab/maftivimab/odesivimab)** and **Ebanga (ansuvimab)** — both of which substantially reduced mortality compared with ZMapp and remdesivir. **No approved therapy exists for Sudan virus disease.**
6. **Prevention. Isolation with strict barrier nursing** and full PPE, dedicated staff and equipment, safe injection practice and safe waste disposal; **contact tracing with 21-day monitoring**; **safe and dignified burials**; community engagement; and avoidance of bushmeat. **Vaccines against *Zaire ebolavirus*: rVSV-ZEBOV-GP (Ervebo)**, a single-dose live vaccine, WHO-prequalified and used in **ring vaccination** of contacts, contacts-of-contacts and healthcare workers; and the two-dose **Ad26.ZEBOV / MVA-BN-Filo (Zabdeno / Mvabea)** regimen for preventive use. **Survivors must use condoms and undergo semen testing until two negative PCR results.**

Latest Update (2023 – present)

7. Outbreak response has become dramatically faster and now follows a standard template. In the **2025 Democratic Republic of Congo (Kasai) outbreak of *Zaire ebolavirus***, declared in September 2025 and declared over in December 2025, **ring vaccination with Ervebo and treatment with the licensed monoclonals were deployed within days of declaration** — the fastest response yet achieved, and one now formalised in WHO's outbreak playbook alongside pre-positioned vaccine stockpiles. For **Sudan virus disease**, against which neither the licensed vaccine nor the licensed monoclonal antibodies are effective, candidate vaccines are held in a **"trial-ready" stockpile** so that randomised ring-vaccination trials can be launched within days, as was done in **Uganda's 2022 and 2025 outbreaks**.

Exam One-Liners

- Family — **filovirus**; reservoir — **fruit bats**; incubation **2–21 days**.
- Transmission — **direct contact with body fluids of a symptomatic or dead person; not airborne**.
- Commonest cause of death — **hypovolaemic shock from vomiting and diarrhoea**, not haemorrhage.
- Confirmatory test — **RT-PCR on blood**; may be negative in the first 3 days.
- Approved drugs (Zaire only) — **Inmazeb and Ebanga**.
- Vaccines — **Ervebo (single dose, ring vaccination)** and **Zabdeno/Mvabea**.
- Persistence site of greatest public health importance — **semen**.

PART IV

Tropical and Vector-Borne Infection

The seasonal Indian febrile illnesses. Each is a distinct question, but all share the same geriatric trap — atypical presentation with a blunted febrile response and a narrow margin between under- and over-resuscitation.

Question 12. Leptospirosis and dengue in the older adult 7 MARKS

ANSWER

Source note: Dengue and leptospirosis are **not covered adequately in the uploaded textbooks**. The geriatric principles below (blunted febrile response, atypical presentation, narrowed homeostatic reserve, the need to consider drug and renal factors) are taken from Pathy's and Kane's; the disease-specific content is drawn from current national and international guidance and is clearly separated.

Textbook Core — 6 Points

1. **Why these matter in the elderly (textbook principle).** Both are **monsoon-season febrile illnesses** that are highly prevalent in India. Older adults have **higher morbidity and mortality** because the **febrile response is blunted or absent**, symptoms are under-reported, coexisting disease masks acute infection, and **acute unexplained functional deterioration or confusion** may be the only presenting feature. **Absence of fever or leucocytosis does not exclude serious infection.**
2. **Dengue — clinical.** WHO classification: **dengue without warning signs**; **dengue with warning signs** (abdominal pain or tenderness, persistent vomiting, clinical fluid accumulation, mucosal bleeding, lethargy or restlessness, liver enlargement >2 cm, rising haematocrit with rapidly falling platelets); and **severe dengue** (shock, severe bleeding, severe organ impairment). Older adults **less often** show the classical rash and retro-orbital pain and **more often** present with **"expanded dengue syndrome"** — encephalopathy, myocarditis, acute kidney injury, hepatitis and decompensation of existing heart failure or diabetes.
3. **Dengue — diagnosis. NS1 antigen** (days 1–5), **IgM ELISA** (after day 5), RT-PCR where available; serial **CBC** (thrombocytopenia with rising haematocrit), liver and renal function, ECG. **Geriatric pitfall:** baseline anaemia, chronic kidney disease and diuretic therapy make **haematocrit an unreliable marker of plasma leakage** — a "normal" haematocrit may already represent significant haemoconcentration.
4. **Dengue — management. No antiviral exists**; treatment is careful fluid replacement. Use **weight-based crystalloids titrated to haematocrit, urine output and haemodynamics**, with **far tighter monitoring in the elderly** because the window between hypovolaemia and **iatrogenic fluid overload / pulmonary oedema** is very narrow. **Paracetamol only** — **avoid NSAIDs and aspirin** (bleeding and acute kidney injury). Platelet transfusion only for **clinically significant bleeding**, never for a platelet number alone. **Withhold antiplatelets and anticoagulants during the thrombocytopenic phase** and restart deliberately — a key geriatric decision.
5. **Leptospirosis — clinical.** Zoonosis from **rodent urine and floodwater**; risk in farmers, paddy workers and those wading through contaminated water — heavily represented in older rural and coastal populations (Kerala, Tamil Nadu, Gujarat, Andaman). Classically **biphasic**: a septicaemic phase (abrupt fever, **severe myalgia especially of the calves**, **conjunctival suffusion**, headache) followed by an immune phase. Severe forms: **Weil's disease** (jaundice + acute kidney injury + haemorrhage), **pulmonary haemorrhage syndrome** (highest mortality), myocarditis and aseptic meningitis.
6. **Leptospirosis — diagnosis and treatment. Microscopic agglutination test (MAT)** is the reference standard; **IgM ELISA / rapid tests** for early diagnosis; blood culture in the first week and urine culture in the second; PCR. **Mild disease — doxycycline 100 mg twice daily for 7 days**. **Severe disease — IV penicillin G or ceftriaxone 1–2 g daily.** Watch for the **Jarisch–Herxheimer reaction**. Supportive care is decisive: **early dialysis for AKI improves survival**, and ventilatory support for pulmonary haemorrhage.

Latest Update (2023 – present)

7. **India's first dengue vaccine was approved on 21 July 2026**: the DCGI granted marketing authorisation to **TAK-003 (Qdenga)**, a live-attenuated tetravalent vaccine given as **two doses three months apart, without pre-vaccination screening** — but licensed only for ages **4–60 years**, so it **does not currently cover the older adult population**, in whom **vector control, early recognition and meticulous fluid management remain the only strategy**. (TAK-003 was WHO-prequalified in May 2024; 7-year TIDES data show ~84% efficacy against hospitalised dengue. India's indigenous single-dose candidate **DengiAll** — ICMR/Panacea Biotec — is in Phase 3.) For leptospirosis, current NCVBDC/ICMR practice supports **presumptive doxycycline for acute undifferentiated febrile illness during monsoon in endemic districts**, and **doxycycline 200 mg weekly chemoprophylaxis** for high-risk post-flood exposure, as used in Kerala's post-flood outbreak response.

If this appears as a 15-mark question, add: a comparison table of dengue vs leptospirosis vs scrub typhus vs malaria in acute undifferentiated febrile illness (a favourite Indian examiner question — conjunctival suffusion and calf tenderness for lepto, eschar and doxycycline response for scrub typhus, thrombocytopenia with haemoconcentration for dengue); the dengue fluid algorithm by group A/B/C; criteria for ICU admission; the Faine's criteria for leptospirosis; complications of both in patients on RAAS blockers, diuretics, metformin and anticoagulants; and the role of the National Centre for Vector Borne Diseases Control (NCVBDC) programme.

Exam One-Liners

- Dengue in the elderly — more **expanded dengue syndrome**, less classical rash.
- Most dangerous dengue error in the elderly — **fluid overload**.
- Never give in dengue — **NSAIDs and aspirin**.
- Leptospirosis triad — **fever + calf myalgia + conjunctival suffusion**.
- Weil's disease — **jaundice + AKI + haemorrhage**.
- Drug of choice — **doxycycline** (mild), **penicillin G / ceftriaxone** (severe).
- Dengue vaccine in India — **TAK-003, ages 4–60 only**.

Question 13. Chikungunya — clinical presentation, differential diagnosis, laboratory tests and management 7 MARKS

ANSWER

Source note: Not covered in the uploaded textbooks. The geriatric principles (blunted febrile response, atypical presentation, narrowed reserve) are from Pathy's and Kane's; disease-specific content is from WHO, NCVBDC and current literature.

Textbook Core – 6 Points

1. **Aetiology.** Chikungunya virus is an **alphavirus (family Togaviridae)**, transmitted by the daytime-biting ***Aedes aegypti*** **and** ***Aedes albopictus*** mosquitoes. **Incubation 3–7 days** (range 1–12). The name is Makonde for "that which bends up", describing the stooped posture caused by joint pain. It is endemic and epidemic in India, with recurrent **post-monsoon outbreaks**, and a major global resurgence occurred in 2024–25.
2. **Acute clinical presentation.** Abrupt **high fever** with **severe, symmetrical, incapacitating polyarthralgia and arthritis** of the small joints of hands and wrists, ankles and feet; a maculopapular rash on the trunk and limbs in about half; headache, myalgia, conjunctivitis and profound fatigue. **In older adults, atypical and severe forms are far more common — encephalitis and encephalopathy, myocarditis, hepatitis, acute kidney injury, Guillain–Barré syndrome, bullous skin lesions**, and decompensation of pre-existing diabetes, heart failure and chronic kidney disease. **Advanced age is the principal risk factor for severe disease and death**: in the 2024–25 La Réunion outbreak, older adults accounted for almost half of all hospitalisations and nearly two-thirds of severe cases with organ failure.
3. **The chronic phase — the defining problem in geriatrics. Persistent or relapsing inflammatory arthralgia and arthritis lasting months to years occurs in up to 40–50% of patients**, and is commoner and more severe with increasing age. It closely resembles **seronegative rheumatoid arthritis**, and it is a major cause of lasting disability, immobility, deconditioning, depression and loss of independence — often more damaging to an older person than the acute illness.
4. **Differential diagnosis. Dengue** (thrombocytopenia, haemoconcentration, bleeding and shock; the arthralgia is less severe and does not persist) — **co-infection is common, and when in doubt manage as dengue, which is the more dangerous**; **Zika** (milder, prominent rash and conjunctivitis); **scrub typhus** (eschar, rapid response to doxycycline); **leptospirosis** (conjunctival suffusion, calf tenderness, jaundice with acute kidney injury); **malaria** and **enteric fever**. For the **chronic arthritis**, the differential becomes **elderly-onset rheumatoid arthritis, polymyalgia rheumatica, reactive arthritis and crystal arthropathy**.
5. **Laboratory tests — timing determines the test. RT-PCR is the test of choice in the first 5–7 days** (the viraemic phase); **IgM ELISA (MAC-ELISA) from about day 5 onwards**, with IgG appearing later and paired sera showing a four-fold rise. Supportive findings: **lymphopenia is characteristic** (in contrast to the thrombocytopenia of dengue), with mild thrombocytopenia, raised transaminases and raised CRP. **Always test simultaneously for dengue (NS1 antigen and IgM).** In the chronic phase, rheumatoid factor and anti-CCP are typically **negative**, which helps separate it from elderly-onset rheumatoid arthritis.
6. **Management. Entirely supportive — there is no specific antiviral.** Rest, adequate fluids and **paracetamol for fever and pain**. **Avoid NSAIDs and aspirin until dengue has been excluded** (risk of bleeding and acute kidney injury), and thereafter use them only with great caution in older adults. For **inflammatory arthritis persisting beyond three months**: a short course of **corticosteroids**, and in refractory cases **hydroxychloroquine or methotrexate** under rheumatology supervision. **Physiotherapy and graded exercise are essential** to prevent contractures, deconditioning and functional decline. Prevention is **vector control** — source reduction of domestic water containers, mosquito nets, repellents and full-sleeved clothing — remembering that ***Aedes*** **bites during the day**, so night-time bed nets alone are inadequate.

Latest Update (2023 – present)

7. Two chikungunya vaccines are now licensed: **IXCHIQ (VLA1553)**, a **live-attenuated** vaccine approved in 2023, and **VIMKUNYA**, a **virus-like-particle (non-live)** vaccine approved in 2025. Following reports of serious adverse events — including encephalitis and deaths — in vaccinees aged 62 to 89 years, the **US FDA suspended the Ixchiq licence in August 2025** and several countries restricted its use above age 60–65; the **EMA's PRAC concluded in July 2025 that the overall benefit–risk balance remained favourable and lifted the contraindication in those aged ≥65**, with updated product information. Because it is non-live, the **VLP vaccine (Vimkunya) is currently the preferred option in older adults**, and early post-authorisation data support its safety in over-65s. **Neither vaccine is yet available in India**, where **vector control and early supportive care remain the only strategy**.

Exam One-Liners

- Virus and vector — **alphavirus**, spread by ***Aedes*****, which bites by day**.
- Hallmark — **severe symmetrical polyarthralgia with stooped posture**.
- Blood picture — **lymphopenia** (dengue gives thrombocytopenia).
- Test in first 5 days — **RT-PCR**; after day 5 — **IgM ELISA**.
- Never give before excluding dengue — **NSAIDs and aspirin**.
- Chronic arthritis in up to **40–50%**; treat with steroids ± hydroxychloroquine/methotrexate.
- Preferred vaccine in older adults — **the non-live VLP vaccine**.

Question 14. Scrub typhus 7 MARKS

ANSWER

Source note: Not covered in the uploaded textbooks; content is drawn from current Indian and international literature and clearly separated from the geriatric principles taken from Pathy's and Kane's.

Textbook Core – 6 Points

1. **Aetiology and epidemiology.** ***Orientia tsutsugamushi***, an obligate intracellular Gram-negative bacterium, transmitted by the bite of the **larval stage (chigger) of trombiculid mites** (*Leptotrombidium* species) — which are both the vector and the reservoir. Endemic across the **"tsutsugamushi triangle"** of South and South-East Asia and the Pacific. India carries a very large burden, with **post-monsoon (August–November) outbreaks** in the sub-Himalayan belt, the north-east, Tamil Nadu, Kerala, Karnataka and Odisha, and it is **one of the commonest identifiable causes of acute undifferentiated febrile illness in India**.
2. **Clinical features.** Incubation **6–21 days**; then abrupt **fever with rigors, severe headache, myalgia** and a maculopapular rash. The pathognomonic sign is the **eschar** — a painless, black, necrotic, cigarette-burn-like lesion with an erythematous halo — which is found in only about **20–50% of Indian patients** and characteristically lies in **hidden, moist, covered sites**: axilla, groin, inframammary fold, perineum, waistline and behind the ears. **It must be actively hunted for by fully undressing and examining the patient**, which is the single highest-yield bedside act in this disease. Also **generalised lymphadenopathy and hepatosplenomegaly**.
3. **Complications — why it kills older adults. ARDS is the commonest severe complication**; also **acute kidney injury, myocarditis, meningoencephalitis, hepatitis with jaundice, septic shock, multi-organ dysfunction, haemophagocytic lymphohistiocytosis** and sensorineural hearing loss. **Older age, delayed treatment, jaundice, hypoalbuminaemia and ARDS predict death.** Untreated mortality reaches 30–70%, but falls to **under 5% with prompt doxycycline** — which is why empirical treatment matters so much.
4. **Investigations. IgM ELISA is the most widely used and practical confirmatory test in India**, becoming positive from about day 5–7. The **Weil–Felix (OX-K agglutination) test** is cheap and still used in peripheral settings but is **insensitive and non-specific**. The **indirect immunofluorescence assay (IFA) is the reference standard**. **PCR from eschar tissue or whole blood** is the most useful test in the **first week**, before antibodies appear. Supportive findings: **thrombocytopenia, raised transaminases, hypoalbuminaemia, raised CRP**, a normal or raised white cell count, and bilateral infiltrates on chest radiograph.
5. **Treatment. Doxycycline 100 mg twice daily for 7 days is the drug of choice**, and **defervescence within 48 hours is characteristic and supports the diagnosis**. Alternatives: **azithromycin 500 mg daily for 3–5 days** (preferred in pregnancy, in doxycycline intolerance and where resistance is suspected), with **intravenous doxycycline or azithromycin in severe disease**; chloramphenicol and rifampicin are further options. Supportive care for ARDS, acute kidney injury and shock determines outcome in severe cases. In older adults, doxycycline is safe and does not need renal adjustment, but must be taken **upright with water to avoid oesophagitis**, and it **interacts with warfarin** (raises INR) and is chelated by antacids, calcium and iron.
6. **Prevention, and the geriatric presentation. There is no vaccine.** Prevention is avoidance of chigger-infested scrub and long grass, protective and **permethrin-treated clothing**, DEET repellent, and clearing vegetation around dwellings. The geriatric point to make: in older adults the illness frequently presents **non-specifically — delirium, functional decline, falls, or unexplained ARDS or acute kidney injury without a recognised fever**. Therefore, **in the post-monsoon season in India, scrub typhus should be actively considered in any older patient with acute undifferentiated febrile illness or unexplained deterioration, an eschar hunted for, and empirical doxycycline started while awaiting serology**.

Latest Update (2023 – present)

7. The landmark Indian randomised controlled trial by **Varghese and colleagues (*New England Journal of Medicine*, 2023)** compared intravenous doxycycline, azithromycin, and the combination in **severe scrub typhus with organ dysfunction**, and showed that **combination intravenous doxycycline plus azithromycin was superior to either drug alone**, reducing the composite endpoint of death at day 7, persistent organ complications and persistent fever. **Combination therapy is now the preferred regimen for severe scrub typhus**, while oral doxycycline monotherapy remains standard for uncomplicated disease. **Empirical doxycycline for acute undifferentiated febrile illness during the post-monsoon season in endemic Indian districts** continues to be recommended practice.

Exam One-Liners

- Organism and vector — ***Orientia tsutsugamushi***, transmitted by the **chigger (larval trombiculid mite)**.
- Pathognomonic sign — **eschar**, present in only 20–50%, in **hidden moist sites**.
- Commonest severe complication — **ARDS**.
- Practical confirmatory test in India — **IgM ELISA**; reference standard — **IFA**; first week — **PCR**.
- Drug of choice — **doxycycline 100 mg BD × 7 days**; response within **48 hours**.
- Severe disease — **IV doxycycline PLUS azithromycin** (NEJM 2023).
- Prevention — **no vaccine**; permethrin-treated clothing and vegetation clearance.

Question 15. Cerebral malaria 6 MARKS

ANSWER

Textbook Core – 5 Points

1. **Definition.** A diffuse, potentially reversible **encephalopathy complicating *Plasmodium falciparum* infection**, defined strictly as **unrousable coma (Glasgow Coma Scale ≤11 in adults; Blantyre ≤2 in children) persisting for more than 30 minutes after a seizure**, in the presence of **asexual parasitaemia**, with **no other identifiable cause of coma**. It is the commonest lethal complication of falciparum malaria, with mortality of **15–20% even with optimal treatment**, and 10–20% of survivors have persistent neurological sequelae. It is rarely reported with *P. knowlesi* and *P. vivax*.
2. **Pathogenesis.** Parasitised red cells express **PfEMP-1** on their surface and **cytoadhere** to endothelial receptors — **ICAM-1, EPCR and CD36** — in cerebral microvessels, producing **sequestration and rosetting**. The result is **microvascular obstruction**, endothelial activation, cytokine release (particularly TNF-α), **blood–brain barrier breakdown**, cerebral oedema and reduced cerebral perfusion. Contributing systemically: **lactic acidosis, hypoglycaemia** and severe anaemia.
3. **Clinical features.** Fever, headache and progressive **impairment of consciousness to coma**; **generalised or focal seizures**; **symmetrical upper motor neurone signs** with decerebrate or decorticate posturing; and **malarial retinopathy** — retinal whitening, vessel discolouration, retinal haemorrhages and papilloedema, which is **the most specific bedside sign** and should be sought on fundoscopy. Characteristically there is **no meningism and no focal lateralising deficit**. Look for the other features of severe malaria: severe anaemia, jaundice, acute kidney injury, ARDS, hypoglycaemia, shock, spontaneous bleeding and DIC. **Older adults have a substantially higher risk of renal failure, pulmonary oedema and death.**
4. **Diagnosis. Peripheral blood smear** — thick film for detection, thin film for species identification and parasite counting (hyperparasitaemia >2–5% indicates severe malaria); repeat 12-hourly if initially negative. **Rapid diagnostic tests** detect HRP-2 (*P. falciparum*) and pLDH, but HRP-2 can remain positive for weeks after treatment. PCR where available. Essential adjuncts: **blood glucose — hypoglycaemia must be excluded and corrected in every case**, lactate, CBC, renal and liver function, coagulation screen and blood cultures. **Lumbar puncture is needed to exclude bacterial meningitis** unless contraindicated — in cerebral malaria the CSF is essentially normal or shows only a minimally raised protein. CT or MRI to exclude an alternative cause of coma.
5. **Treatment. Intravenous artesunate is the drug of choice — 2.4 mg/kg at 0, 12 and 24 hours, then once daily** — and is superior to quinine, having reduced mortality by about **35% in adults in the SEAQUAMAT trial**. Give parenteral therapy for at least 24 hours, then switch to a **full 3-day course of oral artemisinin-based combination therapy** once the patient can swallow. Supportive care is what determines survival: **correct hypoglycaemia with 25% dextrose**, treat seizures with a benzodiazepine followed by a maintenance anticonvulsant, maintain careful fluid balance avoiding both under-filling and pulmonary oedema, provide ventilation and dialysis as required, nurse the unconscious patient appropriately, and treat concurrent bacterial sepsis (common). **Do NOT give prophylactic phenobarbitone (it increased mortality), dexamethasone, routine mannitol, heparin or adrenaline** — all are ineffective or harmful. Watch for **post-artesunate delayed haemolysis** at 1–3 weeks, and check the haemoglobin at follow-up.

Latest Update (2023 – present)

6. The **WHO Guidelines for Malaria (2024–2025 updates)** reaffirm **intravenous artesunate for a minimum of 24 hours** as first-line treatment for all severe malaria, followed by a complete ACT course, and recommend **pre-referral rectal artesunate** where parenteral treatment cannot be started immediately — directly relevant to rural India. India's **National Framework for Malaria Elimination** and the NCVBDC national drug policy follow the same. The important current concern is that **artemisinin partial resistance (*Kelch13* mutations)**, long established in the Greater Mekong subregion, is now **confirmed in East Africa**, which makes a **full ACT course after parenteral therapy and follow-up parasite clearance monitoring mandatory** rather than optional.

Exam One-Liners

- Definition — **unrousable coma >30 min after a seizure with falciparum parasitaemia and no other cause**.
- Key adhesion molecule — **PfEMP-1**; receptors ICAM-1, EPCR, CD36.
- Most specific bedside sign — **malarial retinopathy**.
- Must exclude and correct in every case — **hypoglycaemia**.
- Drug of choice — **IV artesunate 2.4 mg/kg at 0, 12, 24 h then daily**.
- Never give — **steroids, prophylactic phenobarbitone, heparin, routine mannitol**.
- Late complication to check for — **post-artesunate delayed haemolysis**.

PART V

Urinary and Gastrointestinal Infection

Four separate urinary questions that are frequently confused with one another, arranged deliberately from colonisation through to recurrent infection, followed by antibiotic-associated colitis.

Question 16. Asymptomatic bacteriuria in the older adult 7 MARKS RECURRING

ANSWER

Textbook Core — 6 Points

1. **Definition. Significant bacteriuria without any signs or symptoms attributable to urinary infection**. Quantitatively: **≥10^5 CFU/mL** of the same organism in **two consecutive** clean-voided specimens in women, **one** specimen in men, or **≥10^2 CFU/mL** from a catheter specimen.
2. **Prevalence. Bacteriuria in older persons is common and usually asymptomatic.** Prevalence **rises with age** and roughly **parallels the prevalence of incontinence**; it is much higher in institutionalised residents and reaches essentially **100% in those with a chronic indwelling catheter**.
3. **Why it is so common in old age. Vaginal and urethral atrophy**, **obstructive uropathy** (prostatic hypertrophy in men), **"puddling" of urine related to bed rest**, incomplete bladder emptying, and **bladder catheterisation**. Prostatic hypertrophy specifically predisposes older men to urinary infection.
4. **Prognostic significance.** Although some studies suggested increased mortality, **when adjusted for age, the excess mortality is explained by the fatal diseases associated with bacteriuria, not by bacteriuria itself**. Antimicrobial therapy in hospitalised or institutionalised elderly has not been effective because of the **high rate of recurrence**. Eradicating bacteriuria does **not** improve chronic stable incontinence in nursing home residents.
5. **The core rule. In the absence of obstructive uropathy, there is no evidence to support routine antimicrobial therapy for asymptomatic bacteriuria in older persons.** Treatment causes adverse drug events, *Clostridioides difficile* infection, candidiasis and antimicrobial resistance, without benefit. **Do not send screening or surveillance urine cultures.**
6. **Recognised exceptions and cautions.** Treat before **urological procedures with anticipated mucosal bleeding** (e.g. TURP) and in pregnancy. The textbook additionally advises eradicating bacteriuria as part of the **initial assessment of new or worsening incontinence** in non-institutionalised patients. In the nursing home population, **new-onset incontinence, worsening incontinence, unexplained fever and decline in mental or functional status may be manifestations of a true urinary infection** — but other causes must be excluded first.

Latest Update (2023 – present)

7. **IDSA guidance**, reinforced by the AGS "Choosing Wisely" position and current stewardship practice, recommends **against screening for or treating asymptomatic bacteriuria in older adults — including those with delirium or falls** — provided there are **no localising urinary symptoms and no haemodynamic instability**; such patients should instead be assessed for an alternative cause and observed with hydration. **Diagnostic stewardship** — reflex culture only when the urinalysis is abnormal *and* the patient is symptomatic, and removal of "urine culture" from routine admission order sets — is now a core antimicrobial stewardship intervention and is embedded in the stewardship pillar of India's **NAP-AMR 2.0 (2025–2029)**.

Current Practice Update

Item	Textbook	Current recommendation	Exam approach
New/worsening incontinence	Eradicate bacteriuria as part of the initial assessment	Incontinence alone is not an indication to treat; look for another cause	State the textbook position, then give the current standard as your answer
Delirium or falls with bacteriuria	May be a manifestation of infection	Do not treat if no localising urinary symptoms and haemodynamically stable; evaluate other causes	Say "delirium alone does not equal UTI"
Screening cultures	Discouraged in catheterised patients	Discouraged in all older adults — diagnostic stewardship	Extend the principle beyond catheters

MNEMONIC ANCHOR

"No SYMPTOM → No CULTURE → No CURE." And for the only true exception: **"Only cut, then treat"** — treat before urological procedures with mucosal bleeding.

NUMERICAL ANCHORS

≥10^5 CFU/mL (voided) · ≥10^2 CFU/mL (catheter) · two specimens in women, one in men · ~100% prevalence with chronic indwelling catheter · prevalence parallels that of incontinence.

NAMED EXAMINER TRAPS

(a) Pyuria is not infection — most bacteriuric elderly have pyuria. **(b) Cloudy or foul-smelling urine is not an indication to treat** — it usually reflects concentration or diet. **(c)** Do not attribute delirium to bacteriuria until other causes are excluded. **(d)** Asymptomatic **candiduria** also does not need treatment — remove the catheter instead. **(e)** The exception list is short: **urological mucosal procedures and pregnancy** (not renal transplant beyond the early period, not diabetes, not spinal cord injury).

15-SECOND ANSWER SKELETON

Define with quantitative criteria → prevalence rises with age, ~100% with catheter → why (atrophy, obstruction, puddling, catheter) → no independent effect on mortality once age-adjusted → do not screen, do not treat → exceptions (urological procedure, pregnancy) → what to do instead (look for another cause, hydrate, review drugs) → stewardship framing.

EXTRA RECALL BOX

ASB versus UTI in the frail older adult — the practical rule is that you need **bacteriuria + a genuine localising symptom** (dysuria, new urgency/frequency, suprapubic or costovertebral angle pain, new gross haematuria) **or** systemic signs with no other explanation. In a resident who is delirious and bacteriuric, the commonest true culprits are **drugs, constipation, dehydration, pain, retention and hypoxia** — not the urine. Treating ASB is one of the most frequent sources of unnecessary antibiotic use in Indian hospitals and nursing homes.

Exam One-Liners

- Definition — bacteriuria **without attributable symptoms**.
- Treat or not — **do not treat**, except before urological mucosal procedures (and in pregnancy).
- Does it increase mortality — **no**, once adjusted for age and comorbidity.
- Does treatment help incontinence — **no** (chronic stable incontinence in nursing homes).
- Key stewardship message — **do not send the culture in the first place**.

Question 17. Catheter-associated urinary tract infection (CAUTI) 7 MARKS

ANSWER

Textbook Core — 6 Points

1. **Definition and the central rule.** CAUTI is a **symptomatic** urinary infection in a patient with an indwelling urethral catheter, or within 48 hours of its removal. **All patients with chronic indwelling catheters have bacteriuria**, which is frequently **polymicrobial** and whose organisms **change frequently** — therefore **bacteriuria alone is colonisation, not CAUTI**.
2. **Pathogenesis and risk factors. Duration of catheterisation** is the single most important risk factor. Bacteria ascend extraluminally along the catheter–urethral interface or intraluminally after breaks in the drainage system, and form a protective **biofilm** that antibiotics cannot penetrate. Other risks: female sex, diabetes, poor catheter care, and catheter obstruction. Indwelling catheterisation is **still overused in acute hospitals**.
3. **Accepted indications for an indwelling catheter (Kane). Urinary retention** causing persistent overflow incontinence, symptomatic infection or renal dysfunction that cannot be corrected surgically/medically or managed by intermittent catheterisation; **skin wounds or pressure ulcers being contaminated by urine**; care of the **terminally ill or severely impaired** in whom bed and clothing changes are distressing; and **patient preference** when toileting causes excessive discomfort. Incontinence alone and staff convenience are **not** indications.
4. **Presentation in the older adult.** Often **non-specific** — fever without localising signs, **new or worsening delirium**, functional decline, falls, anorexia, suprapubic or flank pain, or new haematuria. The **febrile response may be blunted or absent**. Symptoms are non-specific, so **other sources of infection must be carefully excluded before attributing them to the urinary tract**.
5. **Diagnosis. Do not perform surveillance cultures** to guide management — all chronically catheterised patients are bacteriuric. Send urine culture **only when symptomatic**, and ideally from a **freshly replaced catheter** (biofilm on the old catheter gives misleading organisms). **Pyuria does not diagnose CAUTI** in a catheterised patient. Blood cultures if systemically unwell; ultrasound if obstruction, stones or abscess suspected.
6. **Management and prevention — key principles of catheter care.** Treat **only if symptomatic**; **replace the catheter before starting antibiotics**; give culture-directed therapy for the **shortest effective duration**. Prevention: maintain a **sterile closed gravity-drainage system** and avoid breaking it; secure the catheter to thigh or lower abdomen; wash the entry site with soap and water once daily only; **do not routinely irrigate**; **do not use prophylactic or suppressive antiseptics or antimicrobials**; treat bypassing without obstruction as bladder spasm; and **remove the catheter at the earliest opportunity**.

Latest Update (2023 – present)

7. Practice has shifted to a bundled **"avoid – remove – maintain"** model: nurse-driven removal protocols, a documented **daily catheter necessity review**, and CAUTI as a **mandatory device-associated HAI surveillance indicator**. In India this is reinforced by the **National Action Plan on Antimicrobial Resistance 2.0 (NAP-AMR 2.0, 2025–2029, launched 18 November 2025)**, which makes **infection prevention and control programmes mandatory in all healthcare facilities**. Current IDSA/SHEA guidance continues to recommend **against treating catheter-associated asymptomatic bacteriuria** and **against antibiotic prophylaxis at routine catheter change**, and favours **intermittent catheterisation or external (condom) catheters** over indwelling catheters wherever feasible.

If this appears as a 15-mark question, add: microbiology (*E. coli*, *Klebsiella*, *Proteus* — urease-splitting, struvite stones and encrustation; *Pseudomonas*, *Enterococcus*, *Candida*; rising carbapenem resistance in Indian ICUs); complications (bladder stones, periurethral abscess, epididymo-orchitis, prostatitis, bacteraemia, pyelonephritis, and squamous-cell bladder cancer with very long-term use); antibiotic duration (7 days if prompt response, 10–14 days if delayed response or bacteraemia); catheter material evidence (no clear benefit of silver-alloy or antibiotic-impregnated catheters); and a formal CAUTI prevention bundle with audit indicators.

Exam One-Liners

- All chronic catheters are bacteriuric — **treat the patient, not the culture**.
- Biggest modifiable risk factor — **duration of catheterisation**.
- Never do — **surveillance cultures, routine irrigation, prophylactic antibiotics**.
- Always do — **replace catheter before starting antibiotics**; culture from the new catheter.
- Best prevention — **do not insert it; if inserted, remove it early**.

Question 18. Clinical presentations and diagnosis of acute pyelonephritis 7 MARKS

ANSWER

Textbook Core – 6 Points

1. **Definition and organisms.** Acute bacterial infection of the **renal pelvis and parenchyma**, usually arising by **ascending spread** from the bladder and, less often, haematogenously. Urinary infection is among the commonest infections of older people, and Pathy's emphasises that its pathogenesis is **strongly related to obstructive uropathy**. ***Escherichia coli*** predominates, but Kane's table shows that in older adults ***Proteus, Klebsiella, Enterobacter, Pseudomonas*** **and** ***Enterococcus*** are much more frequent than in the young — and in India, **ESBL-producing and carbapenem-resistant organisms** are now common.
2. **Predisposing factors in the elderly. Obstructive uropathy** — benign prostatic hyperplasia in men, pelvic organ prolapse and cystocele in women; **incomplete bladder emptying with a raised post-void residual**; **catheterisation and instrumentation**; stones; **diabetes**; vaginal and urethral atrophy; immobility and bed rest with urinary stasis; neurogenic bladder; and previous urological surgery. The **age-related fall in GFR of about 30%** and reduced renal reserve mean acute kidney injury occurs readily.
3. **Classic clinical presentation. Fever with rigors, loin or flank pain, costovertebral angle tenderness, nausea and vomiting**, with or without lower urinary tract symptoms (dysuria, frequency, urgency, suprapubic discomfort). **Gram-negative bacteraemia and septic shock may develop abruptly and be life-threatening** — a point Pathy's makes explicitly.
4. **Presentation in the older adult — the examinable half of the question.** It is frequently **atypical and non-localising**. **Delirium or acute confusion, new or worsening urinary incontinence, falls, functional decline, anorexia, generalised weakness and tachypnoea** may be the only features. **Fever is blunted or absent in a substantial proportion**, and Kane's warns that **hypothermia should be treated as sepsis until proved otherwise**; **the absence of leucocytosis does not exclude bacterial infection**. Loin pain and costovertebral tenderness are often absent. The necessary counterbalance: **do not attribute delirium to the urine unless there are localising urinary symptoms or systemic sepsis with no other explanation**, because **asymptomatic bacteriuria is very common and must not be treated**.
5. **Investigations. Urinalysis** — pyuria, nitrites, leucocyte esterase, and **white cell casts, which indicate renal parenchymal involvement**. **Urine culture with sensitivity must be sent before antibiotics in every case** of pyelonephritis (unlike simple cystitis). **Blood cultures** where there is fever, rigors, sepsis or immunosuppression. Also CBC, CRP, renal function and electrolytes, blood glucose, and **serum lactate** if unwell. **Imaging is not routine** but becomes **mandatory** if there is severe sepsis, **failure to defervesce within 48–72 hours**, known stones, a single kidney, diabetes, or suspected obstruction: **ultrasound first** (hydronephrosis, stones, abscess, post-void residual), then **contrast CT**, which is the investigation of choice for **emphysematous pyelonephritis, renal and perinephric abscess, and pyonephrosis**.
6. **Complications and differential diagnosis.** Complications: **bacteraemia and septic shock, acute kidney injury, renal and perinephric abscess, emphysematous pyelonephritis** (almost exclusively in diabetics — a urological emergency requiring drainage or nephrectomy), **pyonephrosis**, **papillary necrosis**, and chronic renal scarring. Differential diagnosis in an older patient: acute cholecystitis, appendicitis, diverticulitis, basal pneumonia, herpes zoster of the flank before the rash appears, renal colic, and **vertebral osteomyelitis or discitis** — all of which can present with fever and back or flank pain.

Latest Update (2023 – present)

7. Current guidance stresses **empirical therapy driven by the local antibiogram** rather than by textbook first choices, because resistance has outrun the traditional agents: in India, **fluoroquinolones and cotrimoxazole are no longer reliable empirical choices**, and **ICMR treatment guidelines** favour intravenous **amikacin**, a **beta-lactam/beta-lactamase inhibitor such as piperacillin-tazobactam**, or a **carbapenem for severe hospital-onset disease**, with **rapid de-escalation and switch to oral therapy once the patient has been afebrile for 48 hours**. Recommended durations have **shortened**: about **7 days** for fluoroquinolone-treated and **10–14 days** for beta-lactam-treated uncomplicated pyelonephritis. Newer agents — **ceftazidime-avibactam, ceftolozane-tazobactam and cefiderocol** — are reserved for confirmed carbapenem-resistant organisms under stewardship control.

Exam One-Liners

- Classic triad — **fever with rigors, loin pain, costovertebral angle tenderness**.
- In the elderly — **delirium, falls, incontinence and functional decline may be the only features**.
- Urinary sign of parenchymal involvement — **white cell casts**.
- Always send before antibiotics — **urine culture** (and blood cultures if septic).
- Imaging indication — **no defervescence in 48–72 hours**, suspected obstruction, stones, diabetes.
- Investigation of choice for complications — **contrast CT**.
- Diabetic emergency — **emphysematous pyelonephritis**.

Question 19. Management of recurrent urinary tract infection in an older female

7 MARKS

ANSWER

Textbook Core – 6 Points

1. **Definition, and the essential first step.** Recurrent UTI is **two culture-proven episodes in 6 months, or three in 12 months**. The first management step is to **confirm that each episode really was a symptomatic, culture-proven infection**. The commonest reason an older woman carries a label of "recurrent UTI" is **repeated treatment of asymptomatic bacteriuria** — which Kane's is explicit about: **bacteriuria in older persons is common and usually asymptomatic, and in the absence of obstructive uropathy there is no evidence to support routine antimicrobial therapy**. Review the actual culture and symptom records before doing anything else.
2. **Look for and correct the reversible cause. Genitourinary syndrome of menopause** — vaginal and urethral atrophy; **incomplete bladder emptying** — measure the **post-void residual**; **pelvic organ prolapse and cystocele**; **constipation and faecal incontinence**; **inadequate fluid intake**; **diabetes with glycosuria**; **stones or obstruction**; **catheter use**; and **immobility or cognitive impairment causing difficulty in toileting**. Examine the perineum, perform a pelvic examination, and assess the functional and cognitive ability to reach and use the toilet — this last is a genuinely geriatric point that is often the actual cause.
3. **Investigations. Urine culture with sensitivity for each episode** — both to direct therapy and to track resistance; blood glucose and HbA1c; renal function; **post-void residual by bladder scan**; and **ultrasound of kidneys, ureters and bladder**. Referral for **cystoscopy and further imaging is indicated only if** there is haematuria, a suspected stone or obstruction, a suspected fistula, persistent infection with the same organism, or failure of conservative management — **routine cystoscopy in every older woman is not indicated**.
4. **Behavioural and fluid measures — first line. Increase fluid intake to about 1.5–2 L per day if current intake is below 1.5 L** (proven in a randomised trial); **timed, complete and double voiding**; treat constipation; perineal hygiene; avoid spermicides; and **avoid unnecessary catheterisation**. If there is a significant post-void residual, manage it with bladder retraining or **clean intermittent catheterisation rather than an indwelling catheter**.
5. **Vaginal oestrogen — the single most important intervention in a postmenopausal woman.** Low-dose **vaginal oestrogen cream, pessary or ring** restores the vaginal epithelium, lowers vaginal pH and re-establishes lactobacilli, significantly reducing recurrence. **Systemic absorption is negligible**, so age is not a barrier, and it can generally be used in women with a history of breast cancer after discussion with the oncologist. **Systemic hormone replacement therapy does NOT work for this indication** — a favourite examiner distinction.
6. **Antiseptic and antibiotic prophylaxis — only after the above. Methenamine hippurate 1 g twice daily** is a urinary antiseptic that is **not an antibiotic** and was shown **non-inferior to daily antibiotic prophylaxis in the ALTAR randomised trial** — it is now the preferred first prophylactic step. **Cranberry products and D-mannose** may be offered (weak evidence). If antibiotic prophylaxis is still required: **low-dose continuous prophylaxis for 3–6 months** — **nitrofurantoin 50–100 mg nightly** (**avoid if eGFR <45**, avoid prolonged use because of pulmonary fibrosis and hepatotoxicity, and note it is on the **Beers list** in renal impairment), **trimethoprim 100 mg nightly**, or **cefalexin 125–250 mg nightly** — or **post-coital prophylaxis** where episodes relate to intercourse. **Self-start standby therapy** with a culture-directed agent is useful in a reliable, cognitively intact woman. **Never perform a test-of-cure culture in an asymptomatic patient.**

Latest Update (2023 – present)

7. The **AUA/CUA/SUFU 2025 amendment** to the recurrent uncomplicated UTI guideline substantially **expanded the non-antibiotic options**: clinicians **may now offer cranberry, methenamine hippurate, and increased water intake (where intake is below 1.5 L/day)** for prophylaxis; **vaginal oestrogen remains a "should recommend" for peri- and postmenopausal women**; and a new recommendation directs clinicians to **evaluate for an alternative cause of symptoms in women whose symptoms persist after microbiological cure**. **NICE (2024)** similarly now recommends **methenamine hippurate as an alternative to daily antibiotic prophylaxis** where behavioural measures and vaginal oestrogen have not been sufficient. This shift is a direct antimicrobial stewardship gain in a population that accounts for a disproportionate share of community antibiotic prescribing.

> **THE STEPWISE ANSWER — WRITE IT AS A LADDER**
>
> **Step 1** Confirm the episodes were true symptomatic culture-proven UTIs, not treated asymptomatic bacteriuria → **Step 2** Find and correct the reversible cause (atrophy, residual urine, prolapse, constipation, diabetes, catheter, toileting ability) → **Step 3** Behavioural measures and fluid intake → **Step 4 Vaginal oestrogen** → **Step 5 Methenamine hippurate** (± cranberry, D-mannose) → **Step 6** Low-dose antibiotic or post-coital prophylaxis, or self-start standby therapy → **Step 7** Refer for cystoscopy/imaging only on specific indications.

Exam One-Liners

- Definition — **2 episodes in 6 months or 3 in 12 months**, culture-proven.
- Commonest reason for the label — **repeated treatment of asymptomatic bacteriuria**.
- Single most effective intervention — **vaginal oestrogen** (systemic HRT does not work).
- Non-antibiotic prophylaxis of choice — **methenamine hippurate 1 g BD** (ALTAR trial).
- Fluids — **increase to 1.5–2 L/day if intake is below 1.5 L**.

- Nitrofurantoin — **avoid if eGFR <45**; avoid long-term.
- Never do — **test-of-cure culture in an asymptomatic woman**.

Question 20. Diagnosis and management of *Clostridioides difficile* 7 MARKS

ANSWER

Textbook Core – 6 Points

1. **Definition and risk factors.** *Clostridioides difficile* is a **Gram-positive, spore-forming, toxin-producing anaerobe** causing **antibiotic-associated diarrhoea and pseudomembranous colitis** through disruption of the colonic microbiota and elaboration of **toxins A and B**. Risk factors: **antibiotics** — especially clindamycin, fluoroquinolones, third-generation cephalosporins, carbapenems and broad-spectrum penicillins — **age 65 and over**, hospitalisation and long-term care residence, **proton pump inhibitors**, previous infection, enteral tube feeding, abdominal surgery, chemotherapy and comorbidity. **Older adults are the highest-risk group and have the highest mortality and the highest recurrence rate.**
2. **Clinical spectrum. Asymptomatic colonisation** is common in long-term care residents — **do not test and do not treat**. **Mild-to-moderate infection**: watery diarrhoea (three or more unformed stools in 24 hours), cramping abdominal pain, low-grade fever and leucocytosis. **Severe infection**: white cell count **≥15,000/mm³** or serum creatinine **>1.5 mg/dL**. **Fulminant infection**: hypotension or shock, ileus, or **toxic megacolon** — which may present with a **paradoxical reduction in diarrhoea** as the colon becomes atonic, a dangerous trap. Kane's advises considering *C. difficile* in any long-term care resident with fever, abdominal cramps and diarrhoea with or without blood or white cells in the stool.
3. **Diagnosis. Test only unformed, diarrhoeal stool from a symptomatic patient.** Do not test formed stool, do not repeat within 7 days, and **never perform a test of cure** — the assays stay positive for weeks. Use a **two-step algorithm**: a sensitive screening test — **glutamate dehydrogenase (GDH) antigen** or a **NAAT/PCR for the toxin gene** — followed by a specific confirmatory **toxin A/B enzyme immunoassay**. GDH-positive with toxin-positive confirms infection; both negative excludes it; a **discordant NAAT-positive but toxin-negative result may represent colonisation** and needs clinical judgement. Adjuncts: CBC, creatinine, albumin, lactate; **abdominal radiograph or CT** if fulminant disease is suspected; and **flexible sigmoidoscopy showing pseudomembranes** when the diagnosis remains uncertain.
4. **General measures. Stop the precipitating antibiotic** wherever possible, or narrow it; **stop the proton pump inhibitor** if it has no indication; **avoid antimotility agents** (loperamide, opiates) in severe disease; correct fluid and electrolyte losses. Infection control: **single room with contact precautions, gloves and gowns**; **hand washing with soap and water — alcohol hand rub does not kill spores**; and environmental cleaning with a **sporicidal, chlorine-based agent**. These are the highest-yield infection-control points for a geriatrics paper.
5. **Treatment of the initial episode. Oral fidaxomicin 200 mg twice daily for 10 days is now preferred**, because it produces markedly lower recurrence; **oral vancomycin 125 mg four times daily for 10 days** is an acceptable alternative and remains the pragmatic first choice in India where cost is limiting. **Metronidazole is no longer first-line** and is reserved for mild disease where neither agent is available, or as adjunctive intravenous therapy in fulminant disease. **Fulminant infection**: **oral or nasogastric vancomycin 500 mg four times daily plus intravenous metronidazole 500 mg eight-hourly**, with **vancomycin retention enemas if there is ileus**, and **early surgical consultation** for subtotal colectomy or diverting loop ileostomy with colonic lavage.
6. **Recurrence — the dominant problem in older adults.** Recurrence occurs in **20–25% after a first episode and 40–60% after subsequent episodes**. **First recurrence**: fidaxomicin (including the extended-pulsed regimen) if vancomycin was used first, or a **tapering and pulsed vancomycin** course. **Second and later recurrences**: tapered/pulsed vancomycin, **vancomycin followed by rifaximin**, **bezlotoxumab** — a single-infusion monoclonal antibody against toxin B, used as an adjunct but with caution in heart failure — or **faecal microbiota transplantation**, which achieves cure rates above **85%**.

Latest Update (2023 – present)

7. The therapeutic landscape has changed with the approval of two **standardised live biotherapeutic microbiome products** for the prevention of recurrent infection — **faecal microbiota, live-jslm (Rebyota)**, given rectally, and **faecal microbiota spores, live-brpk (Vowst)**, given as oral capsules — which provide regulated, quality-controlled, donor-screened alternatives to conventional faecal microbiota transplantation. These followed the **2021 IDSA/SHEA focused update**, which established **fidaxomicin in preference to vancomycin** for both initial and recurrent episodes and introduced **bezlotoxumab** as an adjunct for patients with an episode in the preceding six months. A **2024 Emerging Infections Network survey** found uptake of fidaxomicin still limited by cost, so **oral vancomycin remains the realistic first-line agent in most Indian hospitals**, with fidaxomicin reserved for those at highest risk of recurrence.

Exam One-Liners

- Test only **diarrhoeal stool from a symptomatic patient**; never a test of cure.
- Algorithm — **GDH or NAAT screen, then toxin A/B EIA confirm**.
- Severe disease — **WBC ≥15,000 or creatinine >1.5 mg/dL**.
- Fulminant — **hypotension, ileus or toxic megacolon**; diarrhoea may paradoxically stop.
- First-line — **fidaxomicin 200 mg BD × 10 days**; alternative **vancomycin 125 mg QID × 10 days**.
- Metronidazole — **no longer first-line**.
- Hand hygiene — **soap and water; alcohol gel does not kill spores**.

- Multiply recurrent disease — **FMT, cure rate >85%**.

PART VI

Rheumatology and Systemic Vasculitis

The inflammatory diseases of later life, ordered so that the polymyalgia–giant cell arteritis axis is read before the wider vasculitides, and the inflammatory arthritides together.

Question 21. Polymyalgia rheumatica 7 MARKS RECURRING

ANSWER

Textbook Core — 6 Points

1. **Definition.** An inflammatory condition of older adults — essentially never below **age 50** — producing **pain and morning stiffness of the shoulder and pelvic girdles** with a systemic inflammatory response. It causes **painful muscles with proximal myopathy** and is an important, **completely reversible** cause of frailty that is **often misdiagnosed by clinicians**.
2. **Clinical features.** Bilateral, symmetrical aching of shoulders, neck and hips; **morning stiffness >45 minutes**; difficulty rising from a chair, turning in bed or combing the hair. Constitutional features — **weight loss, low-grade fever, fatigue, anorexia and depression**. Weakness is **pain-limited**, with preserved true power.
3. **Investigations. Raised ESR and CRP** (ESR often >40 mm/h) — the diagnosis is confirmed by finding an elevated ESR in the right clinical setting. Normocytic normochromic anaemia, raised alkaline phosphatase, thrombocytosis. **Normal creatine kinase** — the key discriminator from inflammatory myopathy. RF and anti-CCP negative. Ultrasound shows **subacromial–subdeltoid bursitis, biceps tenosynovitis and trochanteric bursitis**.
4. **Differential diagnosis. Elderly-onset rheumatoid arthritis**, inflammatory myopathy (polymyositis — CK raised), **hypothyroidism**, cervical spondylosis and shoulder osteoarthritis, **multiple myeloma** and occult malignancy, statin-induced myopathy, RS3PE, and other endocrine disorders of insidious onset (hyperthyroidism, hypoadrenalism).
5. **Relationship with giant cell arteritis.** There is a **close association between PMR and temporal arteritis**: about **16–21% of PMR patients develop GCA**, and 40–50% of GCA patients have PMR symptoms. **Any symptom suggesting temporal artery involvement — headache, jaw claudication, scalp tenderness or a recent change in vision — especially with a very high ESR (>75 mm/h), should prompt consideration of temporal artery biopsy. Acute treatment of temporal arteritis with high-dose steroids is needed to prevent blindness.**
6. **Treatment. Prednisolone 12.5–25 mg daily** produces a **dramatic response within 24–72 hours** — a poor response should make you doubt the diagnosis. Taper slowly over 1–2 years. **Methotrexate** is used as a **corticosteroid-sparing agent** for frequent relapses or steroid toxicity; **infliximab has NOT been shown to be beneficial and is not recommended**. Mandatory alongside steroids: **calcium, vitamin D and a bisphosphonate**, glucose and blood-pressure monitoring, gastroprotection, and a steroid alert card.

Latest Update (2023 – present)

7. The **2025/2026 EULAR recommendations for PMR and primary large-vessel vasculitis** now name **sarilumab (IL-6 receptor blocker, 200 mg subcutaneously every 2 weeks)** as the **preferred add-on for relapsing or refractory PMR**, with tocilizumab as an alternative and methotrexate retained as a cheaper option — a glucocorticoid taper as rapid as **14 weeks** is possible on sarilumab. For GCA, suspected cases must be **referred within 24 hours**, and adjunctive **tocilizumab or upadacitinib 15 mg once daily** is recommended for refractory/relapsing disease or where the risk of steroid toxicity is high (upadacitinib FDA-approved April 2025 on the **SELECT-GCA** trial: sustained remission **46% vs 29%** with placebo, with roughly halved cumulative steroid exposure).

Current Practice Update

Item	Textbook	Current recommendation	Exam approach
Steroid-sparing agent	Methotrexate; infliximab not beneficial	Sarilumab preferred for relapsing PMR; tocilizumab alternative; methotrexate still valid	Give steroids as first-line, then cite EULAR 2025 for refractory disease
GCA treatment	High-dose steroids to prevent blindness	Steroids immediately, plus tocilizumab or upadacitinib as steroid-sparing adjunct	Never omit "start steroids before biopsy"
Referral	Consider temporal artery biopsy	Suspected GCA — specialist referral within 24 hours; ultrasound "halo sign" as first-line imaging	Frame GCA as a medical emergency

MNEMONIC ANCHOR

"PMR = Pain (girdle), Morning stiffness, Rapid steroid Response." If any one of the three is missing — **rethink the diagnosis**.

NUMERICAL ANCHORS

Age >50 always · morning stiffness >45 min · ESR often >40 mm/h (GCA suspicion if >75 mm/h) · prednisolone **12.5–25 mg/day** · response within 24–72 h · GCA in 16–21% of PMR · GCA steroid dose 40–60 mg (higher / IV if visual loss) · taper over 1–2 years · sarilumab 200 mg SC 2-weekly · upadacitinib 15 mg OD.

NAMED EXAMINER TRAPS

(a) CK is normal in PMR — a raised CK means myositis, not PMR. **(b)** Failure to respond within a week means the diagnosis is wrong — think elderly-onset RA, myeloma or malignancy. **(c) Never delay steroids awaiting temporal artery biopsy** — the biopsy stays positive for up to 2 weeks. **(d)** Start **bone protection with the very first steroid prescription**, not later. **(e)** A normal ESR does not fully exclude GCA — check CRP too. **(f)** Do not use starting prednisolone above 25 mg for uncomplicated PMR; a high requirement suggests GCA.

15-SECOND ANSWER SKELETON

Define (age >50, girdle pain and stiffness) → clinical features with constitutional symptoms → ESR/CRP up, CK normal, ultrasound bursitis → differential (elderly-onset RA, myositis, hypothyroidism, myeloma) → GCA association 16–21% and the red-flag symptoms → prednisolone 12.5–25 mg with dramatic response → methotrexate for relapse → bone protection → EULAR 2025 sarilumab / upadacitinib.

EXTRA RECALL BOX

PMR as a reversible cause of frailty is a specifically geriatric examination point: an older person with weight loss, immobility, weakness and depression who is labelled "failure to thrive" may have PMR, and **treatment with corticosteroids reverses the frailty completely**. Always check an ESR in unexplained functional decline in the elderly. Conversely, remember that **corticosteroids themselves** cause osteoporosis, diabetes, hypertension, cataract, skin fragility, myopathy, delirium and infection — in older adults the steroid course frequently causes more morbidity than the PMR.

Exam One-Liners

- Never occurs below — **age 50**.
- Key negative test — **normal CK**.
- Starting dose — **prednisolone 12.5–25 mg/day**; GCA **40–60 mg**.
- Diagnostic response — within **24–72 hours**.
- Not recommended — **infliximab**.
- New preferred biologic for relapsing PMR — **sarilumab**.

Question 22. Systemic vasculitides in the elderly 15 MARKS

ANSWER

Textbook Core — 10 Points

1. **Definition and classification.** Inflammation of blood-vessel walls causing ischaemia, necrosis and organ damage. The **Chapel Hill Consensus** classifies by vessel size: **large-vessel** (giant cell arteritis, Takayasu arteritis); **medium-vessel** (polyarteritis nodosa, Kawasaki disease); **small-vessel ANCA-associated** (granulomatosis with polyangiitis, **microscopic polyangiitis**, eosinophilic granulomatosis with polyangiitis); and **small-vessel immune-complex** (IgA vasculitis, cryoglobulinaemic, anti-GBM disease).
2. **Which vasculitides matter in old age. Giant cell arteritis (GCA) is the commonest primary systemic vasculitis in adults** and occurs **almost exclusively over the age of 50**, with peak incidence in the seventies. **ANCA-associated vasculitis — especially microscopic polyangiitis — peaks at 65–75 years.** Drug-induced and malignancy-associated (paraneoplastic) vasculitis are proportionally commoner in the elderly.
3. **GCA — clinical features.** New-onset **temporal headache**, **scalp tenderness**, **jaw claudication** (the most specific symptom), **amaurosis fugax or sudden painless visual loss** (anterior ischaemic optic neuropathy), a tender, thickened or pulseless temporal artery, constitutional symptoms, and overlap with polymyalgia rheumatica. **Large-vessel GCA** causes aortitis, thoracic aortic aneurysm, limb claudication and unequal pulses. GCA should be considered in any older patient with cerebrovascular or vertebrobasilar symptoms.
4. **GCA — investigation and treatment. Markedly raised ESR (often >75 mm/h) and CRP**, normochromic anaemia, thrombocytosis, raised alkaline phosphatase. **Temporal artery biopsy** — take a **1–2 cm segment** because of **skip lesions**; or **temporal artery ultrasound** showing the **"halo sign"** and a positive compression sign; CT/MR angiography or PET-CT for large-vessel disease. **Start prednisolone 40–60 mg daily immediately — IV methylprednisolone if there is visual loss — without waiting for the biopsy**, which remains positive for up to 2 weeks. Add low-dose aspirin, bone protection and gastroprotection.
5. **ANCA-associated vasculitis — clinical features.** A subacute constitutional illness (fever, weight loss, malaise, arthralgia) evolving into a **pulmonary–renal syndrome**. **GPA** — upper airway disease (epistaxis, nasal crusting, sinusitis, saddle-nose deformity, subglottic stenosis), lung nodules and cavities, and rapidly progressive glomerulonephritis. **MPA** — RPGN and alveolar haemorrhage **without** granulomas. **EGPA** — late-onset asthma, eosinophilia, **mononeuritis multiplex** and cardiac involvement.
6. **ANCA — investigations. c-ANCA / anti-PR3** (GPA) and **p-ANCA / anti-MPO** (MPA, EGPA). **Urinalysis and microscopy showing dysmorphic red cells and red-cell casts is the single most important bedside test** — never omit it. Rising creatinine, CXR and HRCT, ENT and ophthalmic assessment, nerve conduction studies, and **renal biopsy** showing **pauci-immune necrotising crescentic glomerulonephritis**.
7. **Treatment of ANCA-associated vasculitis. Remission induction** — glucocorticoids plus **rituximab or cyclophosphamide**; **rituximab is preferred in older adults and in relapsing disease** because it avoids cyclophosphamide's bladder toxicity, infertility and malignancy risk. **Plasma exchange** is reserved for severe alveolar haemorrhage and the most severe renal disease. **Maintenance** — rituximab or azathioprine (or methotrexate/mycophenolate) for 2–4 years. **All doses of cyclophosphamide and rituximab must be reduced for age and renal function.**
8. **Mimics and differential diagnosis in the elderly. Infective endocarditis** (fever, splinter haemorrhages, glomerulonephritis, positive ANCA in some), atrial myxoma, **cholesterol embolisation** (livedo reticularis, blue toes, eosinophilia and AKI after an arterial procedure), antiphospholipid syndrome, **malignancy** (myeloma, lymphoma, myelodysplasia), and **drug-induced vasculitis** (hydralazine, propylthiouracil, minocycline, levamisole-adulterated cocaine — typically very high-titre MPO-ANCA). Chronic infections — **tuberculosis, hepatitis B and C, HIV** — must be excluded before immunosuppression, particularly in India.
9. **Geriatric-specific issues.** Presentation is frequently **non-specific** — pyrexia of unknown origin, weight loss, anaemia, raised inflammatory markers, delirium or unexplained functional decline. **Treatment toxicity, not the vasculitis, is the main cause of death in older patients — infection is a commoner cause of death than active disease.** Therefore: **PJP prophylaxis with co-trimoxazole**, bone protection, glucose and blood-pressure monitoring, gastroprotection, **hepatitis B and TB screening**, and vaccination before rituximab are all mandatory.
10. **Monitoring and prognosis.** Follow CRP, creatinine, **urinalysis** and ANCA titre (an imperfect guide to activity); score activity with the **Birmingham Vasculitis Activity Score (BVAS)** and accumulated damage with the **Vasculitis Damage Index (VDI)**. **Increasing age predicts worse renal and overall survival**; relapse is frequent after treatment withdrawal, and end-stage renal disease is the major long-term morbidity in MPA.

Latest Updates (2023 – present)

11. **GCA — upadacitinib.** The phase 3 **SELECT-GCA** trial (*NEJM* 2025) showed that **upadacitinib 15 mg once daily** with a 26-week steroid taper achieved **sustained remission in 46.4% versus 29.0%** with placebo plus a 52-week taper, with roughly **halved cumulative glucocorticoid exposure**; FDA approval followed in **April 2025**, making it the second approved drug for GCA after tocilizumab. The **2025/2026 EULAR recommendations** now require **referral of suspected GCA within 24 hours** and advise adding **tocilizumab or upadacitinib** in refractory or relapsing disease or where steroid toxicity risk is high.
12. **ANCA-associated vasculitis — steroid minimisation. Avacopan**, an oral **C5a receptor antagonist**, is approved

as an adjunct in severe AAV on the basis of the **ADVOCATE** trial, allowing substantial glucocorticoid reduction with better renal recovery; and the **PEXIVAS**-informed **reduced-dose glucocorticoid regimen** (with restriction of plasma exchange) is now standard. Both are especially valuable in older patients, in whom **steroid toxicity and infection drive mortality**.

Current Practice Update

Item	Textbook	Current recommendation	Exam approach
GCA diagnosis	Temporal artery biopsy	Ultrasound halo sign as first-line imaging; biopsy or imaging, both acceptable; fast-track clinics	Mention both; never delay steroids for either
GCA steroid-sparing	Steroids alone ± methotrexate	Tocilizumab or upadacitinib 15 mg OD as adjunct	Quote SELECT-GCA figures (46% vs 29%)
AAV induction	Steroids + cyclophosphamide	Steroids + rituximab preferred in the elderly; reduced-dose steroid regimen; avacopan adjunct	Emphasise steroid minimisation in older adults
Plasma exchange	Used in severe disease	Restricted to severe alveolar haemorrhage and most severe renal disease (PEXIVAS)	Say "restricted, not routine"

Exam One-Liners

- Commonest primary systemic vasculitis in adults — **giant cell arteritis**.
- Most specific GCA symptom — **jaw claudication**.
- Ultrasound sign — **halo sign**; biopsy problem — **skip lesions**.
- Steroids in GCA — **start before the biopsy**; biopsy positive up to 2 weeks.
- Vasculitis of the 65–75 age group — **microscopic polyangiitis**.
- Single most important bedside test in suspected AAV — **urinalysis with microscopy**.
- Renal biopsy in AAV — **pauci-immune crescentic glomerulonephritis**.
- Induction agent preferred in the elderly — **rituximab**.
- Commonest cause of death in the older vasculitis patient — **infection from treatment**.

Question 23. Late / elderly-onset rheumatoid arthritis — how it differs from young-onset disease, and the relevant investigations 15 MARKS RECURRING

ANSWER

Source note. Late-onset rheumatoid arthritis is **not covered adequately in the uploaded textbooks**. Kane's discusses arthritis in older adults generally and provides the **osteoarthritis versus inflammatory arthritis** comparison table (used below); Pathy's covers polymyalgia rheumatica and general prescribing in old age. The disease-specific comparative content is drawn from current rheumatological literature and guidelines and is clearly separated.

Textbook Core — 10 Points

1. **Definitions and the age threshold. Late-onset rheumatoid arthritis (LORA)**, also called **elderly-onset RA (EORA)**, is rheumatoid arthritis whose **onset occurs at or after 60 years** (the WHO definition of "elderly" being 60–74), although many studies use **65 years**; **young-onset RA (YORA)** is onset before this. There is **no agreed international threshold** — the **Japan College of Rheumatology**, which has issued the **only national guideline** on the subject, recommends the term **LORA**. Roughly **one-third of all new RA now begins after the age of 60**, and this proportion is rising as the population ages.
2. **Epidemiology and sex ratio.** YORA is strongly **female-predominant, about 3:1**, with peak onset between 25 and 45 years. In LORA the female preponderance is **much less marked and approaches 1:1**. The **incidence of RA continues to rise with age** into the seventh and eighth decades, so RA is not a disease that "burns out" at the door of old age.
3. **Pathogenesis — why LORA is different.** LORA arises on a background of **immunosenescence and inflammaging**: thymic involution, expansion of **$CD4^+CD28^-$ senescent T cells**, shortened lymphocyte telomeres, and a high baseline **IL-6** level. Importantly, the **HLA-DRB1 shared-epitope association is weaker in LORA than in YORA** — which is consistent with the higher proportion of seronegative disease and with the clinical overlap with polymyalgia rheumatica.
4. **Mode of onset and joint pattern — the core clinical difference. YORA** characteristically has an **insidious onset** over weeks to months, producing a **symmetrical small-joint polyarthritis of the MCP, PIP, wrist and MTP joints, sparing the DIP joints**. **LORA** tends to have an **abrupt, almost "infection-like" acute onset**, is more often **oligoarticular at presentation**, and shows **more frequent involvement of large, proximal joints — especially the shoulders** — producing a girdle distribution that closely mimics polymyalgia rheumatica. **Pitting oedema of the hands and feet** is characteristic of LORA and rare in YORA.
5. **Systemic and constitutional features. Fever, weight loss, profound fatigue, anorexia and a markedly raised ESR at onset** are all **more prominent in LORA**. This may relate to differences in cytokine profile and to the higher IL-6 tone of ageing. Classical severe extra-articular rheumatoid disease (vasculitis, Felty's syndrome, rheumatoid lung) is **not** more frequent in LORA — the systemic picture is one of constitutional inflammation rather than organ-based rheumatoid complications. Because the presentation is dominated by weight loss, weakness, immobility and low mood, LORA is frequently mislabelled as **"failure to thrive"** or as an occult malignancy.
6. **Serology — and two crucial caveats. LORA is more often seronegative** for rheumatoid factor and anti-CCP than YORA. Caveat one: **rheumatoid factor positivity increases with age in the healthy population** as an expression of age-related immune dysregulation, and therefore **loses diagnostic specificity in older adults** — **anti-CCP is the more useful test in this age group**. Caveat two: **seronegative RA is not a mild form of the disease** — it may carry more inflammatory activity and, in some series, a worse prognosis, and it requires the **same intensive treat-to-target strategy** as seropositive disease.
7. **The three phenotypes of LORA, and the differential diagnosis.** Three recognised subsets: **(i) classical RA-like** — usually seropositive, erosive, and behaves much like YORA; **(ii) PMR-like LORA** — girdle pain and stiffness with a very high ESR, usually seronegative, occurring in about **25% of LORA** (PMR-like YORA is exceptional); **(iii) RS3PE-like** — remitting seronegative symmetrical synovitis with pitting oedema. The critical differential is therefore **polymyalgia rheumatica**: in follow-up studies **more than 20% of patients initially diagnosed as PMR are reclassified as seronegative LORA**, and some authorities regard PMR, seronegative LORA and RS3PE as parts of a single spectrum of inflammatory disease of old age. Other differentials at this age: **polyarticular gout and calcium pyrophosphate disease**, **nodal and erosive osteoarthritis** of the hands, **paraneoplastic polyarthritis**, viral arthritis, and **immune checkpoint inhibitor-induced arthritis**. Kane's comparison of **osteoarthritis versus inflammatory arthritis** is the bedside discriminator to quote:

Clinical feature	Osteoarthritis	Inflammatory arthritis
Duration of stiffness	Minutes	**Hours**
Pain	Usually with activity	**Occurs even at rest and at night**
Fatigue	Unusual	**Common**
Swelling	Common but little synovial reaction	**Very common, with synovial proliferation and thickening**
Erythema and warmth	Unusual	**Common**

8. **Disease course and outcome.** LORA presents with **higher disease activity, more extra-articular manifestations, greater disability, more radiographic damage and a higher ESR at onset** than YORA. Yet **with adequate treatment the probability of achieving remission or low disease activity is similar to — and in some series better than — YORA**, partly because of the higher proportion of seronegative patients. Where outcomes are worse in practice, the reason is largely **under-treatment**: older patients receive **higher glucocorticoid and NSAID doses, fewer DMARDs and lower DMARD doses**, and discontinue treatment more often for adverse events. The correct conclusion is that **LORA is under-treated rather than intrinsically treatment-resistant**.
9. **Investigations to send — the second half of the question. (a) To confirm the diagnosis: rheumatoid factor** and **anti-CCP antibody** (anti-CCP being the more specific in this age group); **ANA**; **ESR and CRP**; **complete blood count** (normocytic anaemia of chronic disease, thrombocytosis; leucopenia would suggest Felty's syndrome); **plain radiographs of hands, wrists and feet** for periarticular osteopenia, marginal erosions, joint space narrowing and deformity — remembering that **coexisting osteoarthritic change is almost universal at this age** and must not be over-interpreted; and **musculoskeletal ultrasound with power Doppler, or MRI of hands and shoulders**, which detects synovitis and erosions earlier and helps **distinguish the synovitis of LORA from the bursitis and tenosynovitis pattern of PMR**. Classification uses the **ACR/EULAR 2010 criteria (score ≥6 of 10)**, which are **less sensitive in seronegative large-joint LORA** and therefore support but never replace clinical judgement. **(b) To exclude the differential diagnoses: serum uric acid** and, decisively, **joint aspiration with polarised light microscopy and culture** for crystals and sepsis; **TSH**; **calcium, phosphate, alkaline phosphatase and 25(OH) vitamin D**; **serum and urine protein electrophoresis with free light chains** to exclude myeloma; **creatine kinase**; **chest radiograph**; **temporal artery ultrasound** if there are any giant cell arteritis features; and **age-appropriate malignancy screening** where a paraneoplastic arthritis is suspected. **(c) Baseline safety and pre-treatment work-up: renal function with eGFR**, **liver function tests**, **hepatitis B and C and HIV serology**, **latent tuberculosis screening with a chest radiograph and Mantoux or IGRA — mandatory in India before any biologic or JAK inhibitor**, **lipid profile with cardiovascular risk assessment**, **DEXA** before or early in glucocorticoid therapy, and a **baseline ophthalmic assessment** before hydroxychloroquine.
10. **How the differences change management.** The **treat-to-target principle and the target itself (remission or low disease activity) are the same as in YORA — age alone is never a reason to withhold a DMARD**. **Methotrexate remains anchor therapy**, but must be **dose-reduced for the age-related fall in GFR**, given with **folic acid**, **avoided if eGFR is below 30**, and monitored closely for cytopenias, hepatotoxicity and pneumonitis. **Minimise glucocorticoids** — long-term low-dose steroid used *instead* of a DMARD is the single commonest and most damaging error in LORA (infection, osteoporosis, diabetes, cataract, skin fragility, delirium). **Use NSAIDs sparingly**: Kane's notes that older patients are particularly susceptible to gastrointestinal, CNS and renal adverse effects, and they are flagged in the Beers criteria. **Biologics are as effective as in younger patients** but carry a higher absolute risk of serious infection. **Screen and vaccinate before immunosuppression** (influenza, pneumococcal, recombinant zoster vaccine, COVID-19), and provide **bone protection** with any steroid course. Assess **comorbidity, polypharmacy, cognition, frailty and adherence** before choosing an agent — a comprehensive geriatric assessment is part of the rheumatology decision here.

Latest Updates (2023 – present)

11. A **2024 systematic review and meta-analysis** (Acuña-Rocha *et al.*, *Cureus* 2024, searching to November 2023) confirmed that **EORA presents with more intense disease activity, a higher prevalence of extra-articular manifestations, greater disability and more pronounced radiographic change at onset**, but that **the probability of achieving remission or low disease activity over one to two years is similar to YORA**. It also formally documented the treatment disparity — **EORA patients receive higher glucocorticoid and NSAID doses and fewer or lower-dose DMARDs** — establishing under-treatment, rather than biology, as the main driver of poorer real-world outcomes.
12. **Guidelines and drug safety.** The **Japan College of Rheumatology** has issued the **first and currently only national guideline specific to LORA**, recommending the term LORA and a treat-to-target approach explicitly adapted for comorbidity and frailty; dedicated **ACR and EULAR guidance for LORA is still awaited and is recognised as an urgent gap** (ACR Open Rheumatology, 2025). On drug safety, the **ORAL Surveillance** trial has led regulators and current EULAR recommendations to advise that **JAK inhibitors should not be used first-line in patients over 65**, or in those with cardiovascular risk factors, a smoking history or malignancy risk, because of increased major adverse cardiovascular events, venous thromboembolism, malignancy and herpes zoster — a restriction that falls disproportionately on exactly the LORA population.

Current Practice Update

Item	Traditional teaching	Current recommendation	Exam approach
Prognosis of LORA	Worse than YORA; more disabling	Higher activity at onset, but **similar remission rates with adequate treatment**; poorer outcomes reflect under-treatment	Say "under-treated, not treatment-resistant"
Seronegative disease	A milder form of RA	**Not mild** — may have more inflammatory activity and a worse prognosis; treat to target	A guaranteed viva point

Rheumatoid factor	A diagnostic test for RA	Loses specificity with age; **anti-CCP preferred** in older adults	Always mention both, and why
Steroids	Convenient and effective in the elderly	**Minimise** — bridge only, with bone protection; never as a substitute for a DMARD	Frame as the commonest management error
JAK inhibitors	An oral alternative to biologics	**Avoid first-line over 65** (ORAL Surveillance)	Name the trial

MNEMONIC ANCHOR

The differences of LORA = **"SALADS"** → **S**ex ratio approaches 1:1 (versus 3:1 female in YORA) · **A**cute, abrupt, infection-like onset (versus insidious) · **L**arge proximal joints, especially **shoulders** (versus small hand joints) · **A**nti-CCP and RF more often negative (seronegative) · **D**isease activity, disability, ESR and radiographic damage higher at onset · **S**ystemic constitutional features prominent (fever, weight loss, fatigue).

NUMERICAL ANCHORS

Onset at or after **60** years (many studies use **65**) · about **one-third** of new RA begins after 60 · female:male **3:1 in YORA versus ~1:1 in LORA** · **PMR-like phenotype in ~25%** of LORA · **>20%** of patients initially labelled PMR are reclassified as seronegative LORA · ACR/EULAR 2010 classification score **≥6 of 10** · methotrexate **avoid if eGFR <30** · JAK inhibitors **not first-line over 65**.

NAMED EXAMINER TRAPS

(a) A positive rheumatoid factor in an older adult may mean nothing — age-related immune dysregulation raises RF positivity in healthy elderly people; always send **anti-CCP**. **(b) Seronegative does not mean mild. (c)** Do not settle for a diagnosis of PMR — a "PMR" that **needs more than 25 mg of prednisolone, relapses on every taper, or develops peripheral synovitis or pitting oedema** is seronegative LORA, and needs a DMARD. **(d) Age is not a contraindication to methotrexate or to biologics** — comorbidity and renal function are. **(e)** Interpreting hand radiographs, **do not attribute the changes of coexisting nodal osteoarthritis to RA**; look specifically for **marginal erosions and periarticular osteopenia**. **(f)** Remember the **drug-induced causes** at this age — checkpoint inhibitors and, for the PMR-like picture, statin myopathy. **(g) Latent TB screening before biologics is not optional in India.**

15-SECOND ANSWER SKELETON

Define LORA and the age threshold, note one-third of RA starts after 60 → sex ratio → immunosenescence and weaker HLA-DRB1 link → run through **SALADS** for the clinical differences → serology with the two caveats → the three phenotypes and the PMR / RS3PE overlap → course and outcome (under-treated, not resistant) → **investigations in three groups: confirm, exclude, pre-treatment safety** → management implications (treat-to-target, methotrexate with renal adjustment, minimise steroids, avoid JAKi over 65, screen and vaccinate) → close with the 2024 meta-analysis and the Japanese LORA guideline.

EXTRA RECALL BOX — THE THREE-WAY COMPARISON TABLE (WRITE THIS AND YOU HAVE THE ANSWER)

Feature	YORA	LORA / EORA	Polymyalgia rheumatica
Age at onset	25–45 peak	≥60 (or ≥65)	Never below 50
Sex ratio (F:M)	3:1	~1:1	2–3:1
Onset	Insidious, weeks–months	**Abrupt, infection-like**	Abrupt or subacute
Joints	Symmetrical small joints — MCP, PIP, wrist, MTP; spares DIP	**Large proximal joints, shoulders**; often oligoarticular; may also involve small joints	**Girdles only** — shoulder and pelvic; no true peripheral synovitis
Pitting oedema of hands	Rare	**Characteristic** (RS3PE-like)	May occur
Constitutional symptoms	Less prominent	**Prominent**	Prominent
ESR / CRP at onset	Raised	**Markedly raised**	Markedly raised (ESR often >40)
RF / anti-CCP	Usually positive	**More often negative**	Negative
Erosions	Yes	Yes — more radiographic damage at onset	**No erosions**
Response to low-dose steroid	Partial	Partial; relapses on taper	**Dramatic within 24–72 h to 12.5–25 mg**
Needs a DMARD	Yes	**Yes**	Only if relapsing (methotrexate / sarilumab)

Indian context. RA prevalence in India is around **0.28–0.75%**, and with a rapidly ageing population LORA is an increasing share of the rheumatology workload. Practical answer points: **latent tuberculosis screening is mandatory before biologics or JAK inhibitors** given India's TB burden; **biosimilar TNF inhibitors and rituximab** have made biologic therapy affordable and are the realistic escalation option; methotrexate is inexpensive and remains the anchor drug; **AB-PMJAY and the Ayushman Vay Vandana Card** cover hospitalisation and joint replacement for eligible older adults; and **vitamin D deficiency and osteoporosis must be addressed at the first steroid prescription**.

Exam One-Liners

- Definition — RA with onset at or after **60 years** (many studies use 65).
- Sex ratio — **approaches 1:1**, unlike the 3:1 female predominance of YORA.
- Onset — **abrupt and infection-like**, with **large proximal joint** involvement.
- Serology — **more often seronegative**; RF loses specificity with age, so send **anti-CCP**.
- Commonest mimic — **polymyalgia rheumatica**; over 20% of "PMR" is reclassified as seronegative LORA.
- Characteristic sign — **pitting oedema of hands and feet** (RS3PE overlap).
- Prognosis — worse at onset, **similar remission with adequate treatment**.
- Anchor drug — **methotrexate**, renally dose-adjusted, with folic acid.
- Avoid first-line over 65 — **JAK inhibitors** (ORAL Surveillance).
- Commonest management error — **long-term steroid instead of a DMARD**.

Question 24. Psoriatic arthropathy 7 MARKS

ANSWER

Textbook Core — 6 Points

1. **Definition and classification.** A **seronegative inflammatory arthritis associated with psoriasis**, belonging to the spondyloarthropathy family. **Moll and Wright's five patterns**: (i) asymmetric **oligoarthritis**, (ii) symmetric **polyarthritis** (RA-like), (iii) **distal interphalangeal (DIP) predominant**, (iv) **spondylitis/sacroiliitis**, (v) **arthritis mutilans**.
2. **Clinical features. Dactylitis** ("sausage digit"), **enthesitis** (Achilles tendon, plantar fascia), **DIP joint involvement with nail disease** (pitting, onycholysis, subungual hyperkeratosis, oil-drop sign), inflammatory back pain, and extra-articular features such as **uveitis**. Skin disease usually precedes the arthritis. **Elderly-onset psoriatic arthritis** tends to be more **polyarticular, more acutely inflammatory (higher ESR/CRP) and more erosive** than younger-onset disease.
3. **Investigations. RF and anti-CCP negative**; ESR and CRP raised; uric acid may be raised (a source of diagnostic confusion). Radiology: **erosions with adjacent new bone formation**, the **"pencil-in-cup" deformity**, periostitis, ankylosis and **asymmetric sacroiliitis**. Diagnosis is made with the **CASPAR criteria**; the **PEST** questionnaire is used to screen psoriasis patients.
4. **Differential diagnosis in the older adult. Rheumatoid arthritis** (symmetric, seropositive, spares the DIP joints), **erosive/nodal osteoarthritis** of the DIP joints (Heberden's nodes — a major overlap in this age group), **gout** (common in older men on diuretics), pseudogout, and reactive arthritis.
5. **Management principles. Treat-to-target**, aiming for minimal disease activity. Physiotherapy, graded exercise, weight reduction, smoking cessation and active **cardiovascular risk reduction** — psoriatic arthritis carries a high burden of metabolic syndrome, diabetes and cardiovascular disease. Local corticosteroid injection for a single inflamed joint or enthesis. NSAIDs help mild peripheral disease but are **relatively contraindicated in older adults** (renal impairment, GI bleeding, hypertension, heart failure — Beers criteria).
6. **Drug therapy.** Conventional synthetic DMARDs — **methotrexate** (monitor renal function, reduce dose for the age-related fall in GFR, give folate), leflunomide or sulfasalazine for peripheral disease; csDMARDs do **not** work for axial disease. Biologics for inadequate response or axial involvement: **TNF inhibitors**, **IL-17 inhibitors** (secukinumab, ixekizumab), **IL-12/23** (ustekinumab) and **IL-23 inhibitors** (guselkumab, risankizumab). Targeted synthetic agents: **apremilast** (PDE4 inhibitor — no laboratory monitoring, well tolerated in frail older adults) and **JAK inhibitors** (tofacitinib, upadacitinib). **Screen for latent tuberculosis and hepatitis B before any biologic** — essential in India.

Latest Update (2023 – present)

7. Following the **ORAL Surveillance** safety data, current **EULAR and GRAPPA** recommendations advise that **JAK inhibitors should not be used first-line in patients over 65** or with cardiovascular risk factors, a smoking history or malignancy risk, because of increased **major adverse cardiovascular events, venous thromboembolism, malignancy and herpes zoster**. **IL-17 and IL-23 inhibitors are preferred** in this group — **bimekizumab** (dual IL-17A/F blockade) was approved for psoriatic arthritis in 2023–24 and **guselkumab** has strong axial and peripheral data — while **apremilast** remains the low-monitoring option for frail older adults with mild disease.

If this appears as a 15-mark question, add: pathogenesis (IL-23/IL-17 axis, HLA-B27 in axial disease, the enthesis as the primary site); full CASPAR criteria with scoring; the six GRAPPA domains (peripheral arthritis, axial disease, enthesitis, dactylitis, skin, nails) and domain-based drug selection; a comparison table of PsA versus RA versus osteoarthritis versus gout in the older patient; comorbidity screening (metabolic syndrome, fatty liver, depression, uveitis, inflammatory bowel disease); and vaccination and TB screening before immunosuppression in the Indian context.

Exam One-Liners

- Five patterns — **Moll and Wright**.
- Classic radiological sign — **pencil-in-cup deformity**.
- Classic clinical signs — **dactylitis, enthesitis, nail pitting, DIP arthritis**.
- Diagnostic criteria — **CASPAR**.
- Avoid first-line over 65 — **JAK inhibitors**.
- Safest low-monitoring agent in the frail — **apremilast**.

Question 25. Sjögren's syndrome 7 MARKS

ANSWER

Textbook Core — 6 Points

1. **Definition and classification.** A chronic **autoimmune exocrinopathy** in which **lymphocytic infiltration of exocrine glands** causes dry eyes and dry mouth. **Primary** Sjögren's occurs alone; **secondary** Sjögren's accompanies rheumatoid arthritis, SLE or systemic sclerosis. Strongly female-predominant (about **9:1**), with onset typically in the fifth to sixth decade and a further peak in older women.
2. **Glandular clinical features. Keratoconjunctivitis sicca** — gritty, burning eyes, photophobia, corneal filaments. **Xerostomia** — difficulty swallowing dry food, altered taste, **rampant dental caries**, oral candidiasis, angular cheilitis, difficulty retaining dentures. Also **parotid or submandibular gland enlargement**, dry skin, dry cough from tracheobronchial dryness, and vaginal dryness with dyspareunia.
3. **Extraglandular features.** Non-erosive arthritis, Raynaud's phenomenon, **interstitial lung disease**, **renal tubular acidosis and interstitial nephritis**, peripheral and small-fibre **neuropathy**, cutaneous vasculitis, and autoimmune thyroid disease. Profound **fatigue** is often the most disabling symptom. **The risk of non-Hodgkin (MALT) lymphoma is increased roughly 5–15 fold** — persistent parotid swelling, low C4, cryoglobulins and palpable purpura are the warning signs.
4. **Investigations. Anti-Ro/SSA and anti-La/SSB** antibodies, ANA and RF positive; **raised ESR with a normal CRP**; polyclonal hypergammaglobulinaemia; cytopenias; low complement. Objective tests: **Schirmer's test ≤5 mm in 5 minutes**, ocular staining score, **unstimulated whole salivary flow ≤0.1 mL/min**, and **labial minor salivary gland biopsy** showing focal lymphocytic sialadenitis with a **focus score ≥1**. Diagnosis uses the **ACR/EULAR 2016 criteria (score ≥4)**.
5. **The key geriatric differential — drug-induced dry mouth.** Age-related salivary hypofunction and, far more commonly, **medication** are the usual causes of dry mouth in an older person: **anticholinergics, tricyclic antidepressants, antihistamines, antipsychotics, diuretics, opioids and bladder antimuscarinics**. Also consider previous head and neck **radiotherapy**, IgG4-related disease, sarcoidosis, hepatitis C, amyloidosis, HIV and diabetes. **Always review the drug chart before diagnosing Sjögren's in an older patient.**
6. **Management.** Largely **symptomatic and preventive**. Eyes — preservative-free artificial tears, ocular lubricant ointment at night, topical ciclosporin, punctal plugs. Mouth — frequent sips of water, sugar-free gum, saliva substitutes, and **muscarinic agonists (pilocarpine or cevimeline)**, which must be used cautiously in the elderly (sweating, bradycardia, avoid in asthma, COPD and narrow-angle glaucoma). **Intensive dental care with topical fluoride** and treatment of oral candidiasis. **Hydroxychloroquine** for arthralgia and fatigue; **glucocorticoids ± rituximab or other immunosuppression** for severe systemic disease (vasculitis, ILD, neuropathy, glomerulonephritis). **Stop or substitute the offending anticholinergic drugs.**

Latest Update (2023 – present)

7. Current EULAR practice directs therapy by **systemic activity scoring using the ESSDAI**, with symptom burden captured by **ESSPRI**, rather than treating everyone identically. More importantly, **2024–2025 phase 2 and phase 3 trials of targeted biologics have for the first time shown disease-modifying benefit**: **ianalumab** (anti-BAFF-receptor), **nipocalimab** (FcRn blocker) and **dazodalibep** (CD40-ligand pathway inhibitor) all met their primary endpoints in Sjögren's disease — signalling the first prospect of therapy beyond symptomatic care.

If this appears as a 15-mark question, add: the full ACR/EULAR 2016 criteria with individual item weights; pathogenesis (epithelitis, type I interferon signature, B-cell hyperactivity and BAFF); a table of causes of xerostomia in the older adult; the detailed lymphoma risk-stratification model; management of renal tubular acidosis and hypokalaemic paralysis (a classic Indian presentation); the ESSDAI domains; and the impact of xerostomia on nutrition, dentition, denture tolerance, taste, speech and quality of life in the elderly.

Exam One-Liners

- Antibodies — **anti-Ro/SSA and anti-La/SSB**.
- Objective tests — **Schirmer ≤5 mm/5 min**; **focus score ≥1** on labial biopsy.
- Classic laboratory pattern — **high ESR with normal CRP**.
- Feared complication — **MALT lymphoma**; warning signs: persistent parotid swelling, low C4, cryoglobulins, purpura.
- Commonest cause of dry mouth in an older person — **drugs, not Sjögren's**.
- Secretagogues — **pilocarpine, cevimeline**.

PART VII

Metabolic Bone Disease

The two metabolic bone questions, which share a laboratory differential and should be revised together.

Question 26. Osteoporosis, DEXA and FRAX 15 MARKS RECURRING

ANSWER

Textbook Core — 10 Points

1. **Definition.** A skeletal disorder of **reduced bone mass with micro-architectural deterioration**, leading to bone fragility and increased fracture risk. The **WHO densitometric definition** is a value **2.5 standard deviations or more below the young adult mean — a T-score ≤ −2.5**. **Osteopenia** = T-score between −1 and −2.5; **severe (established) osteoporosis** = T ≤ −2.5 with a fragility fracture.
2. **DEXA. Central dual-energy X-ray absorptiometry is the standard method** for measuring bone mineral density at the **spine, hip and forearm**. The **T-score** compares with the young adult mean; the **Z-score** compares with age-matched peers and is used in younger patients and to flag secondary osteoporosis when ≤ −2.0. US guidelines recommend **DEXA for all women aged 65 or older**; the UK National Osteoporosis Guideline Group instead uses **FRAX first** and DEXA selectively. **Geriatric pitfall:** spinal osteoarthritis, vertebral fractures and aortic calcification **falsely elevate lumbar BMD** in the elderly — rely on the **total hip / femoral neck**.
3. **FRAX.** An **algorithm integrating clinical risk factors, with or without femoral neck BMD, to compute the 10-year probability of a major osteoporotic fracture and of hip fracture**. Inputs: **age, sex, weight and height (BMI), previous fragility fracture, parental hip fracture, current smoking, glucocorticoid therapy, rheumatoid arthritis, secondary osteoporosis and alcohol ≥3 units/day**. FRAX is **country-specific**, and an **Indian FRAX model** is available.
4. **FRAX — use and limitations.** Its main function is to set an **intervention threshold**: treat when the FRAX probability equals or exceeds the risk of someone with a prior fragility fracture. Limitations — it does **not** capture glucocorticoid dose, the number or recency of fractures, **falls**, lumbar spine BMD or diabetes. Probability can be **modulated** by the **trabecular bone score (TBS)**, glucocorticoid dose, hip axis length, fall history and type 2 diabetes.
5. **Major risk factors for fragility fracture. Age, female sex, low BMI, prior fragility fracture, parental hip fracture, height loss >4 cm, glucocorticoid therapy, smoking and excess alcohol**; plus **secondary osteoporosis** — rheumatoid arthritis, hyperthyroidism, primary hyperparathyroidism, hypogonadism, diabetes, inflammatory bowel disease, aromatase inhibitors, anorexia nervosa, organ transplantation, prolonged immobility, COPD and HIV. **In the oldest old, falls risk drives fracture risk at least as much as BMD does.**
6. **Investigating the older patient with a fragility fracture.** Only **3% of hip fractures in the elderly are pathological**, but **over half of patients are osteoporotic and almost all are osteopenic**; after 80, a woman with age-normal bone density already has a T-score of −2.5, so **DEXA for diagnosis alone is chiefly useful under 75**. Take a full **medication (corticosteroid) and alcohol history** and check **CBC, ESR, calcium (mg/dL), phosphate, alkaline phosphatase, creatinine, liver and thyroid function, 25(OH) vitamin D and serum/urine protein electrophoresis**. Secondary osteoporosis is uncommon in this age group, but **hyperthyroidism is the dominant cause**; also consider malnutrition, low body weight, hypogonadism, hyperparathyroidism and myeloma. Perform **vertebral fracture assessment or spine X-ray** if there is height loss or hyperkyphosis.
7. **Non-pharmacological management — for every patient. Calcium 800–1000 mg/day** (up to 1200 mg), **vitamin D 800–1000 IU/day**, **protein ≥1 g/kg body weight/day**, **daily weight-bearing and resistance exercise**, smoking cessation, alcohol ≤2 units/day, and **multifactorial falls prevention** with home hazard assessment and medication review. **Hip protectors** probably reduce hip fracture in nursing and residential care, but **poor acceptance and adherence limit their use**.
8. **First-line drug therapy. Oral bisphosphonates** — **alendronate 70 mg weekly** or **risedronate 35 mg weekly** (gastro-resistant risedronate may improve adherence). Take **fasting, upright, with plain water, 30 minutes before food**. Contraindications: **eGFR <30–35 mL/min**, oesophageal stricture or achalasia, inability to remain upright, and **uncorrected hypocalcaemia or vitamin D deficiency**. Adverse effects: oesophagitis, acute-phase reaction after IV zoledronate, and rarely **osteonecrosis of the jaw and atypical femoral fracture**. **Zoledronic acid 5 mg IV yearly** is the option when oral therapy fails or is not tolerated, and after hip fracture it reduces both **re-fracture and mortality**.
9. **Other agents and sequencing. Denosumab 60 mg subcutaneously 6-monthly** — usable in renal impairment, but monitor calcium and **never stop it abruptly**: discontinuation causes **rebound multiple vertebral fractures**, so it must be followed by a bisphosphonate for 1–2 years. For **very high or imminent fracture risk**, an **anabolic agent first — teriparatide, abaloparatide or romosozumab** (romosozumab is contraindicated within one year of myocardial infarction or stroke) — **always followed by an antiresorptive**. Menopausal hormone therapy and raloxifene/bazedoxifene are options in younger postmenopausal women; **calcitonin has weak fracture-prevention efficacy**.
10. **Follow-up and duration.** Assess **adherence and side effects**; use **bone turnover markers at 3–6 months to verify compliance** with bone-resorption inhibitors; repeat DEXA about every 2 years. Reassess after **5 years of oral**

or 3 years of intravenous bisphosphonate: **continue in high-risk patients; consider a 2-year drug holiday in low-risk patients, reconsidered yearly**; restart immediately if an incident fracture occurs. **Fracture Liaison Services** — systematic case-finding and treatment after every fragility fracture — are the best-established service model, and their benefits are well documented.

Latest Updates (2023 – present)

11. **Screening works; supplements alone do not.** The **SCOOP** randomised trial (*Lancet*) showed that **community FRAX-based screening of older women reduces hip fractures**, supporting systematic case-finding. Conversely, a meta-analysis of 33 randomised trials in 51,145 participants (**Zhao et al., JAMA**) found that **calcium, vitamin D, or both were NOT associated with a lower fracture risk in community-dwelling older adults** — supplements are a necessary adjunct to, but never a substitute for, specific anti-osteoporosis therapy.
12. **The "very high risk" category and anabolic-first sequencing.** Current international guidance (ESCEO/IOF, AACE, Endocrine Society and 2023–2025 updates) formally defines **very high risk** — a fracture within the last 24 months, a fracture while on treatment, multiple fractures, T-score ≤ −3.0, or very high FRAX probability — and recommends starting with an **anabolic agent (romosozumab or teriparatide) followed by an antiresorptive** in this group, because **sequence matters**: anabolic-then-antiresorptive produces substantially greater BMD and fracture-risk reduction than the reverse order.

Current Practice Update

Item	Textbook	Current recommendation	Exam approach
Who to treat	T-score ≤ −2.5 (or ≤ −2.0 per NOF)	Fracture probability (FRAX ± BMD) above an intervention threshold, with a defined very-high-risk group	Give the WHO T-score definition, then say treatment is now risk-based
Calcium + vitamin D	"First line of defence"	Essential adjunct, but does not reduce fracture on its own in community dwellers	Quote the JAMA meta-analysis
Drug sequencing	Start with a bisphosphonate	Anabolic first (romosozumab/teriparatide) in very high risk, then antiresorptive	Say "sequence matters"
Denosumab	Effective antiresorptive	Must never be stopped without follow-on bisphosphonate — rebound vertebral fractures	A guaranteed viva question
Case finding	Opportunistic	Fracture Liaison Service + FRAX-based community screening (SCOOP)	Name FLS as the service model

MNEMONIC ANCHOR

FRAX inputs = **"SCRAP + GAP + BMI (± BMD)"** → **S**moking, **C**orticosteroids, **R**heumatoid arthritis, **A**lcohol ≥3 units, **P**revious fragility fracture · **G**ender, **A**ge, **P**arental hip fracture · plus **BMI** and, optionally, **BMD** (± secondary osteoporosis).

NUMERICAL ANCHORS

T ≤ −2.5 osteoporosis · −1 to −2.5 osteopenia · Z ≤ −2.0 suggests secondary cause · height loss >4 cm · alcohol ≥3 units/day · calcium 800–1200 mg · vitamin D 800–1000 IU · protein ≥1 g/kg · alendronate 70 mg weekly · risedronate 35 mg weekly · zoledronate 5 mg yearly · denosumab 60 mg 6-monthly · eGFR cut-off for bisphosphonates <30–35 · review at 5 years oral / 3 years IV · very high risk if T ≤ −3.0 or fracture within 24 months · only 3% of elderly hip fractures are pathological.

NAMED EXAMINER TRAPS

(a) T-score versus Z-score — know exactly which is used where. **(b) Never stop denosumab** without a follow-on bisphosphonate. **(c) Correct vitamin D deficiency and hypocalcaemia BEFORE** giving a potent antiresorptive — otherwise symptomatic hypocalcaemia. **(d) Lumbar spine DEXA is unreliable in the elderly** — use the hip. **(e)** FRAX **without** BMD is a valid and intended use — it is the entry point in the NOGG pathway. **(f)** FRAX **underestimates** risk in recurrent fallers, in high-dose steroid users and after a very recent fracture. **(g)** A fragility fracture at any T-score is enough to justify treatment — do not wait for the DEXA.

15-SECOND ANSWER SKELETON

Define with WHO T-score → DEXA (sites, T vs Z, elderly pitfall) → FRAX (inputs, 10-year probability, intervention threshold, limitations) → risk factors including falls → exclude secondary causes → lifestyle + calcium/vitamin D/protein/exercise/falls prevention → oral bisphosphonate first line with administration rules and contraindications → zoledronate/denosumab/anabolic sequencing → monitoring, duration and drug holiday → Fracture Liaison Service → SCOOP and anabolic-first updates.

EXTRA RECALL BOX — INDIAN CONTEXT

Osteoporosis is **under-diagnosed and under-treated in India**, with a large **treatment gap** even after hip fracture. Practical points: **vitamin D deficiency is near-universal** in Indian older adults (limited sun exposure, skin pigmentation, covered dress, vegetarian diets), so **check and replete 25(OH) vitamin D first**; **ICMR-NIN** recommends 1000 mg/day calcium for older adults, and dietary calcium intake is typically low; the **ISBMR (Indian Society for Bone and Mineral Research) position statements** guide Indian practice; the **Indian FRAX model** should be used rather than the UK/US one; and **orthogeriatric co-management with early surgery (<48 h) and comprehensive geriatric care after hip fracture** improves function and survival (Prestmo *et al.*, *Lancet* 2015).

Exam One-Liners

- WHO definition — **T-score ≤ −2.5**.
- Gold standard measurement — **central DEXA of hip and spine**.
- FRAX output — **10-year probability of major osteoporotic and hip fracture**.
- FRAX omits — **falls, steroid dose, fracture recency and number, lumbar BMD, diabetes**.
- First-line drug — **oral bisphosphonate**; if eGFR <30 — **denosumab**.
- After hip fracture — **zoledronic acid reduces re-fracture and mortality**.
- Best service model — **Fracture Liaison Service**.

Question 27. Paget's disease of bone — epidemiology, aetiology, pathophysiology, clinical manifestations and management

15 MARKS RECURRING

ANSWER

Source note. Paget's disease is **not covered adequately in the uploaded textbooks**. Pathy's 6th ed. carries only a single index entry (p. 1026), and Kane's mentions it in three places only — the metabolic bone disease laboratory table, the table of disorders of hearing in older adults, and the table of medical illnesses associated with depression. **Those textbook fragments are used below and marked**; the remaining disease-specific content is drawn from the Endocrine Society and UK Paget's Association clinical guidelines and is clearly separated.

Textbook Core — 10 Points

1. **Definition.** Paget's disease of bone (**osteitis deformans**) is a **focal disorder of bone remodelling** in which greatly increased and disorganised **osteoclastic bone resorption** is followed by **vigorous but disorganised compensatory osteoblastic new bone formation**. The result is **expanded, thickened, hypervascular but structurally weak woven bone** with a characteristic **mosaic pattern of irregular cement lines**. It may be **monostotic** or **polyostotic**, and — a key examination point — **once established, it does not spread to previously uninvolved bones**; the extent at diagnosis is the lifelong extent.
2. **Epidemiology.** A disease of **later life** — **rare below 40 years**, with prevalence rising steadily with age to about **1–2% of those over 55** in the United Kingdom. **Male predominance**, roughly **3:2**. There is striking **geographic clustering**: highest rates in the **United Kingdom** (classically Lancashire), Western Europe, Australia, New Zealand and North America; it is **rare in Scandinavia, Asia and Africa** and **uncommon in India**. The **incidence, prevalence and severity have all fallen substantially over the past three decades**, which is strong indirect evidence for an environmental cause.
3. **Aetiology — genetic factors. 15–40% have a positive family history**, with autosomal dominant inheritance and incomplete penetrance. Mutations in **SQSTM1**, which encodes the protein **p62** in the RANK–NF-κB signalling pathway, are the commonest — found in about **40–50% of familial** and **5–10% of sporadic** cases, the **P392L** mutation being most frequent. Other susceptibility loci involve **TNFRSF11A (RANK), TNFRSF11B (osteoprotegerin), CSF1, OPTN and TM7SF4/DC-STAMP** — all genes governing osteoclast formation and function.
4. **Aetiology — environmental factors.** The longest-standing hypothesis is **chronic paramyxovirus infection of osteoclasts** (measles, canine distemper or respiratory syncytial virus), supported by **nuclear inclusion bodies resembling paramyxovirus nucleocapsids** in pagetic osteoclasts — but this remains **unproven and controversial**. Other proposed factors: low dietary calcium and vitamin D in youth, **repetitive mechanical loading** of the skeleton, cattle and rural exposure, and environmental toxins. The falling incidence in the absence of any genetic change points firmly to an environmental contribution.
5. **Pathophysiology — the three phases.**

Phase	Bone biology	Radiological correlate
1. Osteolytic (lytic)	Greatly increased number and size of **osteoclasts with abnormally numerous nuclei**; intense resorption	**Osteoporosis circumscripta** in the skull; **"blade of grass" or "flame-shaped"** advancing lytic front in a long bone
2. Mixed	Vigorous compensatory **osteoblastic** bone formation with **marrow fibrosis and marked hypervascularity**; **alkaline phosphatase rises sharply**	Mixed lysis and sclerosis; cortical thickening begins
3. Sclerotic (burnt-out)	Dense, disorganised **woven bone** with a **mosaic of irregular cement lines**; turnover falls	**Bone expansion, thickened cortex, coarse trabeculae**; "cotton wool" skull, "picture frame" vertebra

   The consequences flow directly from this biology: the bone is **bigger but weaker** (deformity and fracture), **hypervascular** (warmth, bleeding at surgery, arteriovenous shunting), and **distorts adjacent structures** (nerve compression, secondary osteoarthritis).
6. **Distribution.** Involvement is **asymmetric and axially predominant**. In descending order of frequency: **pelvis** (commonest), **lumbar spine, femur, skull, tibia, humerus and sacrum**. Skull and long-bone involvement account for most of the clinically important complications.
7. **Clinical manifestations — general. 70–80% of patients are asymptomatic** and are detected incidentally on a radiograph taken for another reason, or through an **isolated raised alkaline phosphatase**. Kane's specifically notes that **mild asymptomatic elevations of alkaline phosphatase are common in the elderly, but liver disease and Paget's disease should be considered if the elevation is moderate**. When symptomatic, the features are: **bone pain** — deep, boring, **worse at rest and at night** and **not relieved by rest** (unlike osteoarthritis); **bone deformity** — anterolateral bowing of the tibia (**"sabre tibia"**), femoral bowing, **increasing skull size so that the hat no longer fits**, frontal bossing, kyphosis and loss of height; and **increased warmth over the affected bone** from hypervascularity. Kane's also lists **Paget's disease among the medical illnesses that may present with or be accompanied by depression**.

8. **Skeletal complications. Pathological fracture** — characteristically **transverse "chalk-stick" fractures** of a bowed long bone. **Secondary osteoarthritis** of the hip and knee — in practice **the commonest cause of pain** in a patient with Paget's disease. **Protrusio acetabuli**, spinal stenosis and vertebral collapse. Rarely, **osteosarcoma in under 1%** — heralded by a **sudden increase in pain, a new soft-tissue mass and a further rise in alkaline phosphatase**, and carrying a very poor prognosis.
9. **Neurological, otological and cardiac complications. Deafness** is the classic complication of skull disease and is **mixed** — conductive from ossicular and cochlear capsule involvement, and sensorineural from cochlear involvement and eighth nerve compression. Kane's lists Paget's disease among the causes of hearing loss in older adults and states that it **"may lead to both kinds of hearing loss and should be evaluated radiologically and by an alkaline phosphatase determination"**. Skull base disease causes **platybasia and basilar invagination** with brainstem compression and hydrocephalus; spinal disease causes **cord and nerve root compression**, sometimes by a vascular steal mechanism. Extensive polyostotic disease involving more than roughly **15–20% of the skeleton** can cause **high-output cardiac failure** through arteriovenous shunting. Also seen: **hypercalcaemia of immobilisation** and hyperuricaemia with gout.
10. **Investigations and management. Biochemistry** — **serum total alkaline phosphatase** is the key screening and monitoring test; Kane's metabolic bone table gives the diagnostic pattern for Paget's disease as **normal or high calcium, normal or high phosphate, HIGH alkaline phosphatase and NORMAL parathyroid hormone**. Use **bone-specific ALP or P1NP** if there is coexisting liver disease. **Radiology** — **plain radiographs of the relevant regions confirm the diagnosis**; a **radionuclide bone scan** is then done to **map the extent** of disease (more sensitive but less specific). Biopsy only if malignancy is suspected. **Indications to treat** — symptomatic bone pain due to active disease, and to prevent or limit complications: **before elective surgery on pagetic bone** (reduces operative bleeding), neurological compression, hypercalcaemia of immobilisation, high-output cardiac failure, and active disease at **critical sites** (skull, spine, weight-bearing long bones, bone adjacent to a major joint). **Drug of choice** — a **single 5 mg intravenous infusion of zoledronic acid**, which produces a more rapid, more complete and more sustained response than oral bisphosphonates, with **biochemical remission in about 90%** of patients, often lasting many years. Alternatives: **risedronate 30 mg daily for 2 months**, **alendronate 40 mg daily for 6 months**, or intravenous pamidronate; **calcitonin** only if bisphosphonates are contraindicated. **Correct vitamin D deficiency and hypocalcaemia before infusion**; caution if eGFR is below about 35 mL/min; warn about the **acute-phase reaction** after the first dose. **Monitor** total alkaline phosphatase at 3–6 months and then 6–12 monthly, and **retreat no sooner than 12 months** after zoledronate if pain recurs or ALP rises. **Adjunctive care** — analgesia (paracetamol; NSAIDs only with great caution in older adults), physiotherapy, walking aids, a **shoe raise or orthosis for leg-length discrepancy**, **hearing aids and audiological review**, **joint replacement for secondary osteoarthritis**, and surgical decompression for cord or nerve compression.

Latest Updates (2023 – present)

11. **PRISM and PRISM-EZ have overturned the "treat to normalise alkaline phosphatase" doctrine.** The **PRISM** trial randomised 1324 patients to intensive bisphosphonate therapy aimed at normalising bone turnover versus symptomatic treatment; its extension, **PRISM-EZ**, continued 502 patients with **zoledronic acid as the first-choice agent in the intensive arm**, giving a total follow-up of about **7.3 years**. Intensive therapy produced **no reduction in fractures, orthopaedic procedures, progression of deafness, bone pain or quality of life**; indeed the intensive arm showed a signal of **more fractures and more orthopaedic surgery**. The authors concluded that in established Paget's disease, **bisphosphonate therapy should focus on the control of symptoms rather than on the suppression of bone turnover**.
12. **Guidelines and the genetics of prevention.** The **Endocrine Society clinical practice guideline** and the **UK Paget's Association guideline (JBMR 2019)** agree on the substantial fall in incidence, on a similar approach to evaluation (plain radiographs to confirm, then a bone scan to define extent), and both name **a single 5 mg intravenous dose of zoledronate as the treatment of choice** where there is no contraindication. The **ZiPP randomised controlled trial** of **prophylactic zoledronic acid in asymptomatic carriers of SQSTM1 mutations** showed that treatment can prevent or modify the development of new bone lesions but **did not alter clinical outcomes** over the trial period — so **genetic screening with pre-emptive treatment is not routine practice**.

Current Practice Update

Item	Textbook / traditional position	Current recommendation	Exam approach
Treatment target	Suppress bone turnover; normalise alkaline phosphatase	**Treat symptoms, not the number** — intensive therapy does not reduce fracture, surgery or deafness (PRISM-EZ)	State the old doctrine, then answer with PRISM-EZ
First-line drug	Calcitonin and older oral bisphosphonates (etidronate, pamidronate)	**Single 5 mg IV zoledronate**; remission in ~90%, sustained for years	Name the drug, dose and route in one line
Asymptomatic disease	Treat all active disease	Treat only for **critical sites, planned surgery or complication risk**	Give the indication list, not a blanket rule
Genetic testing	Not available	**SQSTM1** testing available, but pre-emptive treatment of carriers is not	Useful closing update line

		routine (ZiPP)	
Retreatment	Repeat when ALP rises	Retreat for **recurrent symptoms**, and not sooner than **12 months** after zoledronate; otherwise re-evaluate the cause of pain	Links to the "pain is usually OA" trap

MNEMONIC ANCHOR

Complications = **"BONE PAIN"** → **B**one pain (deep, night, rest) · **O**steoarthritis (secondary — the commonest cause of pain) · **N**erve compression and deafness · **E**xpansion and deformity (sabre tibia, skull enlargement) · **P**athological "chalk-stick" fracture · **A**rteriovenous shunt → high-output cardiac failure · **I**mmobilisation hypercalcaemia · **N**eoplasia (osteosarcoma <1%).

NUMERICAL ANCHORS

Rare below **40** years · about **1–2%** over 55 in the UK · male:female **3:2** · family history in **15–40%** · SQSTM1 in **40–50% familial / 5–10% sporadic** · **70–80% asymptomatic** · osteosarcoma **<1%** · high-output failure if **>15–20%** of skeleton involved · **zoledronate 5 mg IV single dose**, remission in **~90%** · risedronate **30 mg daily × 2 months** · alendronate **40 mg daily × 6 months** · minimum retreatment interval **12 months** · PRISM-EZ total follow-up **7.3 years**.

NAMED EXAMINER TRAPS

(a) Serum calcium and phosphate are characteristically normal — a raised calcium means immobilisation, a recent fracture, or a coexisting primary hyperparathyroidism or malignancy, not Paget's disease itself. **(b)** The commonest cause of pain in these patients is **secondary osteoarthritis, not active Paget's disease** — so pain with a normal ALP will not respond to a bisphosphonate, and the cause must be re-evaluated. **(c) Paget's disease never spreads to new bones** — new lesions on a repeat scan should make you doubt the diagnosis. **(d)** A **sudden increase in pain with a soft-tissue mass and a further ALP rise = osteosarcoma** until proved otherwise. **(e) Correct vitamin D and calcium before zoledronate**, or you will precipitate symptomatic hypocalcaemia. **(f)** ALP may be **normal in limited monostotic disease**, so a normal ALP does not exclude the diagnosis. **(g)** Give a bisphosphonate **before elective orthopaedic surgery** on pagetic bone to reduce intra-operative haemorrhage.

15-SECOND ANSWER SKELETON

Define (focal disorder of remodelling, osteitis deformans, mosaic woven bone, never spreads to new bones) → epidemiology (rare <40, 1–2% over 55, M>F, geographic clustering, falling incidence, rare in India) → aetiology (SQSTM1/p62 plus putative paramyxovirus) → three phases with radiological correlates → distribution (pelvis, spine, femur, skull, tibia) → 70–80% asymptomatic; bone pain, deformity, warmth → complications by the BONE PAIN mnemonic → investigations (ALP, normal Ca/PO_4/PTH; X-ray to confirm, bone scan to map) → indications to treat → single 5 mg IV zoledronate → monitoring and adjunctive care → close with PRISM-EZ.

EXTRA RECALL BOX — THE METABOLIC BONE DISEASE TABLE (KANE'S), AND THE INDIAN CONTEXT

This table answers half the metabolic bone questions in the paper and should be reproduced verbatim:

Disease	Calcium	Phosphate	Alkaline phosphatase	PTH
Hyperparathyroidism	High	Low / normal	High / normal	High
Osteomalacia	Low / normal	Low	High / normal	High
Hyperthyroidism	High	High	High / normal	Low
Osteoporosis	Normal	Normal	Normal	Normal / high
Paget's disease	**Normal / high**	**Normal / high**	**HIGH**	**NORMAL**

Indian context. Paget's disease is **genuinely rare in India and in people of Indian origin**, and the few reported series come mainly from western India. Therefore, in an older Indian patient with an **isolated raised alkaline phosphatase**, the far more likely diagnoses are **osteomalacia** (very common — low vitamin D, low or normal calcium, low phosphate, raised PTH), **hepatobiliary disease**, and **skeletal metastases** (breast, prostate, lung) — and this differential, worked through the table above, is what earns the marks. Practical Indian points: **vitamin D deficiency must be corrected before any potent bisphosphonate**; zoledronic acid is available and affordable as a generic; and joint replacement for secondary osteoarthritis is covered under **AB-PMJAY**.

Exam One-Liners

- Alternative name — **osteitis deformans**.
- Histological hallmark — **mosaic pattern of irregular cement lines in woven bone**.
- Commonest gene — **SQSTM1 (p62)**, P392L mutation.
- Commonest site — **pelvis**.
- Skull lytic sign — **osteoporosis circumscripta**; sclerotic sign — **"cotton wool" skull**.
- Long-bone lytic sign — **"blade of grass" / flame-shaped front**; fracture — **transverse "chalk-stick"**.
- Biochemistry — **high ALP with normal calcium, phosphate and PTH**.

- X-ray confirms the diagnosis; **bone scan defines the extent**.
- Drug of choice — **single 5 mg IV zoledronic acid**.
- Feared malignant complication — **osteosarcoma (<1%)**.
- Cardiac complication — **high-output cardiac failure** from arteriovenous shunting.
- Current treatment principle — **treat symptoms, not the alkaline phosphatase**.

PART VIII

Pressure Injury and Prescribing Safety

Two geriatric syndromes of iatrogenesis — the injury that marks quality of nursing care, and the prescribing that causes more admissions than any single disease.

Question 28. Pressure ulcers — staging and management

15 MARKS RECURRING

ANSWER

Textbook Core — 10 Points

1. **Definition.** Localised injury to skin and/or underlying tissue, usually over a **bony prominence**, resulting from **sustained pressure, or pressure in combination with shear**. The current NPUAP/NPIAP term is **"pressure injury"**, which better reflects that damage can occur under intact skin.
2. **Pathogenesis.** Sustained pressure exceeding **capillary closing pressure** causes tissue ischaemia. **Shearing forces** — for example when the head of the bed is elevated and the torso slides down, transmitting force to the sacral area — stretch and angulate subcutaneous tissues, leading to **thrombosis of small blood vessels** and destruction of skin from beneath. **Friction** from repeated movement across sheets increases shear, and both are worsened by the **loose, folded skin** that is common in the elderly from loss of subcutaneous tissue and dehydration. **Moisture** from bathing, sweat, urine and faeces compounds the damage.
3. **Sites. More than 90% of pressure ulcers occur in the lower body** — mainly the **sacral and coccygeal areas, the ischial tuberosities and the greater trochanter**; also heels, malleoli and occiput. Critically, **the area of damage beneath the ulcer can be much larger than the visible ulcer**, because pressure and shear are transmitted to deeper subcutaneous tissues.
4. **Risk factors.** Being **bed- or chair-bound**, inability to reposition without assistance, difficulty with ambulation, history of **stroke**, **faecal incontinence**, low body weight, **lymphopenia**, difficulty with independent feeding, impaired nutritional intake, **non-blanchable erythema of intact skin (a Stage 1 injury)**, and dry sacral skin. **Advanced age has NOT been identified as an independent risk factor** — it is the coexisting impairments in mobility, nutrition and skin health that create risk.
5. **Risk assessment.** Commonly used tools are the **Braden, Norton and Waterlow scales**. Current recommendation: apply a risk-assessment tool **on admission to a facility and regularly thereafter**. Because pressure injury shares risk factors with falls, incontinence and functional dependence, **multifactorial interventions delivered by a multidisciplinary team** are effective, just as for other geriatric syndromes.
6. **Staging (NPIAP). Stage 1** — acute inflammatory response limited to the epidermis; **intact skin with non-blanchable erythema** persisting more than 30 minutes; may look red, blue or purple in darker skin; skin unbroken; often over a bony prominence. **Stage 2** — extension through the dermis to the junction of subcutaneous fat; a **blister, abrasion or shallow ulcer** with distinct edges. **Stage 3** — **full-thickness** loss through subcutaneous fat, down to but not through fascia; presents as a **crater with undermining**; base often necrotic and foul-smelling. **Stage 4** — extension through deep fascia, with **bone, tendon or muscle visible**; extensive undermining and **sinus tracts**; **osteomyelitis and septic arthritis** may be present. **Unstageable** — base obscured by slough or eschar. **Deep tissue pressure injury** — persistent non-blanchable deep red, maroon or purple discolouration, or a blood-filled blister.
7. **Staging rules the examiner will test. Medical device-related pressure injuries are staged**, and often **conform to the shape of the device**. **Pressure injuries of mucous membranes are NOT staged** — they are documented only. **Reverse staging is inaccurate for assessing healing**; instead use the validated **PUSH tool (Pressure Ulcer Scale for Healing)**, which scores **size, exudate amount and tissue type** and is sensitive to change over time.
8. **Prevention — the highest-yield section. Identify patients at risk**; **decrease pressure, friction and skin folding**; regular repositioning; use **support surfaces** — non-powered (foam, gel, air or water overlays and mattresses) or powered (alternating-pressure surfaces, which are far more costly); **keep skin clean and dry**; **avoid excessive bed rest — optimise and encourage function**; **avoid over-sedation**; and provide **adequate nutrition and hydration: 30–35 kcal/kg/day, protein 1–1.5 g/kg/day and fluid 1 mL/kcal**.
9. **Management of Stage 1 and 2.** Apply **all preventive measures** to stop progression, plus intensive local skin care. **Clean with warm normal saline or water**; avoid pressure and moisture; **cover open wounds with an occlusive dressing** selected on the basis of granulation and necrotic tissue, type and amount of drainage, surrounding tissue and evidence of infection; prevent further injury and infection, **using antibiotics very judiciously**; and **manage the associated pain**. No single local technique is superior to another — **the important factor is the attention the skin receives, above all relief from pressure**.
10. **Management of Stage 3 and 4. Debride necrotic tissue** — autolytic, chemical/enzymatic, mechanical, sharp or surgical; **undermine and remove eschar** if it is hiding necrotic or infected tissue; **irrigate and cleanse two to three times daily** and dress the wound. **Stage 3** — **routine wound swabs are not warranted** (they grow multiple organisms, miss anaerobes and reflect colonisation, not infection); systemic antimicrobials do not reach adequate concentration in the ulcer and should be reserved for **confirmed bacteraemia, sepsis, osteomyelitis or cellulitis**. **Stage 4** — take a **tissue or bone biopsy for aerobic and anaerobic culture**, give systemic antimicrobials, and obtain **surgical consultation for possible flap repair**. Manage pain at every stage.

Latest Updates (2023 – present)

11. The **EPUAP/NPIAP/PPPIA International Clinical Practice Guideline** and its subsequent implementation updates recommend **prophylactic multilayer silicone foam dressings** over the sacrum and heels in high-risk patients (strong evidence for prevention in ICU and emergency transport), **repositioning every 2–4 hours matched to the support surface and the individual** rather than a rigid 2-hourly rule, and **sub-epidermal moisture (SEM) measurement** to detect impending damage **before visible erythema appears**.
12. On adjunctive therapies, the current evidence base is clear: **high-protein oral nutritional supplements enriched with arginine, zinc and antioxidants accelerate healing of Stage 2–4 ulcers**, whereas there is **insufficient evidence for hyperbaric oxygen, topical oxygen or biological dressings**, and only limited evidence for platelet-derived growth factor. **Preventing pressure injury and managing the underlying causes — rather than focusing on topical dressings — remains what is most likely to be effective.**

Current Practice Update

Item	Textbook	Current recommendation	Exam approach
Terminology	Pressure sore/ulcer, Stages I–IV	Pressure injury, Stages 1–4 plus Unstageable and Deep Tissue Pressure Injury	Use current terms; mention the old ones once
Assessing healing	Staging	Never reverse-stage; use the PUSH tool	Certain viva question
Cleansing	Saline or peroxide	Normal saline or potable water; avoid cytotoxic antiseptics on granulating tissue	Say "saline, not antiseptic"
Repositioning	Regular repositioning	Every 2–4 hours, individualised to the support surface	Do not quote a rigid 2-hourly rule
Prophylactic dressings	Not described	Multilayer silicone foam over sacrum and heels in high-risk patients	High-value latest-update line

MNEMONIC ANCHOR

Risk factors = **"PRESSURE"** → **P**ressure and immobility · **R**educed nutrition and low body weight · **E**lderly friable skin · **S**ensory loss (stroke, neuropathy) · **S**hear and friction · **U**rinary and faecal incontinence (moisture) · **R**educed perfusion (anaemia, peripheral vascular disease, hypotension, lymphopenia) · **E**xisting Stage 1 erythema.

NUMERICAL ANCHORS

>90% occur in the lower body · Stage 1 erythema persists >30 minutes · reposition every 2–4 hours · energy **30–35 kcal/kg/day** · protein **1–1.5 g/kg/day** · fluid **1 mL/kcal** · irrigate 2–3 times daily · PUSH tool scores 3 components (size, exudate, tissue type).

NAMED EXAMINER TRAPS

(a) Never reverse-stage a healing ulcer — a Stage 4 that improves is a "healing Stage 4". **(b) Mucosal pressure injuries are not staged. (c)** Do **not** send routine wound swabs — they reflect colonisation. **(d)** Systemic antibiotics do **not** penetrate a Stage 3 ulcer — reserve them for cellulitis, osteomyelitis or sepsis. **(e) Advanced age is not an independent risk factor. (f)** Do not debride a **stable, dry, adherent eschar on an ischaemic heel** — it acts as a biological dressing. **(g)** Unstageable means you cannot see the base — it is not a separate depth.

15-SECOND ANSWER SKELETON

Define (pressure ± shear over a bony prominence) → pathogenesis (capillary closure, shear, friction, moisture) → sites >90% lower body → risk factors and Braden/Norton/Waterlow on admission → the four stages plus unstageable and DTPI → staging rules (devices staged, mucosa not, no reverse staging, PUSH tool) → prevention bundle with nutrition figures → Stage 1–2 management → Stage 3–4 debridement and the culture/antibiotic rules → silicone foam and nutrition updates → "prevention beats dressings".

EXTRA RECALL BOX

Pressure injury is a geriatric syndrome, sharing risk factors with falls, incontinence and functional dependence — which is why **multidisciplinary, multicomponent intervention** works, exactly as in delirium prevention. Two further points examiners like: **(i) an unavoidable pressure injury** is a recognised concept at the end of life (Kennedy terminal ulcer) — documentation and comfort, not blame, is the response; and **(ii) pressure injuries are a recognised marker of quality of care and of elder neglect** — in India, an unexplained Stage 3–4 sacral ulcer in a dependent older person should trigger consideration of **neglect under the Maintenance and Welfare of Parents and Senior Citizens Act, 2007**, and referral through **Elder Line 14567**.

Exam One-Liners

- Definition — injury from **pressure, or pressure with shear**, over a bony prominence.
- Commonest sites — **sacrum, ischial tuberosity, greater trochanter, heel**.

- Stage 1 — **non-blanchable erythema of intact skin**.
- Stage 4 — **bone, tendon or muscle visible**.
- Risk tools — **Braden, Norton, Waterlow**.
- Healing tool — **PUSH**; never reverse-stage.
- Nutrition — **30–35 kcal/kg, protein 1–1.5 g/kg, fluid 1 mL/kcal**.
- Single most effective measure — **relief of pressure**.

Question 29. Beers criteria, STOPP/START and anticholinergic burden 15 MARKS

RECURRING

ANSWER

Textbook Core — 10 Points

1. **The three forms of suboptimal prescribing. Overuse** (drugs without a valid indication), **misuse** (potentially inappropriate medications, wrong dose or inadequate monitoring) and **underuse** (omission of indicated, beneficial drugs). **Polypharmacy** — conventionally five or more drugs — is the principal modifiable driver of adverse drug reactions, together with the **number of comorbid conditions and the number of prescribers**.
2. **Scale of the problem.** In the **Three-City (3C) study**, representative of community-dwelling French adults aged 65 and over, **21.7% were receiving an inappropriate medication** — **6.4% an anticholinergic** and **9.2% a long half-life benzodiazepine**. European studies using Beers or McLeod lists report rates of **10–20%**; a French geriatric short-stay unit found **66% had at least one inappropriate medication on admission**. Rates in **nursing homes are much higher** and are linked to hospitalisation and death. Commonly implicated: cerebral vasodilators, long half-life benzodiazepines, anticholinergic antidepressants, and two or more psychotropics of the same class; also PPIs continued without indication and antipsychotics over-prescribed in nursing homes.
3. **Beers criteria — what they are.** An **explicit list**, first developed by **Mark Beers in 1991** for institutionalised older people using a literature review and **Delphi method**, since adapted for ambulatory patients and **updated regularly by the American Geriatrics Society**. It applies to adults **≥65 years in ambulatory, acute and institutional settings — but not in hospice or end-of-life care**.
4. **Structure of the Beers criteria.** Five core tables plus supporting tables: **(i)** potentially inappropriate medications to avoid in most older adults; **(ii)** PIMs to avoid because of **drug–disease or drug–syndrome interactions**; **(iii)** drugs to **use with caution**; **(iv)** clinically important **drug–drug interactions** to avoid; **(v)** drugs to avoid or **dose-adjust for reduced kidney function**; plus a table of **drugs with strong anticholinergic properties**. Classic examples: benzodiazepines and Z-drugs (falls, fractures, delirium), first-generation antihistamines, tricyclic antidepressants, antipsychotics for behavioural symptoms of dementia (increased stroke and mortality), NSAIDs, skeletal muscle relaxants, sliding-scale insulin, and PPIs beyond 8 weeks without indication.
5. **Limitations of the Beers criteria.** It was designed for **collective (population and quality-indicator) evaluation, not for individual patients** — a criticism Beers himself accepted. It **does not cover underuse**; it is **US-specific**, and in one European study about **half the listed medications were unavailable in most European countries**; and **few studies have demonstrated a clinical benefit** from applying the criteria. Some analyses found Beers-listed drugs account for only a small proportion of actual ADRs.
6. **The drugs that actually cause harm.** In Budnitz *et al.*, only **3.6%** of emergency admissions for adverse drug events involved drugs "always inappropriate" by Beers and 5.2% those inappropriate in certain conditions, whereas **33.3% followed just three drugs — warfarin (17.3%), insulin (13%) and digoxin (3.2%)**. Pirmohamed *et al.* similarly implicated **low-dose aspirin, diuretics, warfarin and NSAIDs**. The lesson: **encourage good prescribing and monitoring rather than merely stigmatising "inappropriate" drugs** — most avoidable errors occur at the **monitoring stage (60–80%)**.
7. **STOPP and START.** Developed by **Irish geriatricians using a Delphi method** and organised **by physiological system**, which makes them more usable at the bedside. **STOPP** = Screening Tool of Older Persons' Prescriptions — detects potentially inappropriate prescriptions. **START** = Screening Tool to Alert to Right Treatment — detects **prescribing omissions**. In 715 older patients admitted acutely, an ADR was identified in 90 (12.5%), of which **STOPP identified 91% versus 48% for Beers**; STOPP-detectable prescriptions contributed to **11.5% of admissions versus 6% for Beers**. STOPP is therefore especially useful when the presentation is non-specific, such as **falls or delirium**.
8. **START and the problem of underuse.** Applied to 600 acutely admitted patients over 65, **at least one potentially useful medication had been omitted in 57.9%**, rising to **72.2% in those over 85**, with **women at twice the risk of men**. The omissions were mainly cardiovascular: **statins in atherosclerotic disease (26%), anticoagulants in atrial fibrillation (9.5%), antiplatelets in arterial disease (7.3%), and calcium and vitamin D in osteoporosis with fracture (6%)**. START takes **under 3 minutes** to apply once the list of conditions and treatments is available.
9. **Anticholinergic burden.** The **cumulative anticholinergic effect of multiple drugs**, each individually regarded as only mildly anticholinergic. **Peripheral effects**: dry mouth, blurred vision, constipation, urinary retention, tachycardia, reduced sweating and heat intolerance. **Central effects**: sedation, confusion, **delirium, falls and accelerated cognitive decline**. Older adults are more susceptible because of reduced central cholinergic reserve, increased blood–brain barrier permeability, and reduced hepatic and renal clearance. Common culprits: **amitriptyline, oxybutynin and tolterodine, chlorpheniramine, hydroxyzine, promethazine, olanzapine and clozapine, hyoscine, dicyclomine, trihexyphenidyl, prochlorperazine**. Quantify with the **Anticholinergic Cognitive Burden (ACB) scale**, Anticholinergic Risk Scale or Anticholinergic Drug Scale — an **ACB score ≥3** is associated with cognitive decline, falls and mortality. Prescribing an antimuscarinic bladder drug alongside a **cholinesterase inhibitor** is a classic **prescribing cascade** and a pharmacological contradiction.
10. **Other tools and the practical approach.** The **Medication Appropriateness Index (MAI)** is an **implicit** tool that evaluates **ten criteria for each drug**: indication, efficacy, dosage, correct directions, practical directions, drug–drug interactions, drug–disease interactions, duplication, duration and cost. A workable **deprescribing sequence**: **(i)** list

every drug including OTC and herbal preparations, **(ii)** confirm a current indication for each, **(iii)** apply STOPP/START and Beers, **(iv)** calculate the anticholinergic burden, **(v)** rank by risk–benefit against the patient's goals and life expectancy, **(vi)** withdraw **one drug at a time**, tapering where withdrawal effects are expected, and **(vii)** monitor for withdrawal syndromes and disease recurrence.

Latest Updates (2023 – present)

11. **2023 AGS Beers Criteria update** (*J Am Geriatr Soc* 2023;71:2052–81) — key changes: **avoid initiating aspirin for primary cardiovascular prevention**, and **consider deprescribing** it in existing users; **avoid warfarin as initial therapy** for VTE or non-valvular atrial fibrillation (DOACs preferred; **rivaroxaban avoided**); **avoid ALL sulfonylureas**; **use SGLT2 inhibitors with caution** (euglycaemic ketoacidosis, urogenital infection); **avoid initiating oral or transdermal oestrogen**; anticholinergics reorganised into **antihistamine, CNS/antiparkinson and gastrointestinal** groups with explicit emphasis on **cumulative anticholinergic burden**; ticagrelor and skeletal muscle relaxants added; ~28–33 rarely used drugs removed; and a caution added **against abruptly stopping digoxin in HFrEF**. AGS also published an **"Alternative Treatments" companion (JAGS 2025)** giving safer pharmacological and non-pharmacological substitutes.
12. **STOPP/START version 3 (2023, *Age and Ageing*)** expanded the tool to **133 STOPP and 57 START criteria**, with new sections covering **antithrombotics, drugs that add to anticholinergic burden, drugs that predictably worsen frailty and falls, and drugs to avoid in limited life expectancy**. Meanwhile the **OPERAM** and related multicentre trials show that structured medication review reduces drug-related hospital admissions **only when the recommendations are actually implemented — implementation, not detection, is now the bottleneck**.

Current Practice Update

Item	Textbook	Current recommendation	Exam approach
Aspirin	Widely used; a common cause of ADR	2023 Beers: avoid initiating for primary prevention; consider deprescribing	Quote it as a named 2023 change
Anticoagulation	Warfarin standard; a leading ADR cause	Avoid warfarin as initial therapy; DOAC preferred, rivaroxaban avoided	Pair with "warfarin caused 17.3% of ADR admissions"
Sulfonylureas	Not specifically restricted	Avoid all sulfonylureas; SGLT2 inhibitors with caution	High-yield diabetes overlap
STOPP/START	2008 version, 65 STOPP / 22 START	Version 3 (2023): 133 STOPP / 57 START	Quote both versions and the numbers
Anticholinergics	Considered drug by drug	Cumulative burden quantified (ACB); target a score below 3	Name the scale and the threshold

MNEMONIC ANCHOR

"STOPP the wrong · START the missing · SCORE the anticholinergics." And for the deprescribing sequence: **"LIST – INDICATION – SCREEN – SCORE – RANK – ONE AT A TIME – MONITOR."**

NUMERICAL ANCHORS

Polypharmacy ≥5 drugs · Beers applies from age 65 · Three-City 21.7% inappropriate (6.4% anticholinergic, 9.2% long-acting benzodiazepine) · 66% of geriatric short-stay admissions had ≥1 PIM · STOPP detected 91% vs Beers 48% of ADRs · admissions attributable 11.5% vs 6% · omission in 57.9%, rising to 72.2% over 85 · MAI has 10 criteria · STOPP/START v3 = 133 + 57 · ACB threshold ≥3 · warfarin 17.3%, insulin 13%, digoxin 3.2% of ADR admissions.

NAMED EXAMINER TRAPS

(a) Beers is **US-specific** and **excludes hospice and end-of-life care**. **(b)** Remember that **START is about underuse** — candidates routinely forget it. **(c)** "Potentially inappropriate" is **not** "absolutely contraindicated" — it requires clinical judgement and shared decision-making. **(d) Do not abruptly stop digoxin** in a current user with HFrEF (2023 Beers caveat). **(e)** The drugs causing the most ADR admissions — warfarin, insulin, digoxin, antiplatelets — are largely **NOT** on the Beers list, so the criteria are a supplement to, not a substitute for, good monitoring. **(f)** Most avoidable errors occur at the **monitoring** stage, not the prescribing stage.

15-SECOND ANSWER SKELETON

Three types of suboptimal prescribing (overuse, misuse, underuse) → scale of the problem with figures → Beers: origin, Delphi, ≥65, five tables, examples → limitations of Beers → what actually causes ADRs (warfarin, insulin, digoxin) → STOPP/START: system-based, detects underuse, better ADR yield with figures → anticholinergic burden: mechanism, culprits, ACB ≥3, prescribing cascade → MAI and the seven-step deprescribing sequence → 2023 Beers changes and STOPP/START v3.

EXTRA RECALL BOX — INDIAN CONTEXT

Indian-specific prescribing hazards for the older adult: widespread **over-the-counter availability** of antibiotics, NSAIDs and benzodiazepines despite **Schedule H1**; heavy use of **irrational fixed-dose combinations**; concurrent **AYUSH and allopathic** prescribing with unrecognised interactions (for example ashwagandha with sedatives, or bhasma preparations with heavy-metal content); multiple prescribers with no shared record; and cost-driven **non-adherence and self-tapering**. Practical answer points: use a **single reconciled medication list at every transition of care**, engage the family caregiver, prefer once-daily and fixed-dose regimens for adherence in the frail, and note that **no Indian consensus PIM list has been formally adopted nationally** — Beers and STOPP/START are used with local adaptation, since many listed drugs differ in availability.

Exam One-Liners

- Polypharmacy — **≥5 medications**.
- Beers — **explicit** list, AGS, age ≥65, not for hospice.
- STOPP — inappropriate prescriptions; **START — prescribing omissions**.
- STOPP versus Beers for detecting ADRs — **91% versus 48%**.
- MAI — **implicit** tool, **10 criteria per drug**.
- Anticholinergic scale and threshold — **ACB ≥3**.
- 2023 Beers headline change — **avoid aspirin for primary prevention**.
- Latest tool version — **STOPP/START v3 (133 + 57)**.

APPENDIX

Consolidated Night-Before Master Sheets

The three original rapid-recall grids, retained in full. Question numbers in these sheets refer to the original question sets; use the Table of Contents to map them to the numbering used in this volume.

Night-Before Master Sheet — Questions 63 to 75

A. The single highest-yield line for each topic

Q	Topic	The one sentence that earns the mark
63	Herpes zoster / PHN	Antiviral within **72 hours for 7 days**; PHN = pain beyond **3 months**; prevention is the **recombinant zoster vaccine from age 50**.
64	CAUTI	All chronic catheters are bacteriuric — **treat the patient, not the culture**; the best prevention is **early removal**.
65	Asymptomatic bacteriuria	**Do not screen and do not treat**, except before urological procedures with mucosal bleeding.
66	IPC and AMS	**Hand hygiene** is the most effective IPC measure; **NAP-AMR 2.0 (2025–29)** makes IPC mandatory in every Indian facility.
67	Leptospirosis and dengue	In dengue the elderly die of **fluid overload as often as of shock**; in leptospirosis, **doxycycline early, dialysis early**.
68	COVID-19	**Delirium and silent hypoxia** are the presentations to know; antiviral within **5 days**, steroids only if hypoxic.
69	PMR	Girdle pain with a **normal CK** and a **dramatic response to 12.5–25 mg prednisolone**; watch for **GCA**.
70	Psoriatic arthropathy	**Seronegative** arthritis with **dactylitis, enthesitis, nail disease and pencil-in-cup**; avoid JAK inhibitors over 65.
71	Systemic vasculitides	**GCA above 50, MPA at 65–75**; start steroids before the biopsy; **infection kills more older patients than the vasculitis**.
72	Sjögren's syndrome	**Anti-Ro/La**, Schirmer ≤5 mm, focus score ≥1; the commonest cause of dry mouth in an older person is a **drug**.
73	Osteoporosis / DEXA / FRAX	**T ≤ −2.5**; FRAX gives 10-year probability; **never stop denosumab** without a follow-on bisphosphonate.
74	Pressure ulcers	Four stages plus **unstageable and DTPI**; **never reverse-stage**; prevention beats dressings.
75	Beers / STOPP-START / ACB	**STOPP the wrong, START the missing, score the anticholinergics (ACB ≥3)**.

B. Steroid-sparing agents — the whole rheumatology block on one line each

Condition	First line	Steroid-sparing / advanced
Polymyalgia rheumatica	Prednisolone 12.5–25 mg/day	**Sarilumab** preferred (EULAR 2025); tocilizumab; methotrexate
Giant cell arteritis	Prednisolone 40–60 mg (IV if visual loss)	**Tocilizumab** or **upadacitinib 15 mg** (SELECT-GCA: 46% vs 29%)
ANCA-associated vasculitis	Steroids + **rituximab** (preferred in elderly)	**Avacopan**; reduced-dose steroid regimen (PEXIVAS)
Psoriatic arthritis	Methotrexate (peripheral only)	**IL-17 / IL-23 inhibitors**; apremilast if frail; avoid JAKi over 65
Sjögren's syndrome	Symptomatic + hydroxychloroquine	Rituximab for systemic disease; ianalumab / nipocalimab emerging

C. Numbers most likely to be asked

Number	What it belongs to
72 hours / 7 days / 3 months	Zoster antiviral window, course, PHN definition
51% and 66%	Zostavax reduction in herpes zoster and PHN
≥10^5 CFU/mL	Significant bacteriuria (voided specimen)
45 minutes / >40 mm/h / >75 mm/h	PMR morning stiffness; PMR ESR; ESR raising suspicion of GCA

12.5–25 mg vs 40–60 mg	Prednisolone in PMR vs GCA
16–21%	PMR patients who develop GCA
46% vs 29%	SELECT-GCA sustained remission, upadacitinib vs placebo
T ≤ −2.5 / −1 to −2.5 / ≤ −3.0	Osteoporosis / osteopenia / very high risk threshold
800–1200 mg and 800–1000 IU	Daily calcium and vitamin D
5 years oral / 3 years IV	Bisphosphonate review point before a drug holiday
30–35 kcal/kg, 1–1.5 g/kg protein, 1 mL/kcal	Nutrition target in pressure injury
>90% / >30 minutes / 2–4 hourly	Lower-body ulcers, Stage 1 erythema, repositioning interval
91% vs 48%	STOPP vs Beers detection of ADRs
57.9% and 72.2%	Prescribing omission, all ages ≥65 and over 85
133 + 57 / ACB ≥3	STOPP/START v3 criteria; anticholinergic burden threshold

D. Indian policy and programme hooks to drop into any answer

Programme / instrument	Where it fits
NAP-AMR 2.0 (2025–2029), launched 18 Nov 2025	CAUTI, asymptomatic bacteriuria, IPC and stewardship (Q64, 65, 66)
ICMR AMRSN and ICMR AMSP guidelines	Hospital antibiogram, stewardship programme structure (Q66)
NCVBDC dengue and leptospirosis programmes; **TAK-003 approved 2026 for ages 4–60**	Dengue and leptospirosis (Q67)
NPHCE — geriatric clinics, district and regional geriatric centres	Vaccination access, falls and osteoporosis services, chronic care (Q63, 68, 73)
ISBMR position statements; **Indian FRAX** model; **ICMR-NIN** calcium recommendation	Osteoporosis (Q73)
MWPSC Act 2007 and **Elder Line 14567**	Neglect presenting as a Stage 3–4 pressure injury (Q74)
Schedule H1; AYUSH–allopathy co-prescribing; irrational fixed-dose combinations	Prescribing safety and stewardship (Q66, 75)
AB-PMJAY, **Rashtriya Vayoshri Yojana**, **ADIP-ALIMCO**	Financing of fracture surgery, mobility and assistive devices (Q73, 74)

FIVE SENTENCES WORTH MEMORISING VERBATIM

1. "Bacteriuria in older persons is common and usually asymptomatic; in the absence of obstructive uropathy there is no evidence to support routine antimicrobial therapy."
2. "Advanced age has not been identified as an independent risk factor for pressure injury — it is the coexisting impairments in mobility, nutrition and skin health that create the risk."
3. "Polymyalgia rheumatica is a totally reversible cause of frailty that is often misdiagnosed; corticosteroids reverse the frailty it produces."
4. "Prevention and managing the underlying causes, rather than focusing on topical dressings, is most likely to be effective in pressure injury."
5. "It is more relevant to encourage good prescribing practices with the aim of reducing avoidable iatrogenic events than to stigmatise inappropriate treatments."

Night-Before Master Sheet — Questions 168 and 169

A. The one sentence per topic

Q	Topic	The sentence that earns the mark
168	Paget's disease	Focal disorder of remodelling with **high ALP but normal calcium, phosphate and PTH**; X-ray confirms, bone scan maps; **single 5 mg IV zoledronate**; **treat symptoms, not the alkaline phosphatase**.
169	Late-onset RA	**SALADS** — sex ratio 1:1, acute onset, large proximal joints, seronegative, higher disease activity, systemic features; **under-treated, not treatment-resistant**.

B. Numbers most likely to be asked

Number	What it belongs to
1–2% over 55; rare below 40; M:F 3:2	Paget's disease epidemiology
SQSTM1 in 40–50% familial, 5–10% sporadic	Paget's genetics; P392L mutation
70–80% asymptomatic; osteosarcoma <1%	Paget's presentation and worst complication
>15–20% of skeleton	Threshold for high-output cardiac failure
Zoledronate 5 mg once; remission ~90%; retreat ≥12 months	Paget's drug of choice
Risedronate 30 mg × 2 months; alendronate 40 mg × 6 months	Oral alternatives in Paget's
Onset ≥60 (or 65); one-third of RA	LORA definition and burden
F:M 3:1 versus ~1:1	YORA versus LORA sex ratio
PMR-like in ~25%; >20% of PMR reclassified	LORA phenotypes and the PMR overlap
ACR/EULAR 2010 score ≥6/10; eGFR <30	RA classification; methotrexate cut-off

C. Two trials and two guidelines to name

Trial / guideline	What to say about it
PRISM / PRISM-EZ	Intensive bisphosphonate therapy to normalise turnover gave **no reduction in fracture, orthopaedic surgery, deafness or quality of life**, with a signal of harm — **treat symptoms, not the number**.
ZiPP	Prophylactic zoledronic acid in **SQSTM1 carriers** modified new lesion development but did not change clinical outcomes — genetic screening with pre-emptive treatment is **not routine**.
Endocrine Society / UK Paget's Association	Both name **single 5 mg IV zoledronate** first-line; X-ray to confirm, bone scan to define extent.
ORAL Surveillance	**JAK inhibitors not first-line over 65** — MACE, VTE, malignancy, herpes zoster.

THREE SENTENCES WORTH MEMORISING VERBATIM

1. "Paget's disease may lead to both conductive and sensorineural hearing loss and should be evaluated radiologically and by an alkaline phosphatase determination." (Kane's)
2. "In patients with established Paget's disease of bone, bisphosphonate therapy should focus on control of symptoms rather than suppression of bone turnover." (PRISM-EZ)
3. "Seronegative rheumatoid arthritis is not a mild form of the disease — it requires the same intensive treat-to-target therapy as seropositive disease."

THE LINK BETWEEN THE TWO QUESTIONS

Both are **diseases of bone and joint in old age that are frequently missed because the presentation is constitutional rather than musculoskeletal**. Paget's is usually found through an **isolated raised alkaline phosphatase**; LORA is usually found through **weight loss, fatigue and a very high ESR**. In each case the examiner is testing whether you will **work systematically through the differential — the metabolic bone table for one, the OA-versus-inflammatory-arthritis table for the other — rather than jumping to the diagnosis in the question.**

Night-Before Master Sheet — Questions 103 to 116

A. The single highest-yield line for each topic

Q	Topic	The sentence that earns the mark
103	DOTS and DR-TB	**BPaLM for 6 months** is the preferred MDR/RR-TB regimen; XDR (2021) = pre-XDR + resistance to **bedaquiline or linezolid**.
104	GeneXpert	Cartridge PCR detecting **MTB + rifampicin resistance in 2 hours** via the ***rpoB* hot-spot**; **never use for test of cure**.
105	FUO	In over-65s, **GCA/PMR is the commonest identifiable cause**; the highest-yield "tests" are the **repeated examination and the drug chart**.
106	Shock and septic shock	Septic shock = **vasopressor-dependent MAP <65 plus lactate >2**; **no antibiotic substitutes for source control**.
107	Procalcitonin	**Suppressed by interferon-γ**, so it stays low in viral infection; best proven role is **shortening antibiotic duration**.
108	Cerebral malaria	**IV artesunate 2.4 mg/kg at 0, 12, 24 h then daily**; exclude and correct **hypoglycaemia**; no steroids.
109	Chikungunya	**Lymphopenia** (not thrombocytopenia); **no NSAIDs until dengue is excluded**; chronic arthritis in 40–50%.
110	Scrub typhus	**Hunt for the eschar in hidden moist sites**; doxycycline 100 mg BD with **defervescence in 48 hours**.
111	Ebola	Death is from **hypovolaemic shock, not haemorrhage**; **Ervebo ring vaccination** and **Inmazeb/Ebanga** for Zaire only.
112	*C. difficile*	**Fidaxomicin first-line**; **soap and water, not alcohol gel**; test only diarrhoeal stool, never a test of cure.
113	Influenza	**High-dose/adjuvanted/recombinant vaccine from 65**; oseltamivir **30 mg BD if CrCl 30–60**.
114	Vaccination	**Immunosenescence** drives the schedule: influenza annually, PCV20/21 from 50, RZV ×2 from 50, RSV from 75.
115	Acute pyelonephritis	Classic triad may be absent — **delirium and falls may be the only features**; **white cell casts** mean parenchymal disease.
116	Recurrent UTI	**Vaginal oestrogen first, methenamine second, antibiotics last**; systemic HRT does not work.

B. The geriatric principles that run through the whole module (quote these anywhere)

1. "Febrile response may be blunted or absent in some older individuals with bacterial infections… A poor febrile response may also be a negative prognostic factor." (Kane's)
2. "The absence of leucocytosis in older patients should also not exclude consideration of a bacterial infection." (Kane's)
3. "Hypothermia in older patients should promptly be treated as sepsis unless proven otherwise." (Kane's)
4. "Acute unexplained functional deterioration should alert the physician to a potential acute infectious process." (Kane's)
5. "A useful rule in geriatric care is to suspect tuberculosis when a patient is inexplicably failing." (Kane's)
6. "The frequency of gram-negative bacilli increases in each category" of infection in older adults — so empirical cover must include them. (Kane's)
7. "Bacteriuria in older persons is common and usually asymptomatic… in the absence of obstructive uropathy, no evidence exists to support the routine use of antimicrobial therapy." (Kane's)

C. Numbers most likely to be asked

Number	What it belongs to
BPaLM 6 months; bedaquiline 400 mg → 200 mg thrice weekly	MDR/RR-TB regimen and dosing
81-bp *rpoB*; 2 hours; 16 CFU/mL (Ultra)	GeneXpert target, turnaround, Ultra limit of detection
>38.3 °C, >3 weeks; LTC fever >37.8 °C	FUO definition and its geriatric modification
SOFA ≥2; MAP <65; lactate >2; mortality >40%	Sepsis-3 and septic shock
30 mL/kg in 3 h; antibiotics <1 h; MAP 60–65 if ≥65 y	Septic shock bundle and SSC 2026
Rises 2–4 h, peaks 12–24 h; >0.5 ng/mL	Procalcitonin kinetics and antibiotic threshold

Artesunate 2.4 mg/kg at 0, 12, 24 h; GCS ≤11	Cerebral malaria
Eschar in 20–50%; doxycycline 100 mg BD × 7 d; response in 48 h	Scrub typhus
Incubation 2–21 days; semen persistence ≥18 months	Ebola
WBC ≥15,000 or creatinine >1.5 mg/dL; recurrence 20–25% then 40–60%	*C. difficile* severity and recurrence
HR 0.75 hospitalisation, 0.52 death; oseltamivir 30 mg BD if CrCl 30–60	Influenza vaccine benefit and antiviral dosing
PCV from 50; RZV ×2 from 50; RSV all ≥75	Current adult immunisation schedule
2 in 6 months or 3 in 12 months; methenamine 1 g BD; fluids 1.5 L	Recurrent UTI

D. Indian policy and programme hooks

Programme / instrument	Where it fits
NTEP, Ni-kshay portal, **Ni-kshay Poshan Yojana ₹1000/month**, Pradhan Mantri TB Mukt Bharat Abhiyaan	DOTS and drug-resistant TB (Q103)
Universal drug susceptibility testing; CBNAAT; indigenous **Truenat**	GeneXpert and TB diagnostics (Q103, 104)
NAP-AMR 2.0 (2025–2029); ICMR treatment guidelines and AMRSN	Sepsis, procalcitonin, *C. difficile*, pyelonephritis, recurrent UTI (Q106, 107, 112, 115, 116)
NCVBDC; National Framework for Malaria Elimination; pre-referral rectal artesunate	Cerebral malaria, chikungunya, scrub typhus (Q108–110)
NPHCE geriatric clinics; API / Geriatric Society of India adult immunisation guidance	Vaccination delivery — no adult UIP in India (Q113, 114)
AB-PMJAY and **Ayushman Vay Vandana Card**	Financing of hospitalisation for sepsis, TB and severe infection

THE THREAD THAT LINKS THIS WHOLE MODULE

Every answer in this block turns on the same three geriatric facts: **(i) the febrile and leucocyte responses are unreliable**, so the presentation is delirium, falls and functional decline rather than fever; **(ii) the pathogen spectrum shifts towards Gram-negative organisms**, so empirical cover and local antibiograms matter more; and **(iii) the greatest iatrogenic risks are over-treatment of colonisation, unadjusted drug doses, and fluid overload during resuscitation**. If you open any answer in this module with those three points and close it with the relevant Indian programme, you will have framed it as a geriatrics answer rather than a general medicine one.

THE TAMIL NADU DR. M.G.R. MEDICAL UNIVERSITY · BRANCH XVI

MD Geriatrics Paper III

Geriatrics
(Geriatric Medicine)

Sub. Code 2057 / 4043

19	228	181	17
EXAM SESSIONS	QUESTIONS ASKED	UNIQUE AFTER DEDUP	THEMED SECTIONS

INSIDE THIS DOCUMENT

A How this paper is examined
Question counts across all four papers, the mark scheme, where the marks actually are, and the most-repeated topics.

B Master question list
All 228 questions ever set, de-duplicated to 181 unique entries and grouped into 17 themed sections with repeat and mark-format badges.

C Ebenezer bank — not yet asked
92 Paper III topics that appear in the Ebenezer question bank but have never been set by the university.

D Predicted questions
Most likely essays and short notes for the next session, with the reasoning behind each call.

Complete de-duplicated question bank, organised by system and theme

March 2009 – October 2025 university papers · Ebenezer's Geriatric Question Bank · Frequency analysis and predicted questions

Contents

Part A · How this paper is examined . . . 3

Question counts across all four papers . . . 3

The Paper III mark scheme . . . 4

Where the marks actually are . . . 4

The most-repeated topics . . . 5

What has changed in the examiner's style . . . 5

Part B · Master question list . . . 7

1. Geriatric Assessment, Frailty & Sarcopenia . . . 7

2. Falls, Gait, Balance & Dizziness . . . 7

3. Cognition, Dementia & Delirium . . . 8

4. Parkinson's Disease & Movement Disorders . . . 9

5. Neurology — Stroke, Coma, Seizures & Neuromuscular . . . 10

6. Cardiovascular Medicine . . . 10

7. Respiratory Medicine . . . 11

8. Continence, Bladder & Men's Health . . . 12

9. Renal, Fluid & Electrolyte Disorders . . . 13

10. Bone, Joint & Rheumatology . . . 14

11. Endocrine & Metabolic . . . 14

12. Gastroenterology & Nutrition . . . 15

13. Haematology & Oncology . . . 16

14. Infections & Immunity . . . 16

15. Sleep, Skin, Special Senses & Environmental . . . 17

16. Pharmacology, Prescribing & Perioperative Care . . . 18

17. Rehabilitation, Social & End-of-Life Care . . . 18

Part C · Ebenezer's bank — Paper III topics not yet asked . . . 20

Part D · Predicted questions . . . 24

Paper III · most likely 15-mark essays . . . 24

Paper III · most likely 7-mark notes . . . 24

A model predicted Paper III . . . 25

Cross-paper watchlist — Papers I, II and IV . . . 26

Part A · How this paper is examined

HOW TO READ THIS DOCUMENT

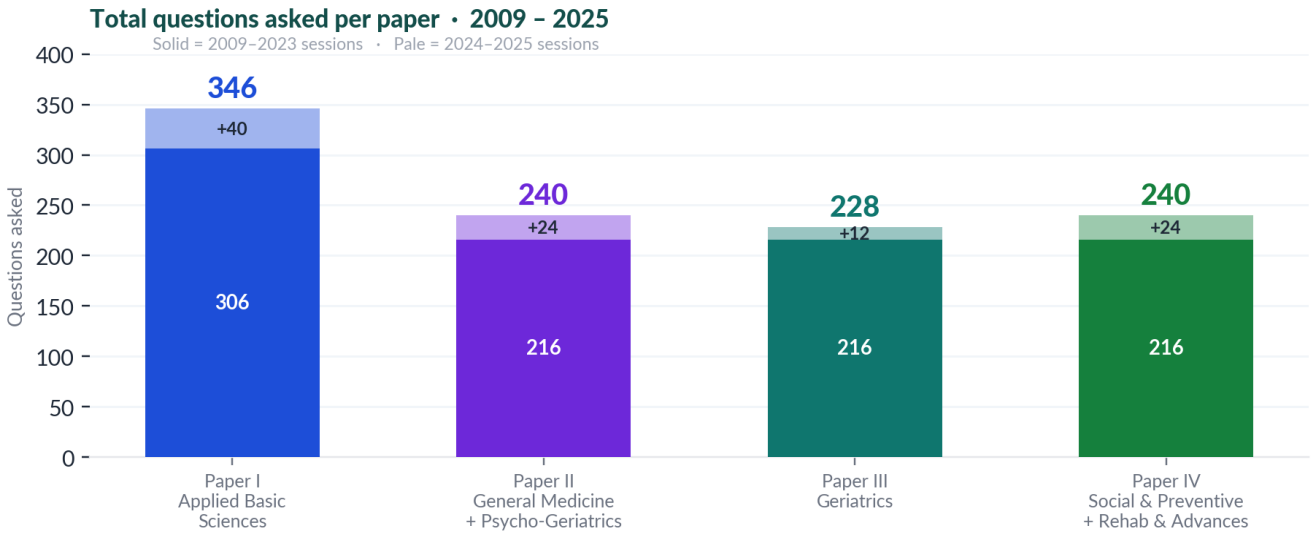
Every question ever set in Paper III between March 2009 and October 2025 has been extracted, merged with the Ebenezer question bank, stripped of year tags and repeats, and re-grouped by system and theme. Nothing has been dropped: the 228 original question slots have each been traced into one of the 181 unique entries in Part B, and the mapping was verified programmatically before this document was generated.

Question counts across all four papers

The four papers do not carry equal question loads. Paper I uses a six-subject short-note format with twenty questions per session, while Papers II, III and IV each use two long essays plus ten short notes — twelve questions per session. Counts below are taken from the question papers available in the source archive.

Paper	Sub. Code	Sessions available	Questions per session	Total questions
Paper I — Applied Basic Sciences	2059 / 4041	17	20 (4+4+3+3+3+3)	**346**
Paper II — General Medicine incl. Psycho-Geriatrics	2056 / 4042	20	12 (2 essays + 10 notes)	**240**
Paper III — Geriatrics	2057 / 4043	19	12 (2 essays + 10 notes)	**228**
Paper IV — Social & Preventive Geriatrics	2060 / 4044	20	12 (2 essays + 10 notes)	**240**
All four papers combined	—	76	—	**1,054**

Paper I is missing the October 2013, 2014, 2015 and December 2024 Paper III sessions from the archive; the October 2024 Paper III paper is also absent. Counts reflect what is actually in the source files rather than every session the university has ever conducted.



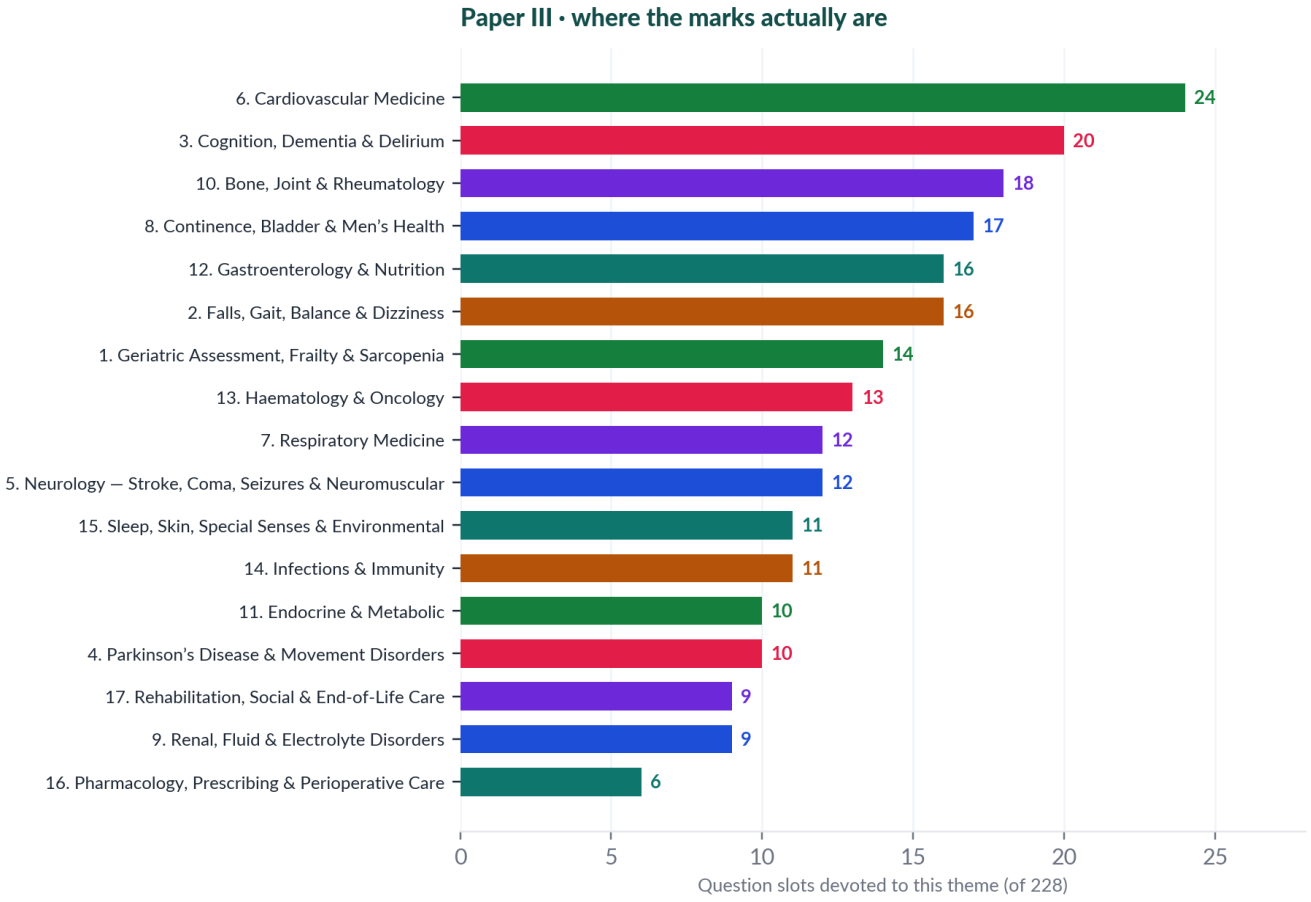
The Paper III mark scheme

Two formats have been used. Sessions up to March 2010 set two 20-mark essays and ten 6-mark notes. From May 2011 onward the paper has settled into two 15-mark essays and ten 7-mark notes, and this is the format you should calibrate every answer to. Each question in Part B carries its mark-format descriptor so you can size your answers correctly.

Period	Section I	Section II	Answer calibration
2009 – 2010	2 × 20 marks	10 × 6 marks	Historic format — treat 20-mark stems as 15-mark essays
2011 – 2025	2 × 15 marks	10 × 7 marks	Essay: ~11 pages / 35 min. Note: ~4 pages / 10 min

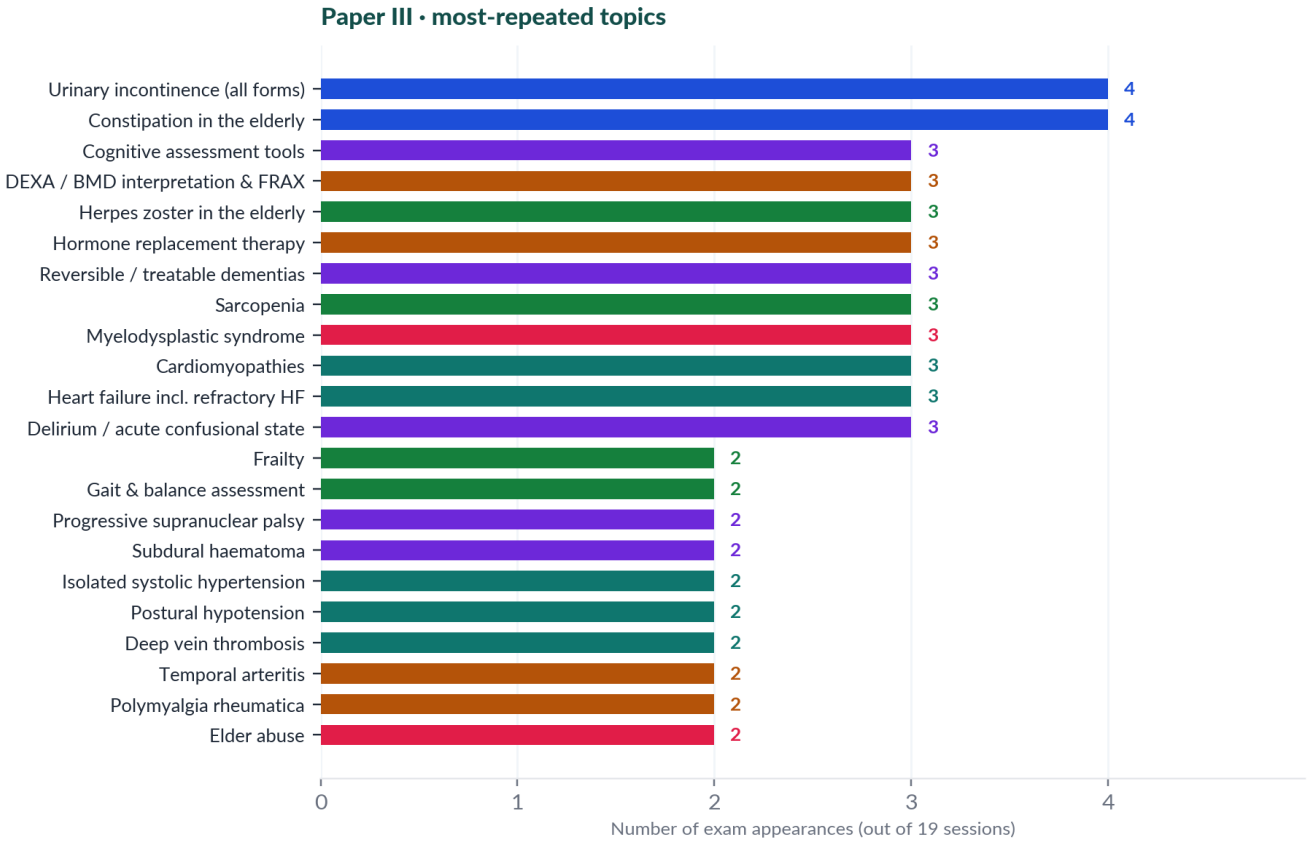
Where the marks actually are

Grouping all 228 question slots by theme shows where this paper actually concentrates. Cardiovascular medicine is the single largest block, and together with cognition and delirium, bone and joint disease, continence, and gastroenterology and nutrition it accounts for close to half the paper. The classical geriatric-syndrome sections — assessment, frailty and falls — are smaller than most candidates expect. Paper III is examined as clinical general medicine seen through a geriatric lens, not as a syndromes paper.



The most-repeated topics

Constipation leads the paper outright with four appearances. Nine further topics have been asked three times each — reversible dementias, sarcopenia, myelodysplastic syndrome, herpes zoster, bone densitometry, hormone replacement therapy, cognitive assessment tools, cardiomyopathy, and the heart-failure cluster. If you prepare nothing else thoroughly, prepare these. The chart counts distinct question slots, so a topic asked once as an essay and twice as a note scores three.



What has changed in the examiner's style

SHIFT 1 · FROM TOPIC LABELS TO NAMED-PATIENT VIGNETTES

Early papers set bare topic labels — "Dementia", "Syncope", "Immobility". Increasingly the examiner builds a patient around the topic: the 75-year-old smoker who became abusive and incoherent on day three of a fever, the 60-year-old on four culprit drugs with recurrent falls, Mr. Alagappan with a PSA of 10 ng/ml, Ms. Ratna asking whether she will inherit her father's Lewy body dementia. Answer in stages — differentials, then investigations, then ward management, then follow-up and counselling.

SHIFT 2 · THE EXAMINER ASKS YOU TO INTERPRET, NOT JUST RECALL

A growing group of questions hands you a test and asks what it means: how the bone mineral density report is interpreted, the FRAX tool, red cell indices, the rise and fall of cardiac enzymes drawn as a graph, BMI cut-offs for Asians. Practise drawing and labelling these — several questions explicitly demand a diagram or a table.

SHIFT 3 · CRITERIA, SCORES AND CLASSIFICATIONS ARE FAIR GAME

Paper III rewards precise instruments: Fried's criteria, clinical frailty scales, GOLD and the ABCD groups, the Mini Nutritional Assessment, FRAX, the WHO classification of myelodysplastic syndrome, the 5 Ms. Learn the actual cut-offs and component lists rather than the general concept.

SHIFT 4 · PRESCRIBING SAFETY HAS BECOME A THEME IN ITS OWN RIGHT

Principles of drug therapy (2013), polypharmacy (2018), drug holiday (2022) and rational prescribing (2023) show a clear and strengthening line of interest, reinforced by drug-induced renal impairment, drug-induced thrombocytopenia and atypical femur fractures due to drugs. Know Beers, STOPP/START, anticholinergic burden and the prescribing cascade, and quote Indian cost and availability where it helps.

Part B · Master question list

All 228 questions ever asked in Paper III, de-duplicated into 181 unique entries and grouped by system and theme rather than by year. Where a topic has been asked more than once, the wording of the fullest version has been kept and the repeat count is shown as a badge. Mark-format badges tell you what length of answer to prepare.

Badge key

ASKED ×3+ · ASKED ×2 · 15-mark essay · 7-mark note

No badge on repeat count means the question has been asked once. High-yield items — asked three or more times — carry a coral badge.

1 Geriatric Assessment, Frailty & Sarcopenia

CGA, functional and frailty assessment, sarcopenia, deconditioning, theories of ageing

1. What is Comprehensive Geriatric Assessment?
   7-mark note
2. Tools of functional assessment / functional assessment in the elderly.
   ASKED ×2 · 7-mark note
3. Describe the pathogenesis, diagnosis, prevention and management of frailty; and its pathophysiology, evaluation and management.
   ASKED ×2 · 15-mark essay
4. Fried's criteria.
   7-mark note
5. Clinical frailty scales.
   7-mark note
6. Sarcopenia in the elderly — etiology, pathogenesis and evaluation.
   ASKED ×3 · 15-mark essay + 7-mark note
7. Write a brief note on deconditioning and its prevention.
   7-mark note
8. Immobility.
   7-mark note
9. Elaborate on the 5 Ms of Geriatrics.
   7-mark note
10. Theories of ageing.
    7-mark note

2 Falls, Gait, Balance & Dizziness

Recurrent falls, risk factors, gait and balance, post-fall syndrome, vertigo, syncope

11. Discuss the approach to recurrent falls in the elderly, with emphasis on prevention and management.
    15-mark essay

12. A 60-year-old known diabetic and hypertensive with coronary artery disease and depression, on oral antidiabetics, diuretics, antihypertensives and antidepressants, presents with recurrent falls over the past week. How will you evaluate and manage the patient?
15-mark essay

13. What are the intrinsic risk factors for falls?
7-mark note

14. Falls prevention strategy in the elderly.
7-mark note

15. Post-falls syndrome.
7-mark note

16. Long lie.
7-mark note

17. Gait and balance assessment.
ASKED ×2 7-mark note

18. What are the phases in human walking? Describe in detail with appropriate diagrams.
7-mark note

19. How will you approach a patient with dizziness?
15-mark essay

20. How would you diagnose and manage a case suspected to have Benign Paroxysmal Positional Vertigo?
ASKED ×2 7-mark note

21. Vestibular rehabilitation.
7-mark note

22. Syncope.
7-mark note

23. Hypersensitive carotid sinus syndrome.
ASKED ×2 7-mark note

3 Cognition, Dementia & Delirium

Dementias, mild cognitive impairment, cognitive tools, BPSD, delirium

24. Discuss dementia in the elderly and its management.
ASKED ×2 20-mark essay + 6-mark note

25. Compare and contrast the clinical features and the areas of the brain involved in the four common dementias.
7-mark note

26. The neurodegenerative disorders associated with Tau, Progranulin or TDP-43 pathology — pathology, clinical presentation, course, prognosis and treatment modalities.
15-mark essay

27. Write a note on Lewy body dementia; and counsel a daughter whose father has been newly diagnosed — etiology, clinical features, treatment, progression, her own risk and whether it can be prevented.
ASKED ×2 7-mark note

28. What is Pick's disease? What is its pathology and treatment?
7-mark note

29. What are the reversible / treatable dementias? How would you investigate and manage them, and what investigations rule them out?
ASKED ×3 7-mark note

30. Mild cognitive impairment — diagnostic criteria, progression and treatment modalities.
15-mark essay

31. Cognitive assessment tools / tests of cognition and rating scales.
ASKED ×3 7-mark note

32. Behavioural symptoms of dementia.
7-mark note

33. Discuss the aetiology, pathogenesis, differential diagnosis and management of acute confusional state in the elderly.
15-mark essay

34. A 75-year-old diabetic and hypertensive smoker developed cough and fever for three days and became incoherent, abusive and drowsy on the third day with stable vitals. What are your differentials and the trigger factors? How would you evaluate and manage him on the ward?
15-mark essay

35. Assessment of delirium.
7-mark note

36. Strategies for prevention of delirium in elderly inpatients.
7-mark note

37. Normal pressure hydrocephalus.
7-mark note

4 Parkinson's Disease & Movement Disorders

Parkinson's disease, parkinsonism-plus, tremor, levodopa and motor fluctuations

38. What are the pathology, pathogenesis and treatment of Parkinson's disease — with emphasis on choice of drug, side effects, newer treatment modalities and the treatment of non-motor symptoms?
15-mark essay

39. Parkinsonism.
6-mark note

40. Drug treatment for Parkinson's disease.
7-mark note

41. Levodopa.
6-mark note

42. Motor fluctuations in Parkinson's disease.
7-mark note

43. Non-motor symptoms of Parkinson's disease.
7-mark note

44. Etiopathogenesis, clinical presentation and management of the Parkinson-plus syndromes in older adults.
15-mark essay

45. Progressive supranuclear palsy — clinical features, management and prognosis.
ASKED ×2 7-mark note

46. Benign essential tremor — clinical presentation, differential diagnosis and treatment.
7-mark note

5 Neurology — Stroke, Coma, Seizures & Neuromuscular

Cerebrovascular disease, coma, epilepsy, cord and nerve disorders, myopathy

47. Discuss coma in the elderly.
20-mark essay

48. Lacunar infarcts.
6-mark note

49. Management of extracranial carotid artery stenosis; and the pros and cons of carotid angioplasty versus carotid endarterectomy in a patient with 80% stenosis of the artery supplying the infarcted territory.
ASKED ×2 7-mark note

50. How would you assess swallow at the bedside in a patient with a recent stroke?
7-mark note

51. Brief note on subdural haematoma.
ASKED ×2 7-mark note

52. Definition, pathophysiology, clinical features, investigations and treatment of epilepsy in the elderly.
15-mark essay

53. Motor neuron disease — presentation, evaluation and treatment modalities.
7-mark note

54. Subacute combined degeneration of the cord — presentation and treatment.
7-mark note

55. Neuritic pain.
6-mark note

56. Inflammatory myopathies.
7-mark note

6 Cardiovascular Medicine

Hypertension, heart failure, arrhythmias, thromboembolism, lipids

57. Discuss the management of hypertension in the elderly.
20-mark essay

58. Isolated systolic hypertension and its management.
ASKED ×2 7-mark note

59. Postural hypotension — evaluation and management.
ASKED ×2 7-mark note + 6-mark note

60. What is the etiology, clinical features, diagnostic evaluation and management of a patient with congestive cardiac failure? What categories of drugs would you use, with their indications and side effect profile? What is your approach to refractory heart failure?
15-mark essay

61. Congestive heart failure — diagnostic evaluation.
7-mark note

62. Refractory cardiac failure.
7-mark note

63. Diastolic dysfunction.
7-mark note

64. Cardiomyopathies — pathophysiology, etiology, clinical features, diagnosis and treatment; including dilated cardiomyopathy.
ASKED ×3 15-mark essay + 7-mark note

65. Infective endocarditis.
7-mark note

66. Atrial fibrillation and its management.
7-mark note

67. Cardiac pacemaker.
6-mark note

68. Write a note on sick sinus syndrome.
7-mark note

69. Explain, with an appropriate diagram, the rise and fall of cardiac enzymes following an acute coronary syndrome.
7-mark note

70. Calcium channel antagonists.
6-mark note

71. Use of statins in the elderly.
7-mark note

72. Causes, treatment and prevention of deep vein thrombosis.
ASKED ×2 7-mark note

73. The causes, diagnosis and management of pulmonary embolism.
15-mark essay

74. Venous thromboembolism and post-operative management of anticoagulation.
7-mark note

75. Anticoagulants in geriatric practice — indications and agents.
7-mark note

7 Respiratory Medicine

COPD, pneumonia, interstitial disease, respiratory failure, sleep apnoea, tuberculosis

76. Describe the risk factors, pathology and clinical features of Chronic Obstructive Pulmonary Disease. How will you differentiate it from bronchial asthma? Discuss the management and complications of COPD.
15-mark essay

77. GOLD criteria for assessment of COPD and treatment options based on this assessment; the new GOLD criteria and the changes from the previous criteria, with a note on the ABCD criteria.
ASKED ×2 7-mark note

78. Metered dose inhalers.
7-mark note

79. Long term oxygen therapy.
7-mark note

80. Discuss the etiology, clinical features and management of community-acquired pneumonia in the elderly.
15-mark essay

81. Interstitial lung disease.
7-mark note

82. Respiratory failure — types, causes and management.
7-mark note

83. Discuss the types, clinical features and management of bronchogenic carcinoma.
15-mark essay

84. Sleep apnoea.
7-mark note

85. Polysomnography.
7-mark note

86. Extrapulmonary tuberculosis.
7-mark note

8 Continence, Bladder & Men's Health

Urinary incontinence, lower urinary tract symptoms, prostate, catheters

87. Classify urinary incontinence and discuss its evaluation and treatment; and discuss the types, diagnosis, management and reversible causes of incontinence in the elderly.
ASKED ×2 15-mark essay

88. Urinary incontinence in males — types, epidemiology, pathogenesis, risk factors, screening, diagnostic evaluation and management.
15-mark essay

89. A 65-year-old normotensive, non-diabetic lady presents with frequent urination, nocturia and intermittent involuntary leakage of urine for six months. What are the causes, and how will you evaluate and manage her?
15-mark essay

90. Bladder retraining protocol.
7-mark note

91. Overactive bladder.
7-mark note

92. Chronic indwelling catheter usage in the elderly.
7-mark note

93. Catheter-associated urinary tract infections — prevention and management.
7-mark note

94. Recommendations regarding treatment of complicated urinary tract infection in males.
7-mark note

95. Algorithm to investigate and treat an elderly male with lower urinary tract symptoms; and the next steps in the investigative and management pathway for a man with LUTS whose PSA is 10 ng/ml.
ASKED ×2 7-mark note

96. Prostate specific antigen.
7-mark note

97. Uroflowmetry.
7-mark note

98. Medical management of benign prostatic hypertrophy.
7-mark note

99. Write a short note on the clinical features, investigation and treatment of obstructive uropathy.
7-mark note

100. What are the causes of erectile dysfunction and what treatment options are currently available?
7-mark note

101. Andropause.
7-mark note

9 Renal, Fluid & Electrolyte Disorders

Acute kidney injury, electrolytes, acid-base balance, renal bone disease

102. Discuss fluid and electrolyte disturbances in the elderly and their management.
15-mark essay

103. Approach to the evaluation and treatment of hyponatremia.
7-mark note

104. Hypocalcemia.
7-mark note

105. Metabolic acidosis.
7-mark note

106. Enumerate the causes and outline the treatment of acute kidney injury in an elderly male; and the differentials and investigations in a 65-year-old male with acute kidney injury.
ASKED ×2 7-mark note

107. Drug-induced renal impairment in the elderly.
7-mark note

108. Renal osteodystrophy — laboratory and radiological abnormalities, and treatment.
7-mark note

109. Anaemia of chronic kidney disease — type, investigation and treatment modalities.
7-mark note

10 Bone, Joint & Rheumatology

Osteoporosis, fracture risk assessment, arthritis, vasculitis, connective tissue disease

110. What is osteoporosis? Describe in detail its risk factors, pathology, clinical features, investigations, management and complications.
ASKED ×2 15-mark essay + 6-mark note

111. Dual energy X-ray absorptiometry — DEXA scan and its clinical uses; and how the bone mineral density test is interpreted, with a note on the FRAX tool.
ASKED ×3 7-mark note

112. FRAX score.
7-mark note

113. A 68-year-old woman had surgery for fracture of the left neck of femur two months ago and asks whether treatment for osteoporosis will benefit her. What is her risk of another fragility fracture? What further tests would you do and what are her treatment options?
7-mark note

114. Atypical femur fractures due to drugs.
7-mark note

115. Selective estrogen receptor modulators.
6-mark note

116. Osteoarthritis management.
6-mark note

117. Late-onset rheumatoid arthritis.
7-mark note

118. Psoriatic arthropathy.
7-mark note

119. Polymyalgia rheumatica.
ASKED ×2 7-mark note + 6-mark note

120. Discuss temporal arteritis.
ASKED ×2 7-mark note

121. Sjögren's syndrome — definition, epidemiology, pathophysiology, presentation and management.
7-mark note

122. Paget's disease.
7-mark note

11 Endocrine & Metabolic

Thyroid disease, glycaemic control, hormone replacement, androgens

123. Treatment of hypothyroidism.
6-mark note

124. Subclinical hypothyroidism.
7-mark note

125. Solitary thyroid nodule.
7-mark note

126. Evaluation and management of hypoglycaemia.
7-mark note

127. SGLT2 inhibitors in type 2 diabetes mellitus.
7-mark note

128. Discuss hormone replacement therapy / hormone replacement in geriatric patients.
ASKED ×3 7-mark note

129. Gynaecomastia.
6-mark note

130. Hirsutism and baldness in elderly women.
7-mark note

12 Gastroenterology & Nutrition

Gastrointestinal bleeding, motility, malabsorption, constipation, nutritional assessment

131. Approach to upper gastrointestinal bleed / haematemesis.
ASKED ×2 7-mark note

132. Gastro-oesophageal reflux disease.
ASKED ×2 7-mark note

133. Gastrointestinal motility disorders in the elderly.
7-mark note

134. Malabsorption / malabsorption syndrome in the elderly.
ASKED ×2 7-mark note

135. Management of chronic constipation, its pharmacological management, and the management of constipation in the elderly.
ASKED ×4 7-mark note

136. How would you assess a patient's nutrition? Write a note on the various methods used and their advantages and disadvantages.
7-mark note

137. What is the Mini Nutritional Assessment score? What are its components, how will you score and interpret it, and what interventions can you suggest?
7-mark note

138. What are the tools used in screening for malnutrition? Enumerate at least three of them, and add a table on Body Mass Index and their cut-offs in Asians.

7-mark note

139. Total parenteral nutrition.

7-mark note

140. Vitamin deficiencies.

7-mark note

13 Haematology & Oncology

Anaemia, myelodysplasia, lymphoma, cancer screening, paraneoplastic disease

141. Anaemia of chronic disease.

7-mark note

142. Blood transfusion.

7-mark note

143. What are the mechanisms of drug-induced thrombocytopenia?

7-mark note

144. Myelodysplastic syndrome — classification, etiology, clinical features, differential diagnosis, investigations and treatment in the elderly.

ASKED ×3 15-mark essay + 7-mark note

145. What are the types of Non-Hodgkin's lymphoma? What are their clinical presentations and prognosis, and what are the principles of therapy?

7-mark note

146. Tumour lysis syndrome — presentation, investigation and treatment.

7-mark note

147. Paraneoplastic syndrome.

7-mark note

148. Hormone therapy in malignancy.

6-mark note

149. Mammography.

6-mark note

150. A 62-year-old woman has a suspicious breast lump on her recent mammogram. What is the ideal approach to a suspicious breast lesion in an older woman?

7-mark note

151. Discuss currently accepted strategies for cancer screening in the elderly.

15-mark essay

14 Infections & Immunity

Atypical presentation, sepsis, vaccination, immunosenescence, specific infections

152. Atypical presentations of infections in the elderly.
6-mark note

153. A 65-year-old male, a known diabetic with chronic kidney disease, is admitted to the emergency ward with altered sensorium, fever, elevated total leucocyte count and hypotension. How will you evaluate and manage this patient?
15-mark essay

154. Approach to pyrexia of unknown origin in older adults.
15-mark essay

155. Herpes zoster infection in the elderly — etiology, clinical features and management.
ASKED ×3 7-mark note

156. Influenza vaccination.
7-mark note

157. Vaccines for the elderly.
7-mark note

158. Immunology and ageing — elaborate on the innate and adaptive immune systems and the impact of ageing, with a note on immunosenescence and cancer.
ASKED ×2 15-mark essay + 7-mark note

159. Clostridium difficile colitis.
7-mark note

15 Sleep, Skin, Special Senses & Environmental

Sleep disorders, pressure ulcers, vision, pruritus, heat-related illness

160. What are the common sleep disorders in the elderly and what is their line of management?
7-mark note

161. Describe the pathophysiology of insomnia and its management.
7-mark note

162. Sleep hygiene.
7-mark note

163. Discuss the assessment and management of chronic pressure ulcers in the elderly, and the prevention of pressure sores.
ASKED ×2 20-mark essay + 7-mark note

164. How would you deal with constant itching in an elderly patient?
7-mark note

165. Visual impairment — enumerate the age-related causes and how you would approach them.
ASKED ×2 7-mark note

166. Macular degeneration — clinical presentation, etiology, diagnosis, treatment and prevention.
7-mark note

167. Heat stroke.
7-mark note

168. Heat exhaustion.
7-mark note

16 Pharmacology, Prescribing & Perioperative Care

Principles of drug therapy, polypharmacy, deprescribing, surgery in old age

169. Describe the principles of drug therapy in the elderly.
15-mark essay

170. Rational drug prescribing in older adults.
7-mark note

171. What do you mean by polypharmacy? How would you prevent it?
7-mark note

172. Drug holiday.
7-mark note

173. Perioperative assessment and management in older people; and a patient admitted with fracture neck of femur after a fall at home, who is diabetic, hypertensive and has COPD and is posted for surgery — what assessments should be done before surgery, and how should he be managed post-operatively and at follow-up?
ASKED ×2 15-mark essay

17 Rehabilitation, Social & End-of-Life Care

Rehabilitation team, therapists, elder abuse, transitional care, palliative care

174. Rehabilitation team.
6-mark note

175. Role of physiotherapy in the elderly.
6-mark note

176. What is the role of an occupational therapist in the life of an elderly person with mobility issues living with an ageing spouse?
7-mark note

177. You are the doctor in charge of a transitional care home accommodating 30 people. What would be your prerequisites for admitting and discharging a patient? Whom would you appoint to manage your patients and what would be their role?
7-mark note

178. Elder mistreatment / elder abuse.
ASKED ×2 7-mark note + 6-mark note

179. Advanced care planning.
7-mark note

180. End of life care issues.
7-mark note

181. Enumerate and expand the principles of palliative care that one should use to treat a patient with end-stage cardiac failure. What are the social issues one should address in this condition?

15-mark essay

Part C · Ebenezer's bank — Paper III topics not yet asked

Every Paper III-relevant heading in Ebenezer's Geriatric Question Bank was cross-checked against the 181 questions in Part B. The topics below appear in the bank but have **never** been set in a Paper III university paper. They are the highest-value gap list you have: a compiler thought them worth including, and the examiner has not yet used them.

Cardiovascular

1. Cardiac cycle, cardiac output and the Frank–Starling law in cardiac failure.
2. Aortic stenosis, aortic sclerosis and mitral regurgitation in the elderly.
3. Unstable angina — pathology, pathophysiology and management.
4. Beta blockers in heart failure.
5. Accelerated hypertension and hypertensive emergency.
6. Secondary hypertension — evaluation and management.
7. Ankle brachial index and toe pressures — indications and interpretation.
8. Head-up tilt test — indication, procedure and interpretation.
9. Complete heart block and tachyarrhythmias in the older adult.
10. Chronic pulmonary thromboembolism.
11. In-stent stenosis — etiopathogenesis and management.
12. Lifestyle modification to reduce cardiovascular risk.
13. CHADS2-VASc score — uses and clinical significance.
14. Drug-eluting stents — pros and cons.
15. Newer cardiac biomarkers in myocardial infarction.

Respiratory

16. ARDS — recognition and management.
17. Pulmonary nodule — approach, investigation and treatment.
18. Pleural effusion — approach, evaluation, differential diagnosis and management.
19. Hospital-acquired pneumonia and aspiration pneumonia.
20. Senile emphysema.
21. Role of CPAP in diseases of the elderly.
22. Pneumococcal vaccination.
23. DOTS, MDR-TB and XDR-TB — regimens and dosing.
24. Diffuse parenchymal lung disease — classification, approach and treatment.

Neurology & Psychogeriatrics

25. Complex partial seizures.
26. Electroconvulsive therapy in the elderly.

27. Cluster headache and tension headache.
28. Wernicke encephalopathy.
29. Transient ischaemic attacks.
30. Thrombolysis in stroke.
31. Stroke rehabilitation.
32. Drug-induced parkinsonism.
33. Multiple system atrophy — clinical features, types, diagnosis and treatment.
34. Hypoxic ischaemic encephalopathy.
35. Traumatic brain injury — post-acute management.
36. Wallenberg syndrome.
37. Approach to meningitis in the elderly.
38. Restless leg syndrome — risk factors, criteria and management.
39. Peripheral neuropathy — common causes, evaluation and management.
40. Ring-enhancing lesion on CT brain.
41. Myasthenia gravis — presentation, laboratory evaluation and treatment.
42. Geriatric depression scale and depression in late life.
43. Suicide risk assessment in older adults.
44. Late-onset schizophrenia and late-life psychotic disorders.
45. Diogenes syndrome.

Gastroenterology, Renal & Metabolic

46. Drug-induced jaundice.
47. Hepatitis B and C infection — investigation and treatment.
48. Ascites — causes and management; liver transplantation.
49. Chronic liver disease and hepatic encephalopathy.
50. Helicobacter pylori and its clinical relevance.
51. Diabetic ketoacidosis and hyperosmolar non-ketotic coma.
52. Alpha glucosidase inhibitors and newer oral hypoglycaemic agents.
53. Diabetic foot syndrome.
54. Ezetimibe — indications, mode of action and side effects.
55. Acute glomerulonephritis and acute pyelonephritis.
56. Chronic kidney disease — conservative management in the elderly.
57. Renal replacement therapy — criteria, haemodialysis and peritoneal dialysis.
58. Hyperkalemia and hypercalcemia — investigation and treatment.
59. Types of shock and septic shock — etiopathogenesis and diagnosis.
60. Faecal incontinence — evaluation and management.

Haematology, Oncology & Infection

61. Multiple myeloma and plasma cell dyscrasias.
62. Chronic lymphocytic leukaemia — classification and treatment.
63. Bone marrow transplant.
64. PET scanning in the older adult.
65. Methotrexate — major side effects of low-dose therapy.
66. Prostate cancer — to screen or not to screen.
67. Fever of unknown origin — evaluation of prolonged fever.
68. Malaria and cerebral malaria — drugs, prophylaxis and management.
69. Leptospirosis — investigation and management.
70. Sterile pyuria and asymptomatic bacteriuria.
71. COVID-19 in the older adult.

Rheumatology, Endocrine & Miscellaneous

72. Hashimoto's thyroiditis.
73. Gout — management in the older adult.
74. Vertebral secondaries with unknown primary.
75. Diffuse idiopathic skeletal hyperostosis.
76. Dermatomyositis and POEMS syndrome.
77. Serotonin syndrome and neuroleptic malignant syndrome.
78. Rhabdomyolysis.
79. Tinnitus.
80. Chronic fatigue syndrome and inanition.
81. Anorexia of ageing.
82. Interleukin-6, telomeres and sirtuins in ageing.

Social, Ethical & Rehabilitative

83. Caregiver burden and how to manage it.
84. Alcoholism in the elderly.
85. Informed consent and decisional capacity.
86. Euthanasia and physician-assisted suicide.
87. Breaking bad news.
88. Assistive devices, mobility aids and artificial limbs.
89. Community-based rehabilitation.
90. NPHCE and the National Policy on Older Persons.
91. Home-based care and long-term care services.
92. Hospice and respite care; bereavement.

WHY THIS LIST MATTERS

92 topics sit in the Ebenezer bank without ever having been examined in Paper III. Several of them — thrombolysis in stroke, stroke rehabilitation, depression and the Geriatric Depression Scale, multiple myeloma, hyperkalaemia and hypercalcaemia, faecal incontinence, and conservative management of chronic kidney disease — are so central to modern geriatric practice that their continued absence is itself a prediction signal. Note in particular that Paper III has never once set a psychogeriatric short note, despite psychiatry sitting squarely in the syllabus.

Part D · Predicted questions

Predictions are built from three signals: how often a topic has been asked, how long it has been since it last appeared, and whether the recent literature has given the examiner a fresh angle. Probability labels are relative, not absolute — treat "very high" as a topic you cannot afford to walk in without, and "moderate" as one worth a prepared skeleton answer.

Paper III · most likely 15-mark essays

Predicted essay	Odds	Reasoning
Comprehensive Geriatric Assessment — domains, tools, evidence base and application in Indian practice.	Very high	Asked only as a 7-mark note in 2013 despite being the organising concept of the whole paper. Paper IV has twice used it as an essay; Paper III never has.
Urinary incontinence — classification, evaluation, reversible causes and management, including incontinence in the male.	Very high	Four appearances, three of them essays (2011, 2016, 2019). The single most reliable Paper III essay stem and unasked since 2019.
Delirium / acute confusional state — aetiology, differentials, prevention and ward management.	Very high	Three essays in scenario form (2016, 2017, 2019) plus notes on assessment and prevention. The examiner clearly favours the ward-scenario framing.
Frailty and sarcopenia — overlapping constructs, assessment instruments and management.	High	Frailty has four entries and sarcopenia three; both were last set around 2019–20 and the AWGS 2019 and Clinical Frailty Scale updates give a fresh angle.
Falls — approach to recurrent falls with drug review, assessment and prevention.	High	Essay in 2011 and as a polypharmacy scenario in 2017. Eight sessions have passed since the last dedicated essay.
Parkinson's disease — motor and non-motor management, and the parkinsonism-plus syndromes.	High	The most frequently examined single disease in Paper III with nine separate entries. Essays in 2012 and 2023.
Dementia — comparison of the four common dementias with investigation of reversible causes.	Moderate	Essay in 2009; reversible dementias recurred in October 2025. A combined stem is the natural next version.
Perioperative assessment of the older surgical patient.	Moderate	Essay in 2022 and again in October 2025 — back-to-back appearances suggest the examiner currently favours it.

Paper III · most likely 7-mark notes

Predicted note	Odds	Reasoning
Cognitive assessment tools — MMSE, MoCA, AMT and clock drawing	Very high	Three appearances (2013, 2017, 2021) — the most repeated short note in the paper.
Constipation in the elderly — evaluation and stepped management	Very high	Four appearances across 2011, 2013, 2017 and 2019 — the highest-frequency short note in Paper III.
DEXA and bone mineral density interpretation with the FRAX tool	Very high	Four entries including October 2025; interpretation-style framing is now the examiner's preference over simple description.
Herpes zoster in the elderly and zoster vaccination	High	Three appearances (2011, 2013, 2023); the recombinant zoster vaccine is the untouched modern angle.

Predicted note	Odds	Reasoning
Hormone replacement therapy	High	Three appearances (2013, 2015, 2020) on a roughly five-year cycle.
Gait and balance assessment	High	Two appearances (2017, 2020) and pairs naturally with the falls theme.
Vaccination in older adults beyond influenza	High	Influenza and generic vaccines have appeared; pneumococcal, zoster and RSV vaccines have never been set in Paper III.
Polypharmacy, deprescribing and the drug holiday	High	Polypharmacy 2018, rational prescribing 2023, drug holiday 2022. Beers and STOPP/START criteria remain unasked by name.
Myelodysplastic syndrome — classification and management	Moderate	Three entries; last set as a full essay in 2021.
Deep vein thrombosis and anticoagulation in geriatric practice	Moderate	DVT twice, VTE prophylaxis once, anticoagulants once; DOAC-era content is overdue.
Pressure ulcers — staging, prevention and stage-wise treatment	Moderate	Essay in 2009 and a note in 2011, then nothing — a fourteen-year gap.
Sleep apnoea and polysomnography	Moderate	Sleep apnoea 2022, polysomnography 2019; the pair runs on a short cycle.

A model predicted Paper III

If you want a single paper to mock-write against, this is the twelve-question set the frequency and recency analysis produces.

	Question	Marks
I.1	An 80-year-old admitted after two days of fever becomes agitated and disoriented on the ward. Discuss the aetiology, differential diagnosis, assessment and prevention of delirium, and outline your management.	15
I.2	Urinary incontinence in the older adult — classification, reversible causes, evaluation and management, with specific reference to incontinence in the male.	15
II.1	Constipation in the elderly — evaluation and stepped pharmacological management.	7
II.2	Reversible dementias — causes and the investigation panel.	7
II.3	Cognitive assessment tools — MMSE, MoCA and AMT, with their limitations in Indian practice.	7
II.4	Interpretation of bone mineral density and the FRAX tool.	7
II.5	Deprescribing — Beers, STOPP/START and anticholinergic burden.	7
II.6	Sarcopenia — AWGS 2019 criteria, assessment and management.	7
II.7	Herpes zoster in the older adult, including recombinant zoster vaccine.	7
II.8	Hyponatraemia and SIADH — evaluation and safe correction.	7
II.9	Heart failure with preserved ejection fraction — diagnosis and four-pillar therapy.	7
II.10	Gait and balance assessment — bedside and instrumented tests.	7

Cross-paper watchlist — Papers I, II and IV

Paper III does not sit in isolation: the same themes recirculate across papers with a different emphasis. These are the highest-probability areas for the other three papers based on the same frequency and recency analysis.

Paper I — Applied Basic Sciences

1. Anatomy: conduction system and blood supply of the heart; bronchopulmonary segments; innervation of the urinary bladder; dermatomes; portocaval anastomosis.
2. Physiology: ageing changes in the heart; Frank–Starling law; lung volumes; the nephron and sites of electrolyte exchange.
3. Biochemistry: gout and purine metabolism; vitamin D; lipid metabolism; bone turnover markers.
4. Pharmacology: pharmacokinetic and pharmacodynamic changes with ageing; oral anticoagulants; drugs in benign prostatic enlargement.
5. Pathology: iron deficiency anaemia and red cell indices; fatty liver; Lewy bodies; inflammaging.
6. Microbiology: inflammatory markers in ageing; pneumococcal vaccines; antibiotic choice in lower urinary tract infection.

Paper II — General Medicine including Psycho-Geriatrics

1. Behavioural and psychological symptoms of dementia — types and management.
2. Depression in older adults and antidepressant choice.
3. Parkinson's disease — motor and non-motor symptoms and drug side effects.
4. COPD by GOLD criteria and asthma by GINA guidelines.
5. Hypercalcaemia work-up; hyperkalaemia in the diabetic.
6. Late-onset rheumatoid arthritis versus early-onset disease.
7. Recurrent urinary tract infection and urinary incontinence in the male.
8. Vertigo, chronic liver disease and peripheral neuropathy.

Paper IV — Social and Preventive Geriatrics

1. NPHCE, NPOP and the social security system for older people in India.
2. Elder abuse, advance directives and the living will.
3. Comprehensive Geriatric Assessment and integrated care of older people.
4. Vaccination schedules and immunosenescence.
5. Rehabilitation team composition and the role of each therapist.
6. Palliative and end-of-life care; breaking bad news.
7. Falls prevention including sensor devices.
8. Cancer screening strategies and stopping rules.

FINAL NOTE ON USING PREDICTIONS

Prediction is a way of ordering revision, not of restricting it. Constipation, reversible dementias and the cognitive assessment tools are near-certainties over any two-session horizon, but every paper carries three or four genuinely novel questions — the 5 Ms of Geriatrics, psoriatic arthropathy and atypical femur fractures were all new in the last two sessions. Prepare the high-frequency core to essay depth, keep a one-page skeleton for everything in Part C, and you will have something structured to write for any question on the paper. One practical caution: December 2024 Paper III is missing from the source set, so a topic that looks overdue here may in fact have been cleared in that session.

MD Geriatric Medicine — High-Yield Examination Answers

Set 1: Assessment, Function, Cognition, Capacity and Geriatric Syndromes

Primary source: Pathy's Principles and Practice of Geriatric Medicine, 6th edition; supported by Kane's Essentials of Clinical Geriatrics and current international and Indian guidelines.

QUESTION 1

Comprehensive Geriatric Assessment — What It Is and Its Usefulness in Clinical Practice

A. 20 High-Yield Points

Definition and Concept

1. **Comprehensive geriatric assessment (CGA)**, also called **multidimensional geriatric assessment**, is a **diagnostic process, usually interdisciplinary**, that determines an older person's **medical, psychosocial, functional and environmental** resources and problems.
2. Its stated objective is to **develop an overall plan for treatment and long-term follow-up**. An assessment **without a linked care plan is NOT a CGA** — a favourite examination point.
3. It **differs from a standard medical evaluation** in three ways: it targets older people with complex problems, it **emphasises functional status and quality of life**, and it uses **interdisciplinary teams with quantitative scales**.
4. A purely psychosocial or environmental home visit **without a medical evaluation** also does not qualify as CGA, because multidimensionality is incomplete.
5. **Historical origin: Dr Marjory Warren**, London infirmary, **late 1930s**. Through evaluation, mobilisation and rehabilitation she got long-bedfast patients out of bed and home, and advocated **assessment and rehabilitation before any long-term institutionalisation**.

Rationale — Why in the Elderly? (five textbook reasons)

6. **Multi-morbidity and complexity** — systematic assessment helps set priorities among multiple conditions.
7. **Unrecognised problems** — many problems are never reported, often because they are **falsely thought to be non-modifiable effects of ageing**.
8. **Chronic conditions** — a diagnosis without information on **functional relevance** is of limited therapeutic value.
9. **Social and environmental interaction** — home layout and social network determine whether a person can stay at home.
10. **Functional status** — the main goal of geriatric medicine is to **prevent or delay functional decline**; benefit is greatest when intervention occurs **early** in the path from robustness to frailty to disability.

Components — What Is Assessed

11. CGA begins with a **functional status "review of systems"** built on three measures: **frailty, basic ADLs, and IADLs**.
12. **Principle of hierarchy / parsimony:** if a patient is **severely ADL-dependent, do not assess frailty**; if the patient is **robust or pre-frail, do not assess basic ADLs**.
13. Frailty is measured by two models — the **phenotype model** (Fried phenotype, Frailty Trait Scale, FRAIL index) and the **cumulative deficit model** (Frailty Index, Clinical Frailty Scale).
14. **Dimensions and tools (Table 96.1):** BADL — **Katz, Barthel, Lawton PSMS**; IADL — **Lawton, OARS**; Social — **Lubben Social Network Scale**; Affective — **Geriatric Depression Scale, Zung**; Cognitive — **MMSE (Folstein), Kahn MSQ**; Gait and balance — **Tinetti, Get-Up-and-Go**; Nutrition — **MNA**; Senses — **whispered voice test, Snellen**; Oral — **GOHAI**.
15. Two screens are **mandatory** in any older person with functional disability: **cognitive impairment and depression**.

Process in Office and Clinic Practice

16. **Step 1 — set priorities:** work first on the problem that **most bothers the patient**, or the problem on which resolution of others depends (**alcoholism, depression**).
17. **Step 2 — task and symptom analysis:** define the exact nature of the impairment and correlate it with **new drugs or dose changes (including over-the-counter), change in living conditions, and intercurrent acute illness**. Then examination, laboratory tests, therapist and social-work input, and a **long-term follow-up / case-management plan**.

Settings and Team

18. **Settings:** home (preventive visits), office and outpatient clinic, **day hospital**, **acute care for the elderly (ACE) unit**, post-acute rehabilitation unit, nursing home (**Resident Assessment Instrument**), and hospital-at-home programmes. Inpatient and day-hospital settings give intensity and speed for the frail; outpatient settings are cheaper.
19. **Team (minimum):** physician, nurse, social worker, physiotherapist, occupational therapist; **optimally** dietician, pharmacist, ethicist, home-care specialist. The **interdisciplinary team conference is critical** — present in most successful trials; teams mature through **forming, storming, norming, performing**.

Evidence of Usefulness — The Exam Core

20. **Proven benefits:** CGA **improves function and survival while reducing healthcare utilisation and costs**. Supporting data:

- **Improved diagnostic accuracy — 1 to more than 4 new diagnoses per patient**.
- **1993 Stuck/Rubenstein meta-analysis (28 trials, ~10,000 patients):** mortality down **22%** (hospital-based, 12 months) and **14%** (all programmes); living at home up **47%** (hospital-based) and **26%** (all); readmissions down **12%**; cognitive improvement up **47%**; physical function up **72%** on hospital units.
- **2011 Cochrane meta-analysis:** inpatient CGA increases the likelihood of being **alive and in one's own home**, with potential cost reduction.
- **Better prescribing** — CGA reduces unnecessary and inappropriate medication use.
- **Targeting matters:** best results when patients are at **high risk of deterioration yet retain rehabilitation potential** — exclude those **"too well or too ill."**
- **Programmes with hands-on care and/or long-term follow-up** outperform purely consultative programmes.
- **Weak areas:** outpatient CGA showed **no mortality benefit** in one meta-analysis; **preventive home visits are not recommended routinely** (benefit only with CGA-based extended follow-up in relatively well elders; **number needed to visit about 40** to prevent one nursing-home admission).
- **Oncology (SIOG task force):** CGA detects many problems missed by regular assessment and is associated with **fewer treatment side effects**.

B. Latest Updates (2023–Present)

1. German DGG S3 Guideline on CGA in Hospital (2024) — the first S3-level CGA guideline. Twelve evidence-based and eight consensus-based recommendations. Strongest evidence in **acute geriatric, oncology and orthopaedic/trauma wards**; weaker in emergency departments and general surgery. Core elements: **minimum CGA duration of 15 minutes** and **six core dimensions — self-help ability, mobility, cognitive function/delirium, affect, nutrition and social situation**; the **G8 questionnaire** is recommended for screening mainly in oncology.

2. Italian National CGA Guideline (2024) — 25 scientific societies plus the National Institute of Health, GRADE methodology. Sixteen recommendations from **117 studies** across primary care, clinics, emergency departments, hospital wards and nursing homes. CGA **reduces hospitalisation, mortality, institutionalisation and delirium risk**, improves prescribing appropriateness, and **maintains functional activities**. The **Multidimensional Prognostic Index (MPI)** is specifically recommended to predict negative outcomes.

3. Japanese CGA-Based Healthcare Guidelines 2024. The main objective of CGA is stated to be **personalised care through holistic management plans and targeted interventions — not merely status evaluation or prognostication**.

4. ASCO Geriatric Oncology Guideline Update (2023). Geriatric assessment should be used in **all patients over 65 with cancer**; all older adults with GA-identified deficits should receive **GA-guided management**, and the **Practical Geriatric Assessment (PGA)** is the recommended tool. Evidence base: **GAP70+** and **GAIN** trials.

5. ASCO Global (Resource-Stratified) Geriatric Assessment Guideline (2025) — adapts the 2023 guideline for **resource-limited settings** using ADAPTE methodology. Directly relevant to Indian practice.

6. Cochrane 2022 — Community CGA. Twenty-one studies, 7,893 participants, 10 countries, evaluating domiciliary CGA and CGA in general practice, with outcomes of death, nursing-home admission, unplanned hospital admission and emergency visits.

7. Meta-analysis of CGA in acute hospitalisation (2025) — CGA **may improve short-term survival and reduce readmission rates** after acute hospitalisation.

8. Perioperative CGA is now mainstream. The **2023 World Society of Emergency Surgery guidelines** advocate frailty assessment and multidisciplinary geriatric-surgical management in older trauma patients; the UK **NELA Best Practice Tariff (April 2023)** mandates perioperative team involvement in frailer laparotomy patients. CGA outperforms traditional preoperative risk models in predicting **postoperative delirium, infection and prolonged stay**.

9. Indian context. Evidence is predominantly from high-income countries and hospitalised patients; research on CGA in primary care and community settings in low- and middle-income countries is lacking. Under **NPHCE**, community services are delivered by **ASHA workers** with clinics at PHCs, but implementation is inconsistent. Short culturally adapted tools such as **EHSAS** are being validated for rural India because full CGA is impractical with the available workforce.

C. Current Practice Update

Aspect	Pathy's 6th ed. (2022)	Current position (2023–2026)
Standardisation	Deliberately broad; "no single optimal blueprint"	**Minimum standards defined** — German S3: at least 15 minutes, six mandatory core domains
Prognostic tool	Many scales listed; none preferred	**MPI specifically recommended** (Italian guideline)
Oncology tool	SIOG could not recommend any specific tool	**PGA specifically recommended** (ASCO 2023)
Oncology strength	CGA "possibly" improves survival	**GA mandatory at 65 and above**; GA-guided management proven to cut severe chemotoxicity
Outpatient CGA	"Less impressive"; no mortality benefit	Italian guideline **supports** primary-care CGA for reducing hospitalisation and institutionalisation
Preventive home visits	Cannot be recommended as routine practice	**Largely unchanged** — Cochrane 2022 still shows uncertain benefit
Perioperative CGA	Not covered	Now **embedded in surgical pathways** (NELA BPT, WSES 2023, POPS model)
Emergency department	Not addressed	Recognised as important but **evidence still weak** (German S3)

D. Exam One-Liners

- **CGA without a care plan and follow-up is not a CGA.**
- **Marjory Warren, 1930s London** — mother of CGA and of British geriatrics.
- **Function is the common language of geriatrics; assessment is its core method.**
- **Hierarchy rule:** ADL-dependent, skip frailty testing; robust or pre-frail, skip basic ADL testing.
- **Targeting rule:** benefit is greatest in those **"not too well, not too ill"** — high risk with rehabilitation potential.
- **Hands-on care plus long-term follow-up** separates effective from ineffective programmes.
- **1993 meta-analysis:** mortality down 22%, living at home up 47%, physical function up 72% on hospital units.
- **Two mandatory screens** in functional disability: **cognition and depression**.
- **German S3 six core domains:** self-care, mobility, cognition/delirium, affect, nutrition, social situation.
- **ASCO 2023:** every cancer patient 65 and above gets a geriatric assessment, using the **PGA**.
- **Italian 2024:** use the **MPI** for prognosis.
- **India:** CGA delivery constrained by workforce — hence short screening tools within **NPHCE**.

QUESTION 2

Tools of Functional Assessment

A. 20 High-Yield Points

Framework

1. **Functional assessment** measures **what a person actually does in daily life**, not merely what diseases they have. Tools exist to make this measurement **standardised, reproducible and comparable across settings**.
2. **Purpose of using standardised tools:** they allow **information exchange between acute, rehabilitation and community settings**, permit **measurement of effectiveness**, foster **comparison of treatments**, and **minimise repeated collection of identical information** by different team members.
3. **Three levels of measurement (ICF, 2001): impairment** (body function or structure), **activity limitation** (difficulty executing a task), and **participation restriction** (problems in involvement in life situations). Different tools address different levels.
4. **Three functional core measures** in CGA: **frailty, basic ADLs, and IADLs**. The **hierarchy rule** determines which to use — severely ADL-dependent patients are not tested for frailty; robust patients are not tested for basic ADLs.
5. **Two methods of data capture: report** (by the patient or a proxy — tells you what they do under real living conditions) and **performance testing** (tells you what they can do under standard conditions). **Discrepancies between the two are common and informative.**

Basic ADL Tools

6. **Katz Index (1963)** — six items: **bathing, dressing, toileting, transferring, continence, feeding**. Graded independent/dependent, letters A to G. The classic **hierarchy of ADL loss**.
7. **Barthel Index (1965; modified 1988)** — ten items: feeding, bathing, grooming, dressing, bowels, bladder, toilet use, transfer, mobility, stairs. Scored **0–100** (or 0–20). It is an **observation of what the patient does**, and is the **RCP/BGS-endorsed ADL scale**, but has a **ceiling effect in ambulatory patients**.
8. **Lawton Physical Self-Maintenance Scale (PSMS, 1969)** — six items: **toileting, feeding, dressing, grooming, physical ambulation, bathing**. Each graded on **five levels**, dichotomised to a total of **0–6** (6 = independent). Published in the same paper as the Lawton IADL scale.

IADL Tools

9. **Lawton IADL scale (1969)** — eight items: **telephone, shopping, food preparation, housekeeping, laundry, transport, medications, finances**. IADLs need "instruments," and are the **first functions to be lost**.
10. **OARS Multidimensional Functional Assessment Questionnaire** — assesses IADLs alongside social and economic resources; widely used in surveys.
11. **Clinical rule:** if difficulty is reported, always ask **"how does she manage?"** — is the task neglected, or is someone else doing it?

Mobility, Gait and Balance Tools (high-yield numbers)

12. **Gait speed** over a 4-metre track: **below 0.6 m/s is abnormal**; **0.6–0.8 m/s is high-risk**. Each **0.1 m/s fall in gait speed equals a 7% rise in fall risk**.
13. **Timed Get-Up-and-Go (Mathias, Nayak and Isaacs, 1986): more than 12 seconds indicates high fall risk** (sensitivity 77%, positive predictive value 93% in the community).
14. **Tinetti Performance-Oriented Mobility Assessment (1986):** 14 tasks, **score below 24 out of 28 indicates fall risk**; takes 10–15 minutes.
15. **Short Physical Performance Battery (SPPB):** combines gait speed, **30-second chair stand** and the **4-stage balance test** (each position held **10 seconds**). **Berg Balance Scale:** 14 items, 0–56 (0–20 wheelchair-bound, 21–40 walks with assistance, 41–56 independent).

Composite, Global and Domain-Specific Tools

16. **Functional Independence Measure (FIM)** — scores **18 functions** across self-care, sphincter control, mobility, locomotion, communication and social cognition; the standard rehabilitation outcome measure.
17. **RCP/BGS endorsed scales (Table 98.3):** ADL — **Barthel Index**; vision, hearing and communication — **Lambeth Disability Screening Questionnaire**; memory — **Abbreviated Mental Test (10 items)**; depression — **Geriatric Depression Scale (30-item, 15-item short form)**; morale — **Philadelphia Geriatric Center Morale Scale**.
18. **Other domain tools (Table 96.1):** nutrition — **Mini Nutritional Assessment (MNA)**, SNAQ; special senses — **whispered voice test, Hearing Handicap Inventory, Snellen chart**; oral health — **GOHAI**; social network — **Lubben Social Network Scale**; cognition — **MMSE**; mood — **GDS, Zung**.
19. **Frailty tools: Fried phenotype** (weight loss, exhaustion, weakness, slow gait, low activity); **FRAIL scale** (Fatigue,

Resistance, Aerobic, Illnesses more than 5, Loss of weight 5%) — answerable in 15–30 seconds; **SOF index** (weight loss, cannot rise from chair 5 times without arms, reduced energy); **Clinical Frailty Scale** (9 points, seconds to apply); **Frailty Index** (30 or more deficits, continuous 0–1).

20. **Screening questions usable at the bedside:** "*Do you need help at home?*" (function) · "*Do you have trouble walking or lose your balance? Have you had a fall? Are you afraid of falling?*" (mobility) · "*Are you sad?*" (mood) · "*Have you lost weight?*" (nutrition) · "*Are you on seven or more medicines?*" (polypharmacy) · "*Do you wet yourself?*" (continence).

B. Master Comparison Table

Domain	Screening tool	Assessment tool	Key threshold
Frailty	FRAIL scale, Clinical Frailty Scale	Fried phenotype, Frailty Index	3 or more of 5 = frail
Basic ADL	—	**Katz, Barthel, Lawton PSMS**	Barthel below 60 = dependent
IADL	—	**Lawton IADL, OARS**	Loss precedes ADL loss
Gait / mobility	Gait speed, TUG	Tinetti POMA, SPPB, Berg	Gait below 0.8 m/s; TUG above 12 s; Tinetti below 24
Cognition	Mini-Cog, AD8	MMSE, MoCA, SLUMS	MMSE below 24
Mood	PHQ-2	GDS-15/30, PHQ-9, CSDD	GDS-15 of 5 or more
Nutrition	MNA-SF, SNAQ	MNA full, BMI, weight loss	MNA-SF 11 or less
Vision	Snellen	Formal ophthalmic review	Below 6/18
Hearing	Whispered voice test	Audiometry, HHIE	Fails at 2 feet
Social	Lubben Social Network Scale	OARS social section	LSNS-6 below 12
Rehabilitation outcome	—	**FIM**	18 items

C. Latest Updates (2023–Present)

1. WHO ICOPE — from deficit to capacity. The ICOPE 5-step pathway (screen, assess, intervene, follow up, community engagement) screens **six domains — mobility, cognition, vitality/nutrition, vision, hearing and psychological well-being** — either by a professional or by self-assessment through the **ICOPE Monitor** app. Step-2 tools are named explicitly: **SPPB for mobility; Mini-Cog, MoCA, MMSE or GPCOG for cognition; MNA, DETERMINE, MUST or SNAQ65+ for nutrition; PHQ-9 for mood; pure tone and speech audiometry for hearing**.

2. Minimum data sets are now mandated. The **German S3 guideline (2024)** requires **six core dimensions in at least 15 minutes**; the **Italian guideline (2024)** recommends the **Multidimensional Prognostic Index (MPI)** for prognosis; **ASCO 2023** mandates the **Practical Geriatric Assessment (PGA)** in oncology. The trend is **domain-mandated but tool-agnostic**.

3. Gait speed is being repositioned as the single best functional marker. A 2025 Age and Ageing study of about 3,600 older adults found that among all components of frailty and sarcopenia, **low physical performance (slow gait speed) was the best single predictor of incident basic and instrumental ADL disability**; sarcopenia as a whole construct was not associated with increased disability risk. **EWGSOP2 defines low physical performance as gait speed of 0.8 m/s or less.**

4. Automated and digital tools. The **electronic frailty index (eFI2)** is replacing the original eFI in English primary care from 2025, allowing **population-level functional risk stratification from routine records**. mHealth and wearable-based functional assessment gives **objective, quantitative data** in place of subjective observer-rated scales, but a 2024 systematic review found **high risk of bias and minimal integration into daily practice**.

5. Diagnostic accuracy data for screening instruments. In emergency-department settings, seven screens pooled across 1,663 participants gave **sensitivity 0.85 and specificity 0.77** against full CGA; **PRISMA-7 was the most sensitive (0.84–0.98)** and the **Clinical Frailty Scale the most specific**, both with AUROC around 0.90.

6. Indian use. LASI applies Katz-type ADL and Lawton-type IADL items; grip strength was the **most discriminant frailty component in Indian men (79.1%)** and physical activity in women (81.6%). **AWGS (not EWGSOP2) cut-offs** should be used for sarcopenia-related functional measures in Indians, and **BIA rather than DXA** is the practical mass-measurement modality.

D. Current Practice Update

Aspect	Pathy's 6th ed. (2022)	Current position (2023–2026)
Choice of tool	Broad menu; "no consensus on the best scale"	**Domain mandated, tool still flexible**, but specific tools named per setting
Gait speed cut-off	Below 0.6 m/s abnormal; 0.6–0.8 high-risk	**0.8 m/s or less** operative threshold (EWGSOP2/AWGS); best single predictor of disability
Who administers	Trained professional or therapist	**Self-assessment and digital screening validated**; community health workers in LMICs
Data source	Interview and bedside observation	**Automated extraction from electronic records** (eFI2); wearables and sensors
Framing	Deficit-based (ADL/IADL loss)	Complemented by **capacity-based** framing (WHO intrinsic capacity)
Sarcopenia link	Not integrated	Functional tools now feed directly into **sarcopenia case-finding** (SARC-F, grip strength, gait speed)

E. Exam One-Liners

- **Katz and Barthel measure ADL; Lawton measures IADL; Tinetti and TUG measure mobility; MNA nutrition; GDS mood; MMSE cognition; Lubben social.**
- **Barthel is what the patient does, not what they can do** — and has a **ceiling effect** in ambulant patients.
- **FIM has 18 items** across six areas and is the rehabilitation standard.
- **TUG above 12 seconds; Tinetti below 24 of 28; gait speed below 0.6 m/s (Pathy) or 0.8 m/s (EWGSOP2).**
- **0.1 m/s slower gait equals 7% higher fall risk.**
- **PSMS has grooming and ambulation; Katz has continence and transferring** — the classic distinguishing point.
- **Report tells you what they do; performance tells you what they can do** — measure both.
- **WHO ICOPE six domains:** mobility, cognition, vitality, vision, hearing, psychological.
- **A tool is only useful if its result changes the care plan.**

QUESTION 3

Functional Assessment in the Elderly

A. 20 High-Yield Points

Why Function Matters

1. **Functional assessment** means measuring **what the person actually does in daily life**. In geriatrics, **function is the common language** and assessment is the core method.
2. In an older person, **function predicts outcome better than diagnosis**. There is **no direct relationship between disease and functioning** — the same illness produces very different disability in different people, so function must be measured separately.
3. Functional decline results from **medical, functional, psychological, social and environmental** risk factors acting together. Hence assessment must be **multidimensional**.
4. A **baseline functional measure is essential** so that progress can be monitored and the effect of treatment or rehabilitation judged. **Reassess at least weekly during rehabilitation.**
5. Three levels are assessed: **impairment** (the deficit) → **activity restriction** (ADL/IADL loss) → **participation restriction** (loss of social role). Participation restriction is **unique to the individual** and depends largely on the ability to adapt.
6. Mobility must always be tested — **about half of people aged 85 and over report difficulty walking**, and gait disorders threaten independence and cause falls.

The Core Measures

7. The functional "review of systems" rests on three measures: **frailty, basic ADLs, and IADLs**.
8. **Principle of hierarchy:** if the patient is **severely ADL-dependent, do not test frailty**; if **robust or pre-frail, do not test basic ADLs**. Match the depth of assessment to the patient and the setting.
9. **Basic ADLs (personal care):** bathing, dressing, toileting, transferring, continence, feeding, grooming — **Katz, Barthel, Lawton PSMS**.
10. **IADLs (tasks needing instruments):** cooking, shopping, housework, laundry, telephone, transport, money, medicines — **Lawton IADL, OARS**.
11. **IADLs are lost first, ADLs later**, so IADL loss is the earlier warning sign. Always ask **"how does she manage?"**
12. **Always record premorbid function as well as present function**, especially after acute illness, a fall or hospital admission.

Domains to Cover

13. **Mobility, gait and balance:** gait speed (**below 0.6 m/s abnormal; 0.6–0.8 high-risk**), **TUG above 12 seconds**, **Tinetti below 24 of 28**, 30-second chair stand, 4-stage balance test, SPPB, Berg Balance Scale.
14. **Two screens are mandatory** in any older person with functional disability: **cognition** (MMSE) and **depression** (GDS or Zung).
15. **Other essential domains:** nutrition (**MNA**, SNAQ, weight loss), **special senses** (whispered voice test, Snellen), **oral health** (GOHAI), **continence**, and **social network and support** (**Lubben Social Network Scale**).
16. **Also assess:** pain, polypharmacy (Beers/STOPP-START), continence, sleep, alcohol (CAGE, SMAST-G), caregiver capacity, financial resources, and **home environment**.

How to Do It in Practice

17. Use **both report and performance**. Reports tell you what the person does **under real living conditions**; performance tests tell you what they can do **under standard conditions**.
18. **Grade each task** as: independent without difficulty / independent but with difficulty or an aid / needs human help / unable even with help. Distinguishing "independent with difficulty" adds prognostic value.
19. **Perform a task analysis** of the specific problem. *Example:* "difficulty dressing" may be due to choosing clothes, remembering to finish, buttoning, reaching overhead, or bending for shoes — pointing respectively to **cognitive impairment, poor finger mobility, or shoulder, back or hip disease**. Each demands a different treatment.
20. **Correlate any new functional loss** with **new drugs or dose changes (including over-the-counter)**, **changes in living conditions**, and **intercurrent acute illness (even mild)**. Remember the **cascade** — impaired gait leads to depression and withdrawal, and **immobility of any cause produces deconditioning that persists after the cause is removed**. Finally, **link assessment to a plan**: rehabilitation steps of **assessment, goal setting, therapy, aids and adaptations, education, psychological support, evaluation and follow-up**.

B. Practical Bedside Scheme

Step 1 — History Premorbid and current ADL/IADL status · exact task that fails · duration and rate of decline · pain · falls · continence · drugs (including over-the-counter) · nutrition and weight change · mood and motivation · social support and caregiver capacity · housing and environment.

Step 2 — Examination Postural blood pressure · vision and hearing screening · **observation of gait** · musculoskeletal (tone, power, joint range of motion, feet) · neurological (focal signs, sensation, proprioception) · **skin over pressure areas** · cognition (MMSE/MoCA) and mood (GDS).

Step 3 — Performance testing Gait speed over 4 metres · Timed Up-and-Go · 30-second chair stand · 4-stage balance · grip strength · observed transfer and stair climbing.

Step 4 — Baseline investigations Complete blood count, blood chemistry, **serum calcium (mg/dL), sodium (mEq/L), creatinine (mg/dL)**, thyroid function, vitamin B12, **25-hydroxy vitamin D**, haemoglobin (g/dL), fasting glucose or HbA1c, urinalysis.

Step 5 — Formulate and act Identify **reversible causes** → treat disease and pain → **deprescribe** → refer to physiotherapy, occupational therapy, dietician and social work → prescribe aids and adaptations → set goals with the patient → arrange **follow-up and case management**.

C. Latest Updates (2023–Present)

1. WHO ICOPE and intrinsic capacity. The five-step pathway screens **six domains — mobility, cognition, nutrition/vitality, vision, hearing and psychological well-being** — and can be done by a professional or by self-assessment through the **ICOPE Monitor** app. Reference centiles for intrinsic capacity and real-world screening data were published in *Nature Aging* (2023, 2024).

2. German DGG S3 Guideline (2024) defines minimum requirements — **six core dimensions in at least 15 minutes** — the first guideline to set a floor standard for functional assessment in hospital.

3. Age-Friendly Health Systems "4Ms." What Matters, Medication, Mentation and Mobility. In an eight-hospital 2024 evaluation of 3,103 admissions, patients receiving all 4Ms had **fewer 30-day emergency visits (16% vs 22%)**; **Mobility generated the highest "act-on" rate (83%)** of the four.

4. Gait speed as the single best marker. A 2025 longitudinal study found **slow gait speed the best single predictor of incident ADL and IADL disability**, outperforming both frailty and sarcopenia as constructs.

5. Italian CGA guideline (2024) — across 117 studies, CGA **maintains functional activities** and reduces hospitalisation, mortality, institutionalisation and delirium.

6. Oncology. ASCO 2023 requires geriatric assessment including **physical function** in all patients over 65 on systemic cancer therapy, using the **Practical Geriatric Assessment**.

7. Indian data. LASI wave-1 (31,464 adults aged 60 and above) found **about 3% with severe ADL disability and about 6% with severe IADL disability**, with **7.4% ADL dependency**. Rural surveys show far higher figures — **ADL disability 13.7% and IADL disability 38.7%** in rural Goa — associated with sex, education, financial dependence, comorbidity, alcoholism and **sensory impairment**. Functional tools are being adapted for community health worker delivery under **NPHCE**.

D. Current Practice Update

Aspect	Pathy's 6th ed. (2022)	Current position (2023–2026)
Core concept	Function measured as **deficit** (ADL/IADL loss)	**WHO shift to intrinsic capacity** — measure **reserve**, not only deficit
Number of domains	Broad, flexible list	**Standardised minimum sets** — ICOPE six domains; German S3 six dimensions in 15 minutes
Gait speed cut-off	Below 0.6 m/s abnormal	**0.8 m/s or less** widely used; slow gait now the best single predictor of incident disability
Who assesses	Interdisciplinary professional team	**Self-assessment and digital screening validated**; community health worker delivery in LMICs
Hospital framing	ACE units and geriatric assessment programmes	**4Ms Age-Friendly framework** — Mobility as a named, auditable care process
Sarcopenia	Not integrated into functional assessment	Functional assessment now **routinely linked to sarcopenia case-finding**

E. Exam One-Liners

- **Function is the common language of geriatrics.**
- **IADLs go first, ADLs later** — IADL loss is the earlier warning.
- **Two screens you must never omit: cognition and depression.**
- **Hierarchy rule:** ADL-dependent, skip frailty; robust, skip basic ADLs.
- **Record premorbid function, not just current function.**

- **TUG above 12 s; Tinetti below 24; gait speed below 0.6 m/s (Pathy) or 0.8 m/s (EWGSOP2).**
- **New functional decline equals a new drug, a new illness, or a new living situation** until proved otherwise.
- **Task analysis converts a vague complaint into a treatable diagnosis.**
- **WHO ICOPE six domains; 4Ms — What Matters, Medication, Mentation, Mobility.**
- **India (LASI): about 3% severe ADL and 6% severe IADL disability; 7.4% ADL dependency.**
- **Assessment without a linked care plan is not an assessment.**

QUESTION 4

Tests of Cognition and Rating Scales

A. 20 High-Yield Points

Principles

1. **Purpose of cognitive testing:** to establish **strengths and weaknesses of various functions relative to a standardised, norm-referenced scoring system**, so that the **nature and extent of cognitive deficits** can be determined, informing diagnosis and management and acting as a **comparator for past and future assessments**.
2. **A cognitive screen is not a diagnosis.** The MMSE and similar tools give an overall assessment of cognitive function, but **their use alone is not sufficient to establish a diagnosis of Alzheimer's disease or any dementia**.
3. **Cognitive screening is one of only two mandatory screens** (with depression) in any older person with functional disability.
4. **Key aspects of the mental status examination:** state of consciousness · general appearance and behaviour · orientation · **memory (short and long term)** · language · visuospatial function · **executive control functions** · other cognitive functions (calculations, proverb interpretation) · **insight and judgement** · thought content · mood and affect.
5. **Test all three components of memory: immediate recall** (repeating digits), **recent memory** (recalling three objects after a few minutes), and **remote memory** (details of early life).
6. **Executive function** involves the **planning, sequencing and execution of goal-directed activities**, and is **critical to the ability to perform IADLs**. Screen with the **clock-drawing test** (a component of the Mini-Cog) or **verbal fluency** (listing 12 animal names in one minute is normal).
7. **Interpretation depends on education and age.** The MMSE score is **dependent on sociocultural level and subject to a ceiling effect** — a crucial caveat in the Indian population, where illiteracy is common.
8. **Assess premorbid intellectual status** where possible (for example the National Adult Reading Test) to interpret other test scores correctly.
9. **Adapt tests to deficits.** In language impairment or dyspraxia, **recognition memory tests reduce the demand for expressive language** required by recall tests.
10. **Always include a standardised measure of mood** — depression is the commonest mimic of cognitive impairment.

Screening and Assessment Tools

11. **Recommended tools in primary care (Table 72.2):** - **Delirium: Confusion Assessment Method (CAM)** - **Dementia screening: AD8**, **Mini-Cog** - **Dementia assessment: SLUMS**, **MMSE**, **MoCA**
12. **Mini-Cog** screens **both memory (three-item recall) and executive function (clock drawing)** — quick, and relatively education-independent.
13. **MoCA and Addenbrooke's Cognitive Examination-Revised (ACE-R)** are described as **well-validated screening options**; ACE-R **incorporates the MMSE and clock-face drawing**.
14. **MMSE severity bands in Alzheimer's disease: mild 20–24, moderate 10–19, severe below 9.**
15. **Global staging scales combining cognition and ADLs: Global Deterioration Scale (GDS, Reisberg)** and the **Clinical Dementia Rating (CDR) scale**.
16. **Severe Impairment Battery (SIB)** was developed for subjects **too cognitively impaired for standard testing**.
17. **Memory-specific screens:** the **five-word test** and the **Memory Impairment Screen (MIS)** assess **anterograde verbal memory** at an initial consultation. For detailed evaluation, **free and cued recall** paradigms — the **Grober and Buschke test (Free and Cued Selective Reminding Test)** and the **California Verbal Learning Test** — distinguish mild Alzheimer's disease from healthy subjects with **more than 90% precision**, even when MMSE scores are similar.
18. **Domain-specific battery (Table 62.4 — Cardiff memory clinic):**

Domain	Tests
Premorbid ability	**National Adult Reading Test (NART)**
Cognitive screen / battery	**MMSE, MoCA, RBANS, Frontal Assessment Battery (FAB)**
Attention and concentration	Digits forward, digits backward, digit span
Language	**Boston Naming Test**, Irving Names Learning Test
Memory	Story recall (immediate and delayed), Kendrick tests, **California Verbal Learning Test**
Executive function	**Trail-Making Test A and B**, clock drawing
Visuospatial function	**Rey–Osterrieth Complex Figure**, clock drawing
Mood	**GDS-15**

Delirium Instruments

19. **Confusion Assessment Method (CAM)** — the standard; **sensitivity 94%, specificity 89%** with high inter-rater reliability. **Inattention** is tested at the bedside by **months of the year backwards, days of the week backwards, or digit span 3 or 4 backwards**. - **3D-CAM:** a structured **three-minute** screen — **sensitivity 96%, specificity 86% in patients with dementia**; **93% and 96% in those without**. - **Best single item: "months of the year backwards"** (sensitivity 83%, specificity 69%). Best two items: months backwards plus "what is the day of the week?" (sensitivity 93%, specificity 64%). - **CAM-ICU** for intubated patients — inattention tested by squeezing on the letter A in "SAVEAHAART"; level of consciousness by **RASS score other than 0**.

Practical Conduct of Testing

20. Tests must be **acceptable to the person and contain nothing belittling**. **Consent must be obtained**, the purpose explained, and **sensory and physical limitations accommodated** — adequate lighting, earphone amplifiers, suitable seating, minimal distractions. Provide **realistic reassurance with positively phrased feedback that highlights strengths as well as weaknesses**, and at the end **allow patients to comment on their own performance**. Ideally an experienced psychologist or neuropsychologist performs formal testing, though **much useful information can be obtained by any suitably trained professional**. Introduce questions **non-threateningly**, because many patients with early deficits respond defensively.

B. Comparison of the Main Cognitive Tools

Tool	Items / time	Domains	Main strength	Main limitation
AMT (Hodkinson)	10 questions, 2 min	Orientation, memory	Very quick; RCP/BGS endorsed	Crude; misses executive and visuospatial deficits
Mini-Cog	3 min	3-word recall + clock drawing	**Relatively education-independent**; screens executive function	Screen only
AD8	8 items, informant	Change over time	Informant-based, detects change	Needs a reliable informant
MMSE	30 points, 10 min	Orientation, registration, attention, recall, language, construction	Most widely known; severity bands defined	**Education- and culture-dependent; ceiling effect; poor for executive function**; copyrighted
MoCA	30 points, 10–15 min	Adds executive, abstraction, visuospatial, attention	**Superior for mild cognitive impairment and executive dysfunction**	Lower specificity; education adjustment (+1 if 12 years or less)
SLUMS	30 points, 10 min	Adds executive and clock tasks	Free to use; education-stratified cut-offs	Less international validation
ACE-R / ACE-III	100 points, 20 min	Five domains; incorporates MMSE	Differentiates **Alzheimer's from frontotemporal dementia**	Long
RBANS	30 min	Repeatable battery	Serial testing without practice effect	Requires training
Frontal Assessment Battery	6 items	Frontal/executive	Detects subcortical and frontal deficits	Narrow
CAM / 3D-CAM / CAM-ICU	3–5 min	Delirium	**Gold standard for delirium**	Needs training; misses hypoactive cases if hurried
GDS (Reisberg) / CDR	Interview	Global staging	Combines cognition and function	Time-consuming
SIB	40 min	Severe dementia	Usable when MMSE floor is reached	Specialist tool

C. Latest Updates (2023–Present)

1. 2024 NIA-AA Revised Criteria — Alzheimer's disease is now defined biologically. Diagnosis rests on **Core 1 biomarkers** — a positive **plasma p-tau217**, a positive **amyloid PET**, or abnormal **CSF ratios (p-tau181/Aβ42, t-tau/Aβ42, Aβ42/40)**. Cognitive testing now defines the **clinical stage**, not the disease itself. This is the single biggest conceptual change in the field.

2. Blood-based biomarkers have entered clinical use. Diagnostic models combining **plasma p-tau217 with age and ApoE genotype** achieve **AUC around 0.94** for Alzheimer's disease. In very elderly nursing-home and hospital populations, **plasma p-tau181, p-tau217 and GFAP differ significantly between cognitively normal and probable Alzheimer's groups**, while **Aβ42, Aβ40 and the Aβ42/40 ratio do not** — an important practical point, since amyloid ratios perform poorly in the oldest old.

3. Cognitive screens are being repositioned as triage. Rather than diagnosing, MMSE and MoCA increasingly serve to **decide who proceeds to biomarker testing**. Clinical trials now use combinations such as **CDR global score 0.5–1.0**

plus MMSE 21 or above plus a positive Core 1 biomarker for entry.

4. Mild behavioural impairment (MBI) — late-onset persistent neuropsychiatric symptoms — has emerged as an **early indicator of dementia risk that may precede measurable cognitive decline**, and is being studied alongside plasma p-tau217.

5. Digital and mobile cognitive assessment. Mobile cognitive assessment systems have demonstrated **diagnostic equivalence to MMSE and MoCA** in Alzheimer's screening; future direction is **composite risk scores combining digital tests, wearables and plasma p-tau217**. Regulatory and ethical frameworks for data security, algorithmic transparency and equitable access are still being established.

6. Standardised inclusion in assessment frameworks. Cognition/delirium is one of the **six mandatory core dimensions** of the German S3 CGA guideline (2024); **cognition** is one of the **six WHO ICOPE domains**, with **Mini-Cog, MoCA, MMSE or GPCOG** named as step-2 assessment tools; **Mentation** is one of the **4Ms** of Age-Friendly Health Systems.

7. Indian context. In LASI wave-1, **impaired cognition was present in 9.3%** of adults aged 60 and above. Culture- and education-fair instruments are essential: **Hindi Mental State Examination (HMSE)**, **Vernacular MoCA versions**, **Rowland Universal Dementia Assessment Scale (RUDAS)**, **Indian Council of Medical Research Neuro-Cognitive Tool Box**, and **AIIMS Comprehensive Neuropsychological Battery in Hindi** are used in place of a directly translated MMSE, because **the standard MMSE penalises illiteracy heavily**.

D. Current Practice Update

Aspect	Pathy's 6th ed. (2022)	Current position (2023–2026)
Basis of Alzheimer's diagnosis	Clinical, supported by cognitive tests and imaging	**Biological — 2024 NIA-AA criteria** based on Core 1 biomarkers
Role of MMSE/MoCA	Screening and severity grading	**Staging and triage** to biomarker testing; no longer diagnostic on their own
Biomarkers	CSF amyloid and tau "in some centres"	**Plasma p-tau217 in routine diagnostic pathways**; AUC around 0.94 with age and ApoE
Amyloid ratios	Presented as useful	**Aβ42/40 performs poorly in the oldest old**; p-tau217 and GFAP superior
Delivery	Face-to-face testing by trained staff	**Mobile and computerised assessment shown equivalent to MMSE/MoCA**
Preclinical detection	Subjective cognitive decline and MCI	Adds **mild behavioural impairment (MBI)** as an early neurobehavioural marker
Indian practice	MMSE with education caveat	**Education-fair tools mandatory** — HMSE, RUDAS, vernacular MoCA, ICMR NCTB

E. Exam One-Liners

- **A cognitive screen is not a diagnosis** — MMSE alone cannot diagnose Alzheimer's disease.
- **MMSE bands:** mild 20–24, moderate 10–19, severe below 9.
- **MMSE is education- and culture-dependent with a ceiling effect** — the key limitation in India.
- **MoCA beats MMSE for mild cognitive impairment and executive dysfunction.**
- **Mini-Cog = 3-word recall plus clock drawing** — screens memory and executive function in three minutes.
- **Clock drawing and verbal fluency screen executive function**, which drives **IADL** performance.
- **CAM sensitivity 94%, specificity 89%; 3D-CAM takes three minutes.**
- **Best single delirium item: months of the year backwards.**
- **ACE-R incorporates the MMSE and clock drawing** and helps separate Alzheimer's from frontotemporal dementia.
- **Free and cued recall (Grober–Buschke) separates mild Alzheimer's from normal with over 90% precision** even at similar MMSE scores.
- **Global staging: Reisberg GDS and CDR** combine cognition with function.
- **SIB is for patients too impaired for standard testing.**
- **2024 NIA-AA: Alzheimer's disease is now a biological diagnosis** — plasma **p-tau217**, amyloid PET, or CSF ratios.
- **Always pair cognitive testing with a mood scale**, and obtain consent and premorbid ability first.

QUESTION 5

Cognitive Assessment Tools

A. 20 High-Yield Points

Classification — the Framework to Open With

1. Cognitive assessment tools fall into **four functional groups**, and stating this classification first earns marks: - **Delirium instruments** — CAM, 3D-CAM, CAM-ICU, 4AT - **Brief screening tools** — Mini-Cog, AD8, AMT, GPCOG, MIS, six-item screener - **Cognitive screening batteries** — MMSE, MoCA, SLUMS, ACE-III, RUDAS - **Detailed neuropsychological batteries and staging scales** — RBANS, CVLT, Trail-Making, FAB, Boston Naming, Rey–Osterrieth; CDR and Reisberg GDS for staging
2. **A second axis is the informant dimension — performance-based** (patient does the task: MMSE, MoCA) versus **informant-based** (a relative reports change: **AD8, IQCODE**). Informant tools detect **change from a personal baseline** and are invaluable when education confounds performance tests.
3. **The tool must match the question:** *Is this delirium?* → CAM. *Is there any cognitive problem?* → Mini-Cog or AD8. *How severe and in which domain?* → MoCA, MMSE, SLUMS. *Which dementia is it?* → ACE-III with a full battery. *How advanced is it?* → CDR or Reisberg GDS.

Delirium Tools

4. **CAM** — sensitivity **94%**, specificity **89%**, high inter-rater reliability. Four features: **acute onset and fluctuating course, inattention, disorganised thinking, altered level of consciousness** (features 1 and 2 plus either 3 or 4).
5. **Inattention testing at the bedside: months of the year backwards, days of the week backwards, digit span 3 or 4 backwards**; or the examiner may simply observe **inappropriate distraction or loss of the thread of conversation**.
6. **3D-CAM** — three-minute structured version: **sensitivity 96%, specificity 86% in patients with dementia**; **93% and 96% without dementia**. **CAM-ICU** for intubated patients uses the "SAVEAHAART" letter-A task and the **RASS**.

Brief Screens

7. **Mini-Cog — three-item recall plus clock drawing**; screens **memory and executive function** in about three minutes and is **relatively education-independent**, making it the most practical screen in Indian outpatient settings.
8. **AD8** — an **eight-item informant questionnaire** detecting change over time; useful when the patient's education level makes performance testing unreliable.
9. **Abbreviated Mental Test (Hodkinson, 10 items)** — the **RCP/BGS-endorsed** memory and cognition screen; extremely fast but crude.
10. **Memory Impairment Screen and the five-word test** assess **anterograde verbal memory** specifically at a first consultation.

Screening Batteries

11. **MMSE (Folstein, 1975)** — 30 points across orientation, registration, attention/calculation, recall, language and construction. **Interpretation is complex because thresholds depend on education and age**; it has a **ceiling effect** and **tests executive function poorly**. Severity bands: **mild 20–24, moderate 10–19, severe below 9**.
12. **MoCA (Nasreddine, 2005)** — 30 points; adds **trail-making, cube copy, clock drawing, verbal fluency, abstraction and delayed recall**, so it detects **mild cognitive impairment and executive/subcortical dysfunction far better than the MMSE**. **Add 1 point if formal education is 12 years or less.**
13. **SLUMS (Saint Louis University Mental Status)** — 30 points; free of charge, with **separate cut-offs for those with and without a high-school education**.
14. **ACE-R / ACE-III** — 100 points across **attention/orientation, memory, verbal fluency, language and visuospatial function**; ACE-R **incorporates the MMSE and clock-face drawing**, and the **VLOM ratio helps separate Alzheimer's disease from frontotemporal dementia**.
15. **RUDAS (Rowland Universal Dementia Assessment Scale)** — designed to be **culturally and linguistically fair**, needing no literacy; highly relevant to multilingual, variably literate Indian populations.

Detailed and Domain-Specific Tools

16. **Domain mapping (Table 62.4):** premorbid ability — **NART**; screening battery — **MMSE, MoCA, RBANS, FAB**; attention — **digit span forward and backward**; language — **Boston Naming Test**; memory — **story recall, California Verbal Learning Test**; executive — **Trail-Making A and B, clock drawing**; visuospatial — **Rey-Osterrieth Complex Figure**; mood — **GDS-15**.
17. **Free and cued recall tests** — the **Grober and Buschke test** and the **CVLT** — separate mild Alzheimer's disease from normal ageing with **more than 90% precision**, even when MMSE scores are identical, because they demonstrate a **hippocampal amnestic pattern (cueing does not improve recall)**.

18. **Staging scales: Clinical Dementia Rating (CDR)** and the **Reisberg Global Deterioration Scale** combine cognition with ADL impact. The **Severe Impairment Battery** is used when impairment is too severe for standard testing.

Rules of Use

19. **Practical conduct:** obtain **consent**, explain the purpose, ensure **adequate lighting, hearing amplification, suitable seating and no distractions**, introduce questions **non-threateningly**, phrase feedback **positively**, and give the patient a chance to comment on their own performance at the end. Assess **premorbid intellectual status** first, and **always add a mood scale**.
20. **Two cardinal caveats:** - **Depression and delirium must be excluded before any score is attributed to dementia** — screening tools become unreliable in delirium, and depression is the commonest reversible mimic. - **No cognitive screening test is appropriate for determining decision-making capacity.** Screens of **executive function** such as the **clock-drawing test** or the **Executive Interview** correlate better with decisional capacity than the MMSE, but capacity requires its own task-specific assessment.

B. Comparison Table — Choosing the Right Tool

Clinical question	Tool of choice	Time	Notes
Is this delirium?	**CAM / 3D-CAM / CAM-ICU**	3–5 min	Sensitivity 94%, specificity 89%
Rapid screen in a busy clinic	**Mini-Cog**	3 min	Education-independent; memory + executive
Screen when the patient cannot be tested	**AD8 or IQCODE** (informant)	3 min	Detects change from baseline
General cognitive assessment	**MoCA**	10–15 min	Best for MCI; add 1 point if education 12 years or less
Severity grading in known dementia	**MMSE**	10 min	Mild 20–24, moderate 10–19, severe below 9
Free alternative with education cut-offs	**SLUMS**	10 min	Education-stratified
Differentiating dementia subtypes	**ACE-III**	20 min	VLOM ratio; Alzheimer's vs frontotemporal
Illiterate or multilingual patient	**RUDAS, HMSE, vernacular MoCA**	10 min	Culture- and literacy-fair
Detecting early Alzheimer's memory pattern	**Grober–Buschke, CVLT**	20–30 min	Cueing fails to improve recall
Frontal or subcortical deficit	**FAB, Trail-Making B, verbal fluency**	5–10 min	Subcortical screen
Global staging	**CDR, Reisberg GDS**	Interview	Combine cognition and function
Severe dementia	**SIB**	40 min	Below the MMSE floor

C. Latest Updates (2023–Present)

1. 2024 NIA-AA Revised Criteria define Alzheimer's disease **biologically** through **Core 1 biomarkers — plasma p-tau217, amyloid PET, or CSF p-tau181/Aβ42, t-tau/Aβ42 or Aβ42/40**. Cognitive tools now **stage the disease rather than diagnose it**.

2. Plasma p-tau217 is the practice-changing test. Models combining **plasma p-tau217, age and ApoE genotype** achieve **AUC about 0.94**. In the very elderly, **p-tau181, p-tau217 and GFAP discriminate probable Alzheimer's from normal cognition, while Aβ42, Aβ40 and the Aβ42/40 ratio do not** — so amyloid ratios should not be relied on in the oldest old.

3. Digital cognitive assessment. Mobile cognitive assessment systems show **diagnostic equivalence to MMSE and MoCA** in Alzheimer's screening. Future work is on **composite scores integrating digital tests, wearables (for example sleep fragmentation) and plasma p-tau217**, with cross-ethnic validation and regulatory frameworks still pending.

4. Mild behavioural impairment (MBI) — late-onset persistent neuropsychiatric symptoms — is now recognised as an early dementia-risk indicator that may **precede measurable cognitive decline**, and is being validated against plasma p-tau217.

5. Mandated inclusion in assessment frameworks. German S3 CGA guideline (2024): cognition and delirium is one of six core dimensions. **WHO ICOPE:** cognition is one of six intrinsic-capacity domains, with **Mini-Cog, MoCA, MMSE and GPCOG** as named step-2 tools. **Age-Friendly 4Ms:** cognition sits under **Mentation**. **ASCO 2023 Practical Geriatric Assessment** includes a cognitive domain for every cancer patient aged 65 and above.

6. Indian tools and data. LASI wave-1 found **impaired cognition in 9.3%** of those aged 60 and above. Because the MMSE penalises illiteracy heavily, Indian practice uses the **Hindi Mental State Examination (HMSE)**, **RUDAS**, **vernacular MoCA versions**, the **ICMR Neuro-Cognitive Tool Box**, and the **AIIMS Comprehensive Neuropsychological Battery in Hindi**.

D. Current Practice Update

Aspect	Pathy's 6th ed. (2022)	Current position (2023–2026)
What the tools do	Screen for and grade cognitive impairment	**Stage the disease** and **triage to biomarker testing**
Diagnosis of Alzheimer's	Clinical, supported by imaging and sometimes CSF	**Biological (2024 NIA-AA)** — Core 1 biomarkers
Blood tests	Not diagnostically useful	**Plasma p-tau217 in routine pathways**; GFAP adds value
Amyloid measures	CSF amyloid used in some centres	**Aβ42/40 unreliable in the oldest old**
Mode of testing	Paper-and-pencil, face to face	**Mobile and computerised testing equivalent to MMSE/MoCA**
Preclinical markers	Subjective cognitive decline, MCI	Adds **mild behavioural impairment**
Cross-cultural use	MMSE with an education caveat	**Literacy-fair instruments mandatory** in India — RUDAS, HMSE, vernacular MoCA

E. Exam One-Liners

- **Four groups of tools:** delirium instruments, brief screens, screening batteries, detailed batteries and staging scales.
- **CAM for delirium; Mini-Cog or AD8 to screen; MoCA or MMSE to assess; ACE-III to subtype; CDR or Reisberg GDS to stage.**
- **Mini-Cog = 3-word recall plus clock drawing**, and is relatively education-independent.
- **AD8 and IQCODE are informant-based** — they measure change from the person's own baseline.
- **MoCA detects MCI and executive dysfunction better than MMSE; add 1 point if education is 12 years or less.**
- **MMSE has a ceiling effect, is education-dependent, and tests executive function poorly.**
- **RUDAS, HMSE and vernacular MoCA are the literacy-fair choices for Indian patients.**
- **Grober–Buschke free and cued recall demonstrates the hippocampal amnestic pattern of Alzheimer's disease.**
- **ACE-R incorporates the MMSE and clock drawing.**
- **Exclude delirium and depression before interpreting any cognitive score.**
- **No cognitive screening test determines decision-making capacity** — executive tests correlate better than the MMSE, but capacity needs its own assessment.
- **2024 NIA-AA: plasma p-tau217, amyloid PET or CSF ratios define the disease; cognition defines the stage.**

QUESTION 6

Assessment of Decisional Capacity in an Older Person

A. 20 High-Yield Points

Concepts and Definitions

1. **Capacity and competence are not the same.** A **clinician evaluates a patient's capacity to make a decision**; **competence is a legal term** implying that a person **has been deemed competent or incompetent by a court of law**. Someone not competent to act on their own behalf **requires an agent to act for them**.
2. **Competence** refers to a person's ability to **act reasonably after understanding the nature of the situation being faced**.
3. **Capacity is decision-specific and time-specific.** A patient may have capacity for one decision and not another, and may regain capacity later.
4. **Capacity is presumed until proved otherwise.** A **diagnosis of dementia — even by type — is not a sufficient indicator of a person's ability to comprehend and express a meaningful preference**. Intellectual deficits are **spotty**: a person may get lost or forget things yet still make decisions **with appropriate prompting**.
5. **The ethical foundation is autonomy** — the capacity of each adult and cognitively competent individual to **process information and freely decide on a course of action that best suits their own values, preferences, goals and desires**. Respect for autonomy underpins **informed consent**.
6. **Preconditions of valid informed consent: competence to understand and decide**, and **voluntariness in deciding**. The **information element** is **disclosure of relevant information** about the condition and the proposed course of action.
7. **An unwise decision is not incapacity.** In a patient with capacity, autonomy means "*you may not do things to me without my permission*" — **even if a patient's decision to refuse treatment seems unwise, if they have capacity their refusal stands**, and the clinician's **duty of care does not diminish**; all other care continues without prejudice.
8. **Just as it is wrong to infantilise patients** by directing questions to others who answer faster, **it is equally wrong to prejudge their ability to participate in decisions about their own care**.

The Four Elements of Decisional Capacity

9. **Decisional capacity is predicated on four elements** — the single most examinable list: - **Understanding** — the ability to comprehend the disclosed information about the nature of the situation or medical problem, the procedures involved, and the risks and benefits of undergoing or not undergoing testing or treatment. - **Appreciation** — of the significance of the disclosed information and the potential risks and benefits **for one's own situation and condition**. - **Reasoning** — the ability to engage in a reasoning process about the risks and benefits. - **Choice** — being able to **express a choice** whether or not to undergo further testing or treatment.
10. **The four US legal standards for determining decisional capacity** (a parallel, equally examinable list): - The patient is **aware of the treatment options and expresses a treatment preference clearly and consistently**. - The patient can give a **conventionally understandable reason** for the decision that is **consistent with their past behaviour**. - The patient **remembers and can tell back the information** about diagnosis, prognosis and treatment options given by the practitioner. - The patient can **state what their decision means for their own state of health**.
11. **The sliding-scale principle (Figure 95.1):** the **standard of decisional capacity required rises with the risk of the intervention**. A **fully cognisant patient with good health literacy** can consent to high-risk options such as a **phase 1 clinical trial**; a **cognitively impaired patient lacking decisional capacity could not consent to experimental treatment** and, given reduced life expectancy in advanced dementia, would be unlikely to benefit from highly toxic therapy even if it is standard of care. **The area between is the domain of best clinical judgement.** Hence **"staging the ageing is as important as staging the cancer."**

Assessment in Practice

12. **Distinguish memory from understanding** — a person may fail a memory test yet understand the decision at hand.
13. **No cognitive screening test is appropriate to determine decision-making capacity.** Screening tools that focus on **executive function** — the **Clock Drawing Test** or the **Executive Interview** — are **more likely to be associated with decisional capacity** than the MMSE.
14. **Formal capacity instruments exist but are difficult for older adults**, because they require consideration of **hypothetical situations and complex abstract thinking**. Examples: **MacCAT-T (MacArthur Competence Assessment Tool for Treatment — the reference standard)**, **Aid to Capacity Evaluation (ACE)**, **Capacity to Consent to Treatment Instrument (CCTI)**, **Hopemont Capacity Assessment Interview**, **Understanding Treatment Disclosures**.
15. **A useful practical criterion** in routine informed consent is to **recheck with the patient after an interval** to be certain that they **understand the situation and consistently state the same preference**.

16. **Maximise capacity before declaring its absence.** Before deciding a person cannot decide: correct **sensory deficits** (a deaf patient may need a hearing aid, many older people need glasses), **treat delirium, depression, pain and infection**, review sedating drugs, use **simple language, repetition and rephrasing**, choose a **quiet, distraction-free environment**, and involve a familiar person. Written material must be legible — a leaflet is useless if **the typeface is too small, the colour contrast is poor, or there is no white space**.
17. **Formal assessment of mental state may be necessary**, but **no specific score is associated with an inability to make decisions about one's personal healthcare** — this is an important negative statement to write in an examination.
18. **Where capacity is absent:** consult any **advance care directive** and the **surrogate or proxy decision-maker**. A proxy may be formally appointed through an **enduring power of attorney**, or the patient's wishes may be recorded in an **advance directive**; if none exists, the lead clinician must decide in the patient's **best interests**, or apply for **guardianship**.
19. **Hierarchy of treatment decision-makers** (Australian model, quoted in the textbook): **enduring guardian with authority → guardian with authority → adult son or daughter → parent → sibling → primary unpaid carer → other person with a close personal relationship and frequent contact**. The clinician goes to the first person who is **18 or older, has full legal capacity, and is reasonably available and willing**.
20. **Communication and documentation:** - **Relatives must not be given the impression that the decision is theirs to make** — they may suffer significant distress from the emotional burden. Their role is to provide **valuable information about what would be in the patient's best interests**. - Where families cannot bear to decide, the humane option is **"informed assent"** — explicitly offering them the choice to **defer to the provider's judgement**, documented clearly in the record. - **Anticipate decisions early in admission** so the patient can be involved as much as possible and **proxy decision-making is avoided**. - Clinicians must disclose: **the condition being treated; the nature of the proposed treatment or procedure; anticipated results; recognised alternatives; and recognised serious risks, complications and benefits, including of non-treatment.** - **Document the assessment, the reasoning and the outcome.**

B. Structured Approach to a Capacity Assessment

Step	Action
1. Define the decision	Capacity is **decision-specific** — state exactly which decision is in question
2. Presume capacity	Start from presumption; a diagnosis alone never establishes incapacity
3. Optimise the person	Treat delirium, pain, depression, infection; correct vision and hearing; review sedatives; choose the best time of day and a quiet setting
4. Disclose	Condition, proposed treatment, expected outcome, alternatives, serious risks, and the consequences of non-treatment — in **plain language**
5. Test the four elements	**Understanding** ("tell me in your own words") · **Appreciation** ("what does this mean for you?") · **Reasoning** ("how did you weigh those up?") · **Choice** ("so what would you like to do?")
6. Apply the sliding scale	Higher risk demands a higher standard of capacity
7. Recheck	Re-ask after an interval to confirm **consistency** of preference
8. Conclude and act	If capacity present, the decision stands even if unwise. If absent, consult advance directive, then surrogate, then best interests or guardianship
9. Document	Record the specific decision, the disclosure made, the four elements, the conclusion and the reasoning

C. The Indian Legal Framework

1. **Mental Healthcare Act (MHCA), 2017** is the governing statute across India and was rewritten after India ratified the **UNCRPD in 2007**. Capacity has a **pivotal role**.
2. **Section 4, MHCA 2017:** every person is **presumed to have the capacity to make mental healthcare and treatment decisions** if they can **understand the information, appreciate the foreseeable consequences of the decision, or communicate the decision by speech, gesture or any other means**.
3. **Memory loss, cognitive deficits, or even a formal diagnosis of dementia do not imply incapacity automatically** — the Act emphasises **establishing capacity**, and gives **preference to the choices and wishes of a person who has capacity**.
4. **The opinion of the assessor or a third party that a decision is "unwise" must not influence the capacity assessment.** Capacity is **specific to the task and to the time**.
5. **Advance directives — Section 5(3):** an advance directive is **invoked only when the person becomes incapacitated** and **remains effective until they regain capacity**. The Act does not clearly distinguish advance statements from advance directives.

6. **Nominated representative:** decision-making by a nominated representative is **restricted to those lacking capacity for mental healthcare decisions**. **Supported decision-making** ranges from minimal support to complete support.
7. **Other relevant Indian law:** the **Maintenance and Welfare of Parents and Senior Citizens Act, 2007** (maintenance, protection, and transfer-of-property provisions), the **Rights of Persons with Disabilities Act, 2016** (limited guardianship), the **National Trust Act, 1999**, and **Elder Line 14567** for reporting abuse and neglect.
8. **Practical Indian caveat:** the **MacCAT-T, though the international reference standard, is not standardised for the Indian population**, and **family plays a central role in healthcare decision-making** in Indian and other Asian societies — creating real difficulties in operationalising individual autonomy and advance directives.

D. Latest Updates (2023–Present)

1. Indian evidence base is only now emerging. A 2025 descriptive cross-sectional study of mental capacity and advance directives in Indian persons with mental illness confirmed that **published Indian data remain very scarce**, that **the MacCAT-T is regarded as the gold standard but is not standardised for India**, and that the **central role of the family creates practical difficulty in determining advance directives** and enabling individual decision-making about future treatment.

2. Dementia and Indian law. A published analysis of the assessment of dementia under prevailing Indian laws confirms that under **Section 4 of MHCA 2017 a diagnosis of dementia does not itself establish incapacity**, and sets out the four components of the decision-making process as **understanding, appreciating, reasoning and communicating**. It also distinguishes **capacity** (clinical) from **competence** (legal — relating to valid contracts and financial and personal decision-making).

3. UK practice guidance (2024). NHS England guidance restates the functional test as the ability to **understand the relevant information, retain it long enough to make the decision, weigh up the arguments on each side, and communicate the decision** (by talking, signing or any other means). It provides a **practical checklist**: choose a quiet space free from distractions and noise, avoid jargon, **repeat and rephrase questions**, and **identify in advance what the "relevant information" for that specific decision is**. Essex Chambers publishes decision-specific guidance on what constitutes relevant information for different categories of decision.

4. High prevalence of impairment in institutional settings. Older adults evaluated in long-term care have **rates of capacity impairment between 44% and 69%** — an argument for routine, structured assessment rather than ad hoc judgement.

5. Convergence with capacity-based frameworks. The **German S3 CGA guideline (2024)** mandates cognition and delirium assessment as a core dimension; **WHO ICOPE** includes cognition and psychological well-being. Both feed directly into capacity evaluation, and the **Age-Friendly 4Ms** place **"What Matters"** — eliciting the person's own goals — as the first M, operationalising autonomy in routine care.

E. Current Practice Update

Aspect	Textbook position	Current position (2023–2026)
Legal framework	Generic Western frameworks; Australian guardianship hierarchy quoted	**India: MHCA 2017 governs**, with statutory presumption of capacity and nominated representatives
Effect of a dementia diagnosis	A diagnosis is not a sufficient indicator of incapacity	**Statutorily reinforced** — Section 4 MHCA: dementia does not imply incapacity
Instruments	Formal tools exist but are hard for older adults	**MacCAT-T remains the gold standard but is not standardised for Indian populations**
Cognitive tests	No screening test determines capacity; executive tests correlate better	Unchanged and reaffirmed
Role of family	Consult relatives for best interests, but do not transfer the decision	**In India, family-centred decision-making is normative**, creating tension with individual-autonomy models
Documentation	Recommended	Now expected to be **decision-specific**, recording the relevant information disclosed
Autonomy in routine care	Ethical principle	Operationalised as **"What Matters"** in the 4Ms framework

F. Exam One-Liners

- **Capacity is clinical and decision-specific; competence is legal and determined by a court.**
- **The four elements: Understanding, Appreciation, Reasoning, Choice.**
- **Capacity is presumed** — a diagnosis of dementia never establishes incapacity by itself.
- **An unwise decision is not incapacity.** If the patient has capacity, their refusal stands, and the duty of care does not diminish.
- **Sliding scale:** the higher the risk of the intervention, the higher the standard of capacity required.

- **"Staging the ageing is as important as staging the cancer."**
- **No cognitive screening test determines capacity**; **clock drawing and the Executive Interview** correlate better than the MMSE.
- **No specific MMSE score defines incapacity.**
- **MacCAT-T is the reference instrument** — but is not standardised for India.
- **Maximise capacity first:** treat delirium, depression and pain; correct hearing and vision; simplify language; choose a quiet room and the best time of day.
- **Recheck after an interval to confirm the preference is consistent.**
- **Section 4, MHCA 2017 (India):** presumption of capacity; understand, appreciate consequences, communicate by any means.
- **Section 5(3), MHCA 2017:** advance directive is invoked only on incapacity and lapses when capacity returns.
- **Never make the family feel the decision is theirs** — offer **"informed assent"** where they cannot bear to decide.
- **Anticipate decisions early in the admission** so that proxy decision-making is avoided.

QUESTION 7

Geriatric Syndromes — An Overview

A. 20 High-Yield Points

Concept and Definition

1. **A geriatric syndrome is a clinical condition in older people that does not fit into a discrete disease category.** Geriatric syndromes are **highly prevalent, multifactorial, and associated with substantial morbidity and poor outcomes**.
2. **The defining feature is a many-to-one and one-to-many relationship: multiple contributing causes converge on a single presenting problem**, and **any one problem may arise from many different causes**. This is the opposite of the classical single-disease model.
3. **Because diagnoses often do not tell the whole story in geriatrics, it is more helpful to think in terms of presenting problems.**
4. **The underlying principle is homeostenosis** — the progressive decline in the **reserve capacity of all organ systems** with increasing age, so that **several small deficits combine to cross a threshold** and produce a syndrome that no single deficit would have caused.
5. **The classical list — "the series of I's": Immobility · Instability (falls) · Incontinence · Intellectual impairment · Infection · Impairment of vision and hearing · Irritable colon · Isolation (depression) · Inanition (malnutrition) · Impecunity · Iatrogenesis · Insomnia · Immune deficiency · Impotence.**
6. **Morley's "new geriatric giants"** add **frailty, sarcopenia, anorexia of ageing and cognitive impairment** — reflecting the modern emphasis on pre-disability states.
7. **Illustrative example (immobility):** it may be caused by a **fractured hip, congestive heart failure, a recent fall with fear of falling, or pain from degenerative joint disease** — any or all together. It is essential to **explore the underlying causes with the individual** so that appropriate interventions can be implemented; **pain and fear of falling must be addressed before engaging the person in an exercise programme**.

Shared Risk Factors — the Unifying Concept

8. **Four shared risk factors run across the five best-validated geriatric syndromes** (Tinetti, Inouye, Gill and Doucette, *JAMA* 1995; Inouye et al., *JAGS* 2007): - **Older age** - **Baseline cognitive impairment** - **Baseline functional impairment** - **Impaired mobility**
9. **The five syndromes in which these were demonstrated: pressure ulcers, incontinence, falls, functional decline and delirium.**
10. **Clinical corollary:** because the risk factors are shared, **multiple risk factor reduction is more effective than the regular single-disease medical model**, and **one intervention can prevent several syndromes at once**.
11. **Landmark proof-of-concept trials: Inouye's multicomponent HELP intervention (NEJM 1999)** for delirium prevention, and **Tinetti's multifactorial intervention (NEJM 1994)** to reduce falls in the community — both showing that **targeting shared risk factors works**.
12. **The number of risk factors present determines the total risk**, which rises **proportionally** as they accumulate — demonstrated by Tinetti for dizziness (five to seven significant risk factors).

Iatrogenesis — a Special Emphasis

13. **Iatrogenesis is an all too common problem, and the risk increases with exposure to the care system** — tests, surgical interventions, medications and treatments. In every interaction the **risk–benefit balance must be considered and explained** to the individual and their proxy.
14. **Medication management** needs particular deliberation, because older adults have **marked changes in absorption, metabolism and excretion**. Use **Beers criteria and STOPP-START**, review **anticholinergic burden**, and check **QTc risk with psychotropics**.
15. **Even more dangerous is the careless, hasty application of clinical labels.** A patient who becomes confused in hospital may **not** have dementia; a person with an occasional urinary accident is **not necessarily incontinent**. **Labelling a patient as demented or incontinent is too often the first step towards nursing-home placement — a setting that can make such labels self-fulfilling prophecies.**

Assessment and Management

16. **The clinical method for any geriatric syndrome:** - Treat the **presenting problem as a syndrome, not a diagnosis** - Enumerate **all contributing factors** rather than searching for one cause - Look specifically for the **shared risk factors** — cognition, function, mobility - Search for **reversible contributors**: a **new drug or dose change (including over-the-counter), an intercurrent acute illness, or a change in living conditions** - Intervene on **as many factors as possible simultaneously** - **Link assessment to a care plan and long-term follow-up**

17. **Comprehensive geriatric assessment is the definitive vehicle** for identifying and managing geriatric syndromes, because it is the only method that systematically covers all the contributing domains at once.
18. **Criteria for targeting CGA in diabetes (Table 86.6)** show how syndromes drive referral: **presence of a geriatric syndrome (confusional state, depression, falls, incontinence, immobility, pressure sores)**; pre-frailty or frailty; several coexisting morbidities with complex drug regimens; disability from lower-limb vascular disease or neuropathy requiring rehabilitation; and **absence of a terminal illness or dementing syndrome**.
19. **Cross-syndrome prevention principles: mobilise early ("bed is bad")**, **deprescribe**, **maintain hydration and nutrition**, **correct vision and hearing**, **avoid restraints and catheters**, **screen for cognition and mood**, and **treat pain**. Because pressure injuries, falls, delirium and functional decline share risk factors, **multifactorial interventions delivered by a multidisciplinary team** are effective across all of them.
20. **The major barrier is implementation, not evidence.** Preventive strategies of proven effectiveness for **delirium and falls** exist, but **major barriers have been identified in translating research advances into clinical practice and policy**.

B. The Major Geriatric Syndromes at a Glance

Syndrome	Key shared drivers	First-line action
Falls and instability	Gait/balance impairment, drugs, vision, cognition, environment	Multifactorial assessment; TUG, Tinetti; deprescribe; strength and balance exercise; home hazards
Immobility	Pain, disease, fear, iatrogenesis, environment	Treat pain and cause; early mobilisation; physiotherapy and occupational therapy
Delirium	Cognitive impairment, sensory deficits, drugs, infection, dehydration, immobility	**CAM**; HELP-type multicomponent prevention; treat precipitants
Dementia and cognitive impairment	Age, vascular risk, sensory deficits	Mini-Cog/MoCA; exclude reversible causes; caregiver support
Incontinence	Mobility, cognition, drugs, local pathology	Bladder diary; exclude retention, infection, impaction; timed toileting
Pressure injury	Immobility, nutrition, incontinence, low weight	Braden/Norton; pressure relief; nutrition; skin care
Frailty and sarcopenia	Disease, poor intake, sarcopenia, executive dysfunction	FRAIL screen; resistance exercise plus protein 1 g/kg/day; deprescribe if more than 5 illnesses
Malnutrition and anorexia of ageing	Social, sensory, gastric, hormonal factors	MNA; **MEALS-ON-WHEELS** mnemonic
Polypharmacy and iatrogenesis	Exposure to the care system	Beers, STOPP-START; medication reconciliation
Functional decline	All of the above	CGA and rehabilitation
Depression and isolation	Loss, disability, chronic disease, loneliness	PHQ-2 then GDS-15; social prescribing
Sensory impairment	Age-related	Snellen, whispered voice test; spectacles, cataract surgery, hearing aids
Insomnia	Pain, nocturia, drugs, depression, environment	Sleep hygiene; avoid hypnotics
Elder abuse and neglect	Dependency, caregiver burden, cognitive impairment	Direct enquiry; **Elder Line 14567** in India

C. Latest Updates (2023–Present)

1. The shared-risk-factor model is now the accepted organising principle. The Inouye/Tinetti framework — **four shared risk factors (older age, baseline cognitive impairment, baseline functional impairment, impaired mobility) across five syndromes (pressure ulcers, incontinence, falls, functional decline, delirium)** — remains the definitional basis, with current work focused on **synergistic interactions between risk factors** and on the **basic mechanisms** underlying multifactorial conditions.

2. Screening tools have been packaged. The **Rapid Geriatric Assessment (RGA)** bundles four syndromes — **frailty, sarcopenia, geriatric anorexia and cognitive decline**. Administered to **16,615 people aged 65 and over in Missouri between 2015 and 2024**, it showed that nearly **40% reported five or more illnesses (37.3%)**, linking syndrome screening to multimorbidity risk.

3. Multiple geriatric syndromes are the rule, not the exception. A Chinese community study of 706 adults aged 60 and above (2022–23) assessed **ten syndromes — depressive symptoms, cognitive impairment, pain, falls, sleep disturbance, constipation, polypharmacy, multimorbidity, malnutrition and frailty** — and defined **"multiple geriatric syndromes" as two or more in one individual**. A 2023–24 hospital CGA study in Shanghai similarly evaluated **pain, sleep, constipation, fall risk, urinary incontinence, polypharmacy, nutritional risk and dementia** in a single assessment, using **Mini-Cog, Snellen chart and Timed Up and Go** for in-depth evaluation of those screening positive.

4. Standardised syndrome-based CGA is now guideline-mandated. The **German S3 guideline (2024)** requires **six core dimensions — self-help ability, mobility, cognition/delirium, affect, nutrition and social situation** — which

map almost exactly onto the major geriatric syndromes. The **Italian guideline (2024)**, from 117 studies, showed CGA **reduces hospitalisation, mortality, institutionalisation and delirium risk** and **improves prescribing appropriateness**.

5. WHO ICOPE reframes syndromes as losses of intrinsic capacity. The six domains — **mobility, cognition, vitality/nutrition, vision, hearing and psychological well-being** — are essentially the geriatric syndromes re-expressed as **capacities to be preserved rather than deficits to be treated**.

6. Frailty is now the strongest single predictor of adverse syndrome outcomes. A pooled odds ratio of **9.82 for incident ADL disability in frail older adults** has been reported, and the **NEJM 2024 review by Kim and Rockwood** established **"frailty-guided clinical care"**, stratifying management across **robust, pre-frailty, frailty and end-stage frailty**.

7. Indian burden. LASI wave-1 (31,464 adults aged 60 and above) found **44.3% with no geriatric condition, 32.7% with one, 15.9% with two and 7.1% with three or more**. Individual prevalences: **underweight 26.9%, dizziness 14.5%, impaired vision 13.7%, impaired hearing 9.6%, impaired cognition 9.3%, major depressive disorder 8.2%, injurious falls 5.7%, incontinence 4.0%**, with **7.4% ADL dependency**. **Both a higher number of geriatric conditions and a higher number of chronic conditions predicted ADL dependency.** Frailty affects **29.2%** and pre-frailty **58.8%**. The most disabling multimorbidity combination is **hypertension plus arthritis plus high depressive symptoms**.

D. Current Practice Update

Aspect	Textbook position	Current position (2023–2026)
Definition	Descriptive list of "I's"; concept acknowledged as poorly defined	**Operationalised** around four shared risk factors across five validated syndromes
Number recognised	14 classical "I's" plus new giants	Studies now routinely screen **8–10 syndromes simultaneously** in one CGA
Screening	Clinician-led, opportunistic	**Packaged instruments (Rapid Geriatric Assessment)** and **guideline-mandated core domain sets**
Framing	Deficits and presenting problems	Complemented by **WHO intrinsic capacity** — the same domains framed as capacities
Frailty	One syndrome among many	The **central organising syndrome**, used to guide treatment intensity ("frailty-guided care")
Evidence gap	Multifactorial interventions effective for falls and delirium	Evidence secure; **the barrier is implementation and policy translation**
Indian data	Not addressed	**LASI provides national prevalence**; over half of Indian elders have at least one geriatric condition

E. Exam One-Liners

- **A geriatric syndrome is a multifactorial condition that does not fit a single disease category.**
- **The core principle is homeostenosis** — reduced reserve across all organ systems.
- **Many causes, one presentation; one presentation, many causes.**
- **The series of I's:** Immobility, Instability, Incontinence, Intellectual impairment, Infection, Impairment of vision and hearing, Irritable colon, Isolation, Inanition, Impecunity, Iatrogenesis, Insomnia, Immune deficiency, Impotence.
- **New geriatric giants:** frailty, sarcopenia, anorexia of ageing, cognitive impairment.
- **Four shared risk factors:** older age, baseline cognitive impairment, baseline functional impairment, impaired mobility.
- **Five validated syndromes:** pressure ulcers, incontinence, falls, functional decline, delirium.
- **Multiple risk factor reduction beats the single-disease model** — proved by Inouye (delirium, 1999) and Tinetti (falls, 1994).
- **Iatrogenesis rises with exposure to the care system.**
- **The careless application of a label is more dangerous than the syndrome** — "demented" or "incontinent" can become a self-fulfilling prophecy leading to institutionalisation.
- **CGA is the definitive vehicle for detecting and managing geriatric syndromes.**
- **India (LASI): 55.7% of elders have at least one geriatric condition; 7.1% have three or more; ADL dependency 7.4%.**
- **The evidence is settled; the barrier is implementation.**

End of Set 1.

MD Geriatric Medicine — High-Yield Examination Answers

Set 3: Frailty • Sarcopenia • Deconditioning • Biology & Immunology of Ageing
Primary source: Pathy's Principles and Practice of Geriatric Medicine, 6th edition • Supplementary: Kane's Essentials of Clinical Geriatrics

Q1. Frailty — Pathogenesis, Diagnosis, Prevention and Management RECURS

15–20 High-Yield Points (Textbook)

A. Definition and Concept

1. **Frailty** is the condition in which a person loses the ability to carry out important, practised social activities of daily living when exposed to **psychological or stressful conditions**.
2. Frailty is a state of **pre-disability** and must be distinguished from **disability** and from **comorbidity**; it is now recognised as a **public health priority**.
3. By Fried's phenotype about **6.9%** of the older population are frail; **women are more often frail than men** of the same age.
4. Vitality peaks between **20 and 30 years**, then declines gradually; the decline is **slowed by exercise** and **accelerated by disease**, but everyone eventually crosses the **frailty threshold** if they live long enough.
5. Frailty begins a cascade: **Healthy → Frailty → Disability → Hospitalisation → Institutionalisation → Death** (recovery is possible at each step).

B. Pathogenesis

6. Causes are **multifactorial**: normal ageing physiology interacts with **genes, lifestyle, environment and disease**.
7. In most people frailty results from failure to generate **adequate muscle power** and/or failure of **executive function** to use the available capacity appropriately.
8. **Chronic disease** causes frailty mainly through **excess cytokines** (reduce muscle mass, food intake and cognition) and through a fall in **testosterone**.
9. **Congestive heart failure**: fall in VO_2max; left-sided failure causes gut wall oedema → **bacterial translocation** → endotoxin → **TNF-α** → anorexia, muscle loss, weight loss, hypoalbuminaemia and hypocholesterolaemia. **Weight loss and hypocholesterolaemia are the best predictors of poor outcome.**
10. Angiotensin II activation cleaves actomyosin with clearance by the **ubiquitin–proteasome system**; **ACE inhibitors reverse weight loss and frailty** in some patients with CHF.
11. **COPD**: loss of endurance, poor intake, raised resting metabolic rate, low testosterone and high cytokines. **Diabetes**: low testosterone, high angiotensin II, high cytokines, neuropathy, sarcopenia, poor executive function and accelerated atherosclerosis — mortality is increased in frail diabetics.
12. **Anaemia** causes reduced endurance, weakness, orthostasis, falls, disability and death; **erythropoietin or darbepoetin-α** can reverse this — an example of **single-cause reversible frailty**.
13. **Anorexia of ageing**: social isolation and low mood, reduced smell and raised taste threshold, reduced gastric compliance and adaptive relaxation causing **early satiety**, excess **cholecystokinin**, and high cytokines. In men falling testosterone raises **leptin** and worsens anorexia.
14. Reversible causes of weight loss are remembered by the **MEALS-ON-WHEELS** mnemonic (Medications, Emotional, Alcohol/abuse, Late-life paranoia, Swallowing, Oral, Nosocomial infection, Wandering/dementia, Hyperthyroidism–hypercalcaemia–hypoadrenalism, Enteral problems, Eating problems, Low-salt/low-cholesterol diets, Stones).
15. **Sarcopenia** is the core biological substrate, driven by falling testosterone (**1% per year after age 30**), low IGF-1, low vitamin D, raised **myostatin**, and raised **TNF-α, IL-6 and CRP**. **Progranulin** is described as a biomarker of frailty.

C. Diagnosis

16. Two conceptual models: the **phenotype model** (Fried's criteria, Frailty Trait Scale, FRAIL scale) and the **cumulative deficit model** (Frailty Index, Clinical Frailty Scale).
17. Three commonly compared scales are the **Cardiovascular Health Study (Fried)**, the **Study of Osteoporotic Fractures (SOF)** index, and the **FRAIL** scale of the International Association of Nutrition and Aging.
18. **Principle of hierarchy**: if a patient is severely ADL-dependent it is not worthwhile assessing frailty; if a patient is robust or pre-frail it is not worthwhile assessing basic ADLs.

D. Management and Prevention

19. Management follows the **FRAIL screen algorithm** (Morley): **F**atigue – exclude depression, sleep apnoea, hypothyroidism, anaemia, hypotension and vitamin B12 deficiency; **R**esistance – treat sarcopenia with **resistance exercise plus protein 1 g/kg/day**; **A**erobic – aerobic and balance exercise; **I**llnesses – if more than five, consider **deprescribing**; **L**oss of weight – look for treatable causes (MEALS-ON-WHEELS).
20. Hormonal approaches described: **testosterone replacement** increases muscle mass, strength and power even in non-hypogonadal men; **selective androgen receptor molecules (SARMs)** — steroidal (nandrolone, oxymetholone, oxandrolone) and non-steroidal (2-quinoline, coumarin, phthalimide, bicalutamide, acetothiolutamide); **creatine with exercise** may improve muscle performance.
21. **Prevention** is lifelong: regular exercise keeps the life-slope above the frailty threshold, along with adequate protein

and energy intake, correction of vitamin D and B12 deficiency, tight control of chronic disease, avoidance of polypharmacy, and **comprehensive geriatric assessment** to detect pre-frailty early.

Extra Points to Remember (Recurring Topic)

- **Frailty vs disability vs comorbidity** (Fried 2004): frailty = loss of physiological reserve; disability = loss of function; comorbidity = two or more diseases. They overlap but are not the same — a favourite examiner question.
- The **stressor** is the key concept: a frail person is precipitated into disability by a minor event (urinary infection, a new drug, a short admission) that a robust person would tolerate.
- **Frailty can be single-cause and fully reversible** — anaemia corrected with iron, folate, vitamin B12 or erythropoietin is the classic example quoted in Pathy.
- Draw the **frailty threshold graph** (VO_2max / cardiac output / balance / muscle strength against age, with an "exercise" line above and a "disease" line below the threshold) — earns marks quickly.
- Remember both mnemonics: **FRAIL** (screening and management) and **MEALS-ON-WHEELS** (weight loss).

Latest Updates (2023–Present)

- **Indian burden is far higher than the textbook figure.** Analysis of **LASI wave-1** published in the *Indian Journal of Medical Research* (2024) found **about 30% of Indians aged 60 and above were frail and 58% pre-frail**; rural 31% versus urban 25.6%; women 32.2% versus men 27.4%. A 2024 meta-analysis of Indian healthcare settings (*J Family Med Prim Care*) reported pooled frailty of **42.3%** and pre-frailty of **39.8%** in clinic and hospital populations.
- **ICFSR International Clinical Practice Guidelines** remain the operative standard: screen all adults over 65 with a validated tool (FRAIL, gait speed or PRISMA-7), then offer **progressive resistance training**, address reversible causes, correct **vitamin D deficiency**, provide protein-energy supplementation where indicated, and review medications. **No drug is approved for frailty.**
- **SPRINTT trial** (Bernabei et al.) showed that a multicomponent physical activity plus nutrition programme reduced incident **mobility disability** in community-dwelling frail and sarcopenic older adults with SPPB 3–9 — the strongest randomised evidence to date.
- **Network meta-analyses (2024–2026)** consistently place **multicomponent exercise (resistance + aerobic + balance, with or without a cognitive task) combined with protein supplementation** at the top of the ranking for reversing frailty; protein target has moved to **1.0–1.5 g/kg/day**.
- The **Japanese Nutritional Management Guidelines for Sarcopenia and Frailty (2025)** recommend energy as well as protein supplementation, noting that effects in frail and sarcopenic elders are at least as large as in healthy elders.
- **Deprescribing** is now formally an anti-frailty intervention; anticholinergics, psychotropics and inappropriately continued statins are specifically implicated in worsening frailty status.
- **WHO ICOPE (2nd edition, 2025)** shifts the operational frame from a single frailty syndrome to **intrinsic capacity** across six domains (locomotion, vitality, cognition, psychological, vision, hearing), which is the model being adopted for primary care screening.
- The **2023 update of the Hallmarks of Ageing** (López-Otín, *Cell*) formally added **chronic inflammation** and **dysbiosis**, giving biological legitimacy to the inflammaging basis of frailty.
- **India-specific service context**: NPHCE geriatric clinics and Regional Geriatric Centres, the **Ayushman Vay Vandana Card** (free cover of Rs 5 lakh for all citizens aged 70 and above, irrespective of income), **Rashtriya Vayoshri Yojana** for assistive devices, and **Elder Line 14567** for social support — all relevant to a frailty care plan in an Indian answer.

Current Practice Update

Issue	Textbook (Pathy 6e)	Current Evidence (2023–present)
Prevalence	About 6.9% of older people are frail	Highly population-dependent; **India ~30% frail, 58% pre-frail** (LASI); up to 42% in Indian clinical settings
Drug therapy	Considerable emphasis on testosterone, SARMs, creatine, ACE inhibitors	**No pharmacological agent is recommended for frailty**; testosterone is not an anti-frailty indication; exercise and nutrition are first-line
Protein target	1 g/kg/day	**1.0–1.5 g/kg/day**, higher in acute illness, with leucine-enriched supplements where intake is poor
Exercise prescription	Resistance plus aerobic and balance exercise	**Multicomponent programme** (resistance + power + balance + aerobic, ideally with a cognitive/dual task) for 3–5 months, ranked superior to any single modality
Conceptual frame	Frailty as a discrete pre-disability syndrome	Moving to **intrinsic capacity** (WHO ICOPE 2nd edition) and to a **frailty–sarcopenia continuum**
Medication review	Deprescribe if more than five illnesses	Deprescribing is a **core, evidence-supported intervention** in its own right, independent of illness count

Exam One-Liners

- Frailty = **pre-disability**; disability = established loss of function.
- Fried phenotype prevalence in Pathy = **6.9%**; in India (LASI) = **~30%**.
- Best predictors of poor outcome in CHF-related frailty = **weight loss and hypocholesterolaemia**.
- Management mnemonic = **FRAIL**; weight-loss mnemonic = **MEALS-ON-WHEELS**.
- Only interventions with consistent benefit = **multicomponent exercise + protein + deprescribing + CGA**.
- Biomarker named in Pathy = **progranulin**; inflammatory triad = **TNF-α, IL-6, CRP**.

Q2. Fried's Criteria

15–20 High-Yield Points (Textbook)

1. Fried's criteria constitute the **physical phenotype of frailty**, derived from the **Cardiovascular Health Study (CHS)** and published by Linda Fried and colleagues in 2001.
2. They provide the first **objective, operational definition** of frailty and remain the reference standard against which other tools are compared.
3. **Five components**: unintentional weight loss, exhaustion, weakness, slow walking speed, and low physical activity.
4. **Weight loss**: unintentional loss of **10 lb (about 4.5 kg) in the preceding year** (or 5% of body weight).
5. **Exhaustion**: **self-reported**, based on two items of the CES-D scale ("everything I did was an effort" / "I could not get going" for three or more days in the last week).
6. **Weakness**: **grip strength in the lowest 20%** of the population, adjusted for **sex and body mass index**.
7. **Slowness**: walking speed over **15 feet in the slowest 20%**, adjusted for **sex and height**.
8. **Low physical activity**: weekly energy expenditure in **kcal per week in the lowest 20%**, calculated from a physical activity questionnaire.
9. **Scoring**: **0 = robust; 1–2 = pre-frail (intermediate); 3 or more of 5 = frail**.
10. By this definition about **6.9%** of older people are frail, and **women are classified as frail more often than men** of the same age.
11. The phenotype rests on a **cycle of frailty**: sarcopenia leads to reduced strength and VO_2max, which reduce walking speed and activity, which lower energy expenditure and intake, causing weight loss and further sarcopenia.
12. Fried also separated the concepts of **frailty, disability and comorbidity**, showing they overlap but predict outcomes independently.
13. The phenotype predicts **falls, worsening mobility, incident disability, hospitalisation and death** over three years.
14. **Sarcopenia is regarded as the biological substrate** of the Fried physical phenotype, and the combined construct "physical frailty and sarcopenia" is now used.
15. Two simpler validated alternatives are given alongside it in Pathy: the **Study of Osteoporotic Fractures (SOF) index** and the **FRAIL scale**.
16. **SOF index** (3 items, 2 or more = frail): weight loss; **inability to rise from a chair five times without using the arms**; reduced energy level.
17. **FRAIL scale** (5 items, 3 or more = frail): **F**atigue; **R**esistance (cannot climb one flight of stairs); **A**mbulation (cannot walk one block); **I**llnesses (more than five); **L**oss of weight (5% or more).
18. **Advantages**: objective, reproducible, biologically coherent, extensively validated.
19. **Limitations**: requires a **dynamometer and a measured walking course**; excludes **cognition, mood and social factors**; cannot be applied to wheelchair-bound or acutely ill patients; population-specific cut-offs are needed.

Comparison of the Three Scales (Pathy Table 97.1)

Cardiovascular Health Study (Fried)	Study of Osteoporotic Fractures	FRAIL (IANA)
Weight loss (10 lb in a year)	Weight loss	Fatigue
Exhaustion (self-report)	Inability to rise from a chair five times without using arms	Resistance (climb one flight of stairs)
Weakness (grip strength, lowest 20%)	Reduced energy level	Aerobic (walk one block)
Walking speed (15 ft, slowest 20%)	—	Illnesses (more than five)
Low physical activity (kcal/week, lowest 20%)	—	Loss of weight (5%)
3 or more of 5 = frail	**2 or more of 3 = frail**	**3 or more of 5 = frail**

Latest Updates (2023–Present)

- Fried's phenotype remains the **most used research definition worldwide** and was the instrument used for the national Indian frailty estimates from **LASI wave-1** (modified Fried criteria: exhaustion, weak grip, slow gait, unintentional weight loss, low physical activity).
- Population-specific cut-offs have replaced the original "lowest 20%" approach in clinical use. **Grip strength**: EWGSOP2 uses **<27 kg in men and <16 kg in women**; **AWGS 2019 (Asian, relevant to India)** uses **<28 kg in men and <18 kg in women**. Indian community studies report even lower normative values, so applying Western cut-offs overestimates weakness.
- **Gait speed** cut-off has been standardised at **0.8 m/s or slower** over 4 metres (EWGSOP2); AWGS uses **1.0 m/s** for the Asian population.
- The **Global Leadership Initiative in Sarcopenia (GLIS, 2024)** explicitly separates **sarcopenia** (a muscle disease) from **frailty** (a multisystem syndrome), correcting the earlier tendency to use Fried's criteria as a proxy for sarcopenia.
- Because Fried's phenotype is time-consuming, **FRAIL, PRISMA-7, gait speed alone and the Clinical Frailty Scale**

are preferred for routine screening, with Fried's criteria reserved for research and for confirmation.

- Growing recognition of **cognitive frailty** and **social frailty** (a Social Frailty Index was derived from LASI) as dimensions the purely physical Fried phenotype misses.

Current Practice Update

Issue	Textbook (Pathy 6e)	Current Evidence
Cut-offs	Lowest 20% of the study population for grip, gait and activity	**Absolute, ethnicity-specific cut-offs** (AWGS for Asians: grip <28/<18 kg, gait <1.0 m/s)
Role of the tool	Presented as the primary definition of frailty	Reference standard for **research**; simpler tools (FRAIL, CFS, gait speed) preferred for **clinical screening**
Scope	Physical domain only	Recognition that **cognitive, psychological and social frailty** must be assessed separately
Relation to sarcopenia	Overlapping, sarcopenia as substrate	**GLIS 2024** keeps them conceptually distinct with separate diagnostic pathways

Exam One-Liners

- Fried = **CHS phenotype**, 2001, five criteria, **3 or more = frail, 1–2 = pre-frail**.
- The five: **weight loss, exhaustion, weakness, slowness, low activity**.
- Grip adjusted for **sex and BMI**; gait speed adjusted for **sex and height**.
- Weight loss threshold = **10 lb / 4.5 kg or 5% in one year**.
- Asian (AWGS) grip cut-offs = **28 kg men, 18 kg women**.
- SOF = **2 of 3**; FRAIL = **3 of 5**.

Q3. Clinical Frailty Scales

15–20 High-Yield Points (Textbook)

1. Frailty measurement tools derive from **two different conceptual models**, and the choice of scale follows the model chosen.
2. **Phenotype model** — frailty is a distinct biological syndrome preceding disability. Tools: **Fried's phenotype, Frailty Trait Scale, FRAIL index**. Endorsed by the **ADVANTAGE Joint Action consortium** and the **ICFSR**.
3. **Cumulative deficit model** — frailty is the accumulation of deficits (diseases, functional losses, organ and sensory impairments) with age. Tools: **Frailty Index** and the **Clinical Frailty Scale**.
4. **Rockwood** defined frailty as an **increasing number of disabilities**, which can be regarded as a comorbidity index.
5. **Frailty Index (Mitnitski and Rockwood)**: the proportion of deficits present out of the total number counted (usually 30–70 items). A value of **0.25 or above indicates frailty**; a submaximal limit of about **0.7** is observed.
6. **Clinical Frailty Scale (Rockwood, CMAJ 2005)**: a **global clinical judgement** measure of fitness and frailty, scored on a **9-point pictorial scale**.
7. CFS grades: **1 Very fit, 2 Fit/Well, 3 Managing well, 4 Living with very mild frailty (formerly "vulnerable"), 5 Mildly frail, 6 Moderately frail, 7 Severely frail, 8 Very severely frail, 9 Terminally ill** (life expectancy under 6 months).
8. CFS is **quick, needs no equipment**, and predicts institutionalisation and death; it has also been adapted for **nursing home residents**.
9. The **FRAIL scale** is a five-item questionnaire usable in the clinic without equipment (fatigue, resistance, ambulation, illnesses, loss of weight) and forms the basis of a **management algorithm**.
10. The **SOF index** (weight loss, inability to rise from a chair five times without arms, reduced energy) is a validated three-item alternative shown to predict falls, disability, fracture and death as well as longer indices.
11. The **Frailty Trait Scale** offers a continuous rather than categorical operational definition.
12. Function is assessed alongside frailty with **Katz ADL, Lawton IADL and Barthel Index**; mobility and fall risk with the **Tinetti performance-oriented assessment** and the **Timed Up and Go**.
13. **Principle of hierarchy** in choosing a scale: assess frailty in the robust and pre-frail; assess ADLs in those already dependent. Do not do both.
14. Frailty status must always be interpreted with **environmental and social information** — caregiver support, social network, participation — usually gathered by a nurse or social worker.
15. **Agreement between different frailty scales is limited** (35 published scores show poor concordance), which is the main reason no single scale is universally accepted.
16. No single **biomarker** is adequate for frailty; those studied include TNF-α, IL-6, CRP, HbA1c, testosterone, DHEA, IGF-1, vitamin D, PTH, the **CD4/CD8 ratio**, haemoglobin, albumin, soluble RAGE, DNA methylation and microRNA.
17. Neuroimaging markers are little studied, but **greater white matter hyperintensity volume and loss of white matter integrity** are associated with frailty.

Practical Comparison of Common Scales

Scale	Model	Content	Best use
Fried phenotype	Phenotype	5 physical items, needs dynamometer and walk course	Research reference standard
FRAIL scale	Phenotype	5 questions, no equipment	Clinic and community screening; drives management algorithm
SOF index	Phenotype	3 items including chair rise	Rapid screening, epidemiology
Frailty Index	Deficit accumulation	30–70 deficits, continuous score 0–1	Risk stratification, electronic health records
Clinical Frailty Scale	Deficit accumulation / clinical judgement	9-point pictorial global judgement	Acute hospital, surgical and critical care triage
Edmonton Frail Scale	Multidimensional	9 domains including cognition and mood	Preoperative and multidimensional assessment
PRISMA-7 / Gérontopôle tool	Screening	7 questions / clinician gestalt	Primary care case-finding
Gait speed / TUG	Performance	4 m walk; TUG	Single-item screen (**<0.8 m/s**, TUG **≥20 s**)

Latest Updates (2023–Present)

- **CFS version 2.0** is the current wording: level 4 is now "**living with very mild frailty**" and levels 5–8 are phrased as "living with mild / moderate / severe / very severe frailty", removing stigmatising language.

- The CFS is **only validated in people aged 65 and above** and should **not** be applied to younger adults or to those with stable lifelong disability (cerebral palsy, intellectual disability), where it misclassifies stable disability as frailty — a point emphasised after its widespread use in COVID-19 triage.
- The **electronic Frailty Index (eFI)**, calculated automatically from primary care records, is now used for population-level case-finding; the **Hospital Frailty Risk Score**, derived from ICD-10 codes at discharge, does the same for hospital data.
- CFS is now embedded in routine practice for **perioperative risk assessment, TAVI and cardiac surgery selection, oncology fitness assessment, dialysis decision-making and critical care admission**.
- **Frailty scales are increasingly used as decision aids rather than diagnoses** — a high CFS should trigger comprehensive geriatric assessment and shared decision-making, not automatic withholding of treatment.
- The **WHO ICOPE (2nd edition, 2025)** screening tool is being promoted as the entry point in low- and middle-income settings, measuring **intrinsic capacity** across six domains rather than frailty as such — directly relevant to NPHCE implementation in India.
- Indian validation work has established Hindi and regional-language versions of the **Edmonton Frail Scale, Tilburg Frailty Indicator and FRAIL scale**, but the **modified Fried phenotype** remains the instrument used in national surveys.

Current Practice Update

Issue	Textbook (Pathy 6e)	Current Evidence
CFS wording	Original 2005 labels including "vulnerable"	**CFS 2.0** wording: "living with very mild/mild/moderate/severe frailty"
Population limits	Not specified	CFS **not valid below age 65** or in stable lifelong disability
Frailty Index	Manual count of deficits	**Automated eFI** from primary care records and **Hospital Frailty Risk Score** from ICD-10 data
Purpose	Classification and prognosis	Used as a **trigger for CGA and shared decision-making**, explicitly not as a rationing tool
Screening framework	Frailty scales	**WHO ICOPE intrinsic capacity screening** as the primary-care entry point

Exam One-Liners

- Two models: **phenotype** (Fried, FRAIL, Frailty Trait Scale) and **cumulative deficit** (Frailty Index, CFS).
- Frailty Index = **deficits present / deficits counted**; frail at **≥0.25**; ceiling about **0.7**.
- CFS = **9 points**, 1 very fit to 9 terminally ill; **≥5 = frail**.
- Agreement between published frailty scales is **poor** — the main unsolved problem in the field.
- Single best one-item screen = **gait speed <0.8 m/s**.
- CFS must not be used **below age 65** or in **stable lifelong disability**.

Q4. Sarcopenia — Aetiology, Pathogenesis and Evaluation RECURS

15–20 High-Yield Points (Textbook)

A. Definition and Epidemiology

1. **Sarcopenia** is the excessive loss of muscle mass that occurs in older people, usually defined as lean tissue more than **two standard deviations below** that of young reference subjects.
2. It occurs in **13–24% of people aged 60–70 years** and in **about 50% of those over 80 years**.
3. **Dynapenia** is the age-associated loss of **muscle strength** not due to disease — the counterpart of sarcopenia, which refers to loss of **mass**. Both progress with ageing.
4. **Sarcopenic obesity** ("fat frail") describes those who lose muscle while retaining fat; longitudinally they have the **highest risk of disability and mortality**.
5. **Myosteatosis** — fat infiltration into muscle — is a separate condition linked to insulin resistance: mitochondrial failure or high circulating triglycerides cause intracellular triglyceride accumulation, which alters insulin receptor substrate function and the GLUT transporter.

B. Aetiology

6. Sarcopenia may **originate at birth**: in the **Hertfordshire cohort study** adult grip strength correlated with **birth weight**.
7. **Genetic factors**: people with a single I or double I allele of the **angiotensin-converting enzyme** gene generate more power with regular exercise than those with the D allele. Genome-wide studies implicate **HSD17B11, VCAN, ADAMTSL3, IRS1 and FTO**.
8. The best epidemiological predictors of muscle mass and strength are **age, energy intake, physical activity, IGF-1, testosterone and cytokines**.
9. **Hypovitaminosis D** is associated with reduced muscle strength and falls.
10. Disease-related contributors are the same as for frailty: **heart failure, COPD, diabetes, chronic kidney disease, anaemia, cancer cachexia and inflammatory disease**.

C. Pathogenesis

11. **Testosterone** declines by about **1% per year from age 30 in men** and falls rapidly between ages 20 and 40 in women. It inhibits movement of pluripotential stem cells into the **fat cell lineage** and stimulates the **muscle cell lineage** to produce **satellite cells**, which are essential for repair.
12. Testosterone also **stimulates muscle protein synthesis and inhibits the ubiquitin–proteasome pathway**; replacement increases muscle mass, strength, power and function even in non-hypogonadal men.
13. **Myostatin** is the principal negative regulator of muscle growth, acting through **activin IIb receptors**. Double deletion produces marked hypertrophy in mice ("mighty mouse"), cattle, whippets and one reported human; a **soluble activin IIb receptor** scavenges the ligand and produces muscle and bone hypertrophy.
14. **Motor unit dysfunction**: firing rate falls significantly in the **old-old (over 80)**. **Ciliary neurotrophic factor (CNTF)** declines with age in parallel with strength. **C-terminal agrin fragment** and **neurofilament light chain**, markers of neural degradation, are raised in a subset of sarcopenic patients.
15. **Cytokines**: excess **TNF-α** and **IL-6** cause loss of muscle strength; high **CRP and IL-6** correlate with reduced grip strength and physical performance.
16. **Vascular and metabolic factors**: raised homocysteine and peripheral vascular disease impair muscle blood flow causing atrophy; **creatine with exercise** may improve performance.
17. The final common pathway is an **imbalance of the daily muscle renewal cycle** — either excess atrophy and apoptosis, or reduced hypertrophy and satellite cell production.
18. **Biochemistry (Pathy Fig 97.7)** — **Anabolic arm**: GH / testosterone / creatine → IGF-1 and mechano-growth factor → PI3K → AKT → mTOR → S6K1 → hypertrophy. **Catabolic arm**: glucocorticoids, cytokines (TNF receptor I → IKK → NF-κB), and inactive AKT → **FOXO** → **atrogin-1 (MAFbx)** and **MuRF1** → ubiquitin–proteasome protein degradation → atrophy. **Myostatin–SMAD3** inhibits MyoD; caspase-3 and caspase-8 mediate apoptosis and actomyosin cleavage.

D. Evaluation

19. The best measure is **appendicular skeletal muscle mass by DXA divided by height in metres squared**. Alternatives are **MRI, CT and bioelectrical impedance**; DXA and MRI correlate highly, and **ultrasound** is proving an excellent measure of muscle loss. MRI is superior for visceral compartments, muscle volume, fat infiltration and ectopic fat; **BIA** is the most accessible in primary care but is only an approximation, better for limbs than trunk.
20. Several operational definitions exist — **FNIH Biomarkers Consortium, EWGSOP and AWGS** — and they differ in cut-offs and severity grading, but all agree on including **muscle strength, muscle mass and physical performance**.
21. **Candidate biomarkers**: procollagen type III N-terminal peptide (P3NP), **D3-creatine dilution** (correlates highly with MRI muscle mass), C-terminal agrin fragment, myostatin and follistatin, GDF-11 and GDF-15, and leptin in sarcopenic obesity. None is validated alone.
22. **Treatment**, as given in the frailty chapter, is **resistance exercise plus protein 1 g/kg/day**, correction of vitamin D deficiency, treatment of underlying disease, and consideration of testosterone or SARMs.

> **Extra Points to Remember (Recurring Topic)**

- Learn the **three-word definition triad**: **mass + strength + performance**. Any answer that mentions only muscle mass loses marks.
- Distinguish clearly: **sarcopenia** (mass), **dynapenia** (strength), **myosteatosis** (fat within muscle), **cachexia** (inflammation-driven loss of lean ± fat mass in advanced disease), **sarcopenic obesity** (worst prognosis).
- **Primary sarcopenia** = age-related only. **Secondary sarcopenia** = disease-related, inactivity-related or nutrition-related. **Acute sarcopenia** = under 6 months, typically after hospitalisation; **chronic** = 6 months or more.
- Be able to draw the **anabolic (IGF-1/PI3K/AKT/mTOR) versus catabolic (FOXO/atrogin-1/MuRF1/ubiquitin-proteasome)** diagram — it answers "pathogenesis" in one figure.
- The single most examinable formula: **ASMI = appendicular skeletal muscle mass (kg) / height (m)^2**.
- Sarcopenia has an **ICD-10 code (M62.84)** — it is now formally a disease, not a normal ageing change.

Latest Updates (2023–Present)

- **GLIS — Global Leadership Initiative in Sarcopenia (Delphi consensus, *Age and Ageing* 2024)** produced the first **global conceptual definition**, uniting EWGSOP, AWGS, ANZSSFR, SDOC and the sarcopenic obesity group. Key conclusions: sarcopenia is a **muscle disease**; **low muscle mass is the cardinal, mandatory feature**; **muscle strength** and **muscle-specific function** (function attributable to muscle rather than general physical performance) are the other components; and sarcopenia is **conceptually distinct from frailty, cachexia and malnutrition**.
- The **GLIS outcomes working group consensus (2025)** defined the health outcomes that should be measured once sarcopenia is diagnosed — falls, fractures, disability, hospitalisation, quality of life and mortality — and the preferred tools and cut-offs. A single global **operational** definition with numerical cut-offs is still pending.
- **EWGSOP2 algorithm (Find–Assess–Confirm–Severity)** remains the working clinical pathway: **SARC-F questionnaire ≥4 → assess strength** (grip <27 kg men, <16 kg women; or five-times chair stand >15 s) **→ confirm with muscle mass** (ASMI by DXA <7.0 kg/m^2 men, <5.5 kg/m^2 women) **→ grade severity by performance** (gait speed ≤0.8 m/s, SPPB ≤8, TUG ≥20 s, 400 m walk test).
- **AWGS 2019 Asian cut-offs** are the appropriate ones for Indian patients: grip **<28 kg men, <18 kg women**; ASMI by DXA **<7.0 / <5.4 kg/m^2**, by BIA **<7.0 / <5.7 kg/m^2**; gait speed **<1.0 m/s**; five-times chair stand **≥12 s**. AWGS also introduced "possible sarcopenia" for primary care, allowing lifestyle intervention to start before DXA is available — important given limited DXA access in India.
- Indian community studies report sarcopenia prevalence of roughly **17–25% in community-dwelling elders** and higher in hospital and nursing home populations, with a substantial contribution from **low dietary protein intake** in largely cereal-based diets.
- **Nutrition**: current targets are **1.0–1.5 g/kg/day of protein**, distributed as **25–30 g of high-quality protein per meal**, with **leucine-enriched essential amino acids or HMB** where intake is inadequate, plus correction of vitamin D deficiency. Network meta-analyses (2025) confirm that **resistance exercise plus protein supplementation** is superior to either alone.
- **Pharmacotherapy remains investigational**: myostatin and activin pathway agents (bimagrumab, apitegromab), SARMs, and testosterone have improved lean mass without convincing functional gains. A new practical concern is **preservation of muscle mass during GLP-1 receptor agonist weight loss**, where up to 40% of weight lost may be lean mass — muscle-sparing strategies are an active research area.
- Sarcopenia is increasingly measured opportunistically from **CT scans at the L3 vertebral level** (skeletal muscle index), used as a prognostic marker in oncology and before major surgery.

Current Practice Update

Issue	Textbook (Pathy 6e)	Current Evidence (2023–present)
Definition	Loss of muscle mass more than 2 SD below young adults	**GLIS 2024**: a muscle disease requiring **low muscle mass (cardinal) + low strength + impaired muscle-specific function**
Diagnostic entry point	Muscle mass measurement (DXA)	**Muscle strength first** (EWGSOP2 / AWGS), mass used to confirm; SARC-F for case-finding
Cut-offs	Not specified numerically	EWGSOP2 grip **<27/<16 kg**; **AWGS <28/<18 kg for Asians**; ASMI **<7.0/<5.4–5.5 kg/m^2**
Relation to frailty	Overlapping; "two sides of the same coin"	**Formally distinct entities** with separate pathways (GLIS)
Protein target	1 g/kg/day	**1.0–1.5 g/kg/day**, 25–30 g per meal, leucine-enriched supplements
Drug therapy	Testosterone and SARMs discussed favourably	**No approved drug**; myostatin/activin agents in trials; focus shifted to exercise and nutrition
Disease status	Described as a geriatric syndrome	Recognised disease with **ICD-10 code M62.84**

Exam One-Liners

- Prevalence: **13–24% at 60–70 years, ~50% over 80 years**.

- **ASMI = appendicular skeletal muscle mass / height^2**, measured by DXA.
- Testosterone falls **1% per year after 30**; drives satellite cell production.
- Myostatin acts via **activin IIb receptor**; blockade causes hypertrophy.
- Atrophy genes = **atrogin-1 (MAFbx) and MuRF1**, activated by **FOXO** and **NF-κB**.
- Screening questionnaire = **SARC-F**; score **≥4** is positive.
- Indian/Asian grip cut-offs (**AWGS**) = **28 kg men, 18 kg women**.
- Treatment = **progressive resistance exercise + protein 1.0–1.5 g/kg/day + vitamin D**.

Q5. Deconditioning and Its Prevention

15–20 High-Yield Points (Textbook)

A. Definition and Magnitude

1. **Deconditioning (functional decline)** is the loss of the ability to perform basic activities of daily living, attributed primarily to the effects of immobilisation by **forced bed rest, immobilising devices (catheters, drips), restraint use, and lack of encouragement of independence in self-care**.
2. **Immobility** is defined as restriction of time spent out of bed or chair by **medical orders, restrictive devices, chemical restraints (antipsychotics), lack of mobility aids, or lack of human assistance and encouragement**.
3. **Functional decline occurs in one-third to one-half of hospitalised older patients**; as many as **one-third of older inpatients are restricted to bed or chair rest** during admission.
4. A systematic review of 30 studies found that between **15% and 76%** of hospitalised elders show diminished performance in at least one ADL at discharge; of these, **only half recover function by three months**, and for many the loss of independent living is **permanent**.
5. Functional decline is regarded as a **profound marker of morbidity and mortality**. Just **ten days of bed rest in healthy older adults** produces measurable functional impairment (Kortebein).

B. Causes

6. **Medical**: musculoskeletal (arthritis, osteoporosis, hip and femoral fracture, foot problems), neurological (stroke, Parkinson's disease, neuropathy, normal-pressure hydrocephalus, dementia), cardiovascular (severe heart failure, angina, claudication), severe COPD, acute and chronic pain, malnutrition, and severe systemic illness.
7. **Sensory**: impaired vision, reduced kinaesthetic sense, reduced peripheral sensation.
8. **Iatrogenic and environmental**: forced immobility in hospitals and nursing homes, inadequate mobility aids, cluttered or uneven surroundings, poorly designed chairs and beds, stairs.
9. **Psychological**: depression, apathy, lack of motivation, **fear of falling**, and the health belief that rest is beneficial to recovery.
10. **Drugs**: sedatives and hypnotics (drowsiness, dizziness, ataxia, delirium), typical antipsychotics (rigidity), and antihypertensives causing orthostatic hypotension or bradycardia.
11. **Caregiver-driven dependency**: well-meaning staff and families who bathe, dress and wheel patients who could do these things themselves — "the hospital breeds dependency".

C. Complications of Immobility (system-wise)

12. **Skin**: pressure ulcers (risk multiplied by immobility plus low serum albumin). **Musculoskeletal**: muscle atrophy and loss of strength and endurance, reduced fibre size and capillarity, contractures, disuse osteoporosis with increased bone resorption and reduced formation, degenerative joint disease, **hypercalcaemia**.
13. **Cardiovascular**: deconditioning, reduced plasma volume, **orthostatic hypotension**, deep vein thrombosis and pulmonary embolism. **Respiratory**: reduced tidal volume, atelectasis, aspiration pneumonia.
14. **Gastrointestinal and genitourinary**: anorexia, constipation, faecal impaction and incontinence, urinary retention, urinary infection, bladder calculi, functional incontinence.
15. **Metabolic**: negative **nitrogen and calcium** balance, impaired glucose tolerance, altered body composition and altered drug pharmacokinetics. **Psychological**: sensory deprivation, delirium, depression and anxiety.
16. Reduced bone mass from decreased weight-bearing increases the likelihood that a fall will cause **serious injury**; balance and coordination problems increase fall incidence itself.

D. Risk Factors and Prevention

17. **Risk factors for functional decline in hospitalised elders** (Kane): age **75 and above**, cognitive impairment (missing more than 15 of the first 21 MMSE items), **dependence in two or more IADLs before admission**, presence of a pressure sore, baseline functional dependency, and a history of low social activity.
18. **Prevention begins with the bed rest order**: careful consideration is warranted before prescribing bed rest, and mobility should be assessed for the ability to move in bed, transfer and ambulate safely, including the need for assistance and assistive devices.
19. **Promote mobility actively**: nursing staff should encourage and assist position change in bed, transfer to a chair and ambulation; **organised group walks** on the ward at set times provide diversion and involve the patient in recovery; **bed and toilet seat height should be matched to the patient's lower leg height** to allow safe transfers.
20. **Refer to physiotherapy and occupational therapy** for problems; rehabilitation therapists may supply transfer aids — trapeze, transfer pole or bar, or a raised one-quarter or half-length siderail attached near the top of the bed as a bed-mobility device.
21. **Avoid physical restraints and restrictive full-length siderails**: research demonstrates restrictive devices can be safely eliminated and are associated with strangulation, asphyxiation and the full spectrum of immobility complications. Avoid unnecessary catheters and infusion lines that tether the patient.
22. **Facilitate observation instead of restraint**: nurse high-risk patients near the nursing station, encourage time out of the room, encourage family visits at mealtimes and at night, provide communal dining, and use volunteers or paid companions where family cannot stay.
23. **Multicomponent programmes**: the **Hospital Elder Life Program (HELP)** prevents cognitive and functional decline

in hospitalised elders; **acute geriatric units staffed by an interdisciplinary team** using multidimensional functional evaluation improve outcomes; environmental modification (primary colours, lower windows, better furniture and room design) helps older patients retain function.

24. **Assessment of the immobile patient** should cover the perceived cause of immobility, medical conditions and treatments, pain, mood, fear and motivation, nutritional status including **protein and 25-hydroxy vitamin D**, and both the physical and social environment including caregiving interactions.

Latest Updates (2023–Present)

- **"New horizons in hospital-associated deconditioning" (*Age and Ageing*, 2024)** reframed the syndrome as **a global condition of body and mind**: it is not only loss of muscle (i.e. **acute sarcopenia**) but also **cognitive deconditioning**, defined as delayed mental processing lying on a spectrum with frank delirium at its severe end.
- Quantitative figures now commonly quoted: muscle deconditioning begins within **two days** of immobility, with **2–5% loss of muscle mass per day** when the legs are inactive, and up to **20% loss of baseline strength within one week**.
- Accelerometer studies show hospitalised older adults are **sedentary for over 80% of the day**, and that most in-hospital walking occurs in short, unsupervised bouts — the target of current quality improvement work.
- **CHERISH cluster randomised trial (*JAMA Internal Medicine*, 2022)** — the ward-based **Eat Walk Engage** programme (nutrition, mobility and cognitive engagement embedded in ward routine) reduced hospital-associated complications in older inpatients.
- The **Frailty Care Bundle (2024)** — mobilisation, nutrition and cognitive engagement delivered by nursing staff — reduced hospital-associated decline in older orthopaedic trauma patients and is the current implementation template.
- Campaign-style interventions endorsed by the **British Geriatrics Society (2023)** — **"End PJ Paralysis"** and **"Get Up, Get Dressed, Get Moving"** — are now standard ward culture initiatives, targeting daily out-of-bed time before 11 a.m., own clothing, and removal of unnecessary lines and catheters.
- **Prehabilitation** (structured exercise, nutrition and psychological preparation before elective surgery or chemotherapy) has become a mainstream strategy for preventing perioperative deconditioning in older patients.
- In critical care, the **ABCDEF bundle** with early mobilisation reduces ICU-acquired weakness and post-intensive care syndrome; in general medicine, **hospital-at-home** and **early supported discharge** models reduce deconditioning by avoiding the ward environment altogether.
- Post-discharge, trials of **high-intensity progressive home-based physical therapy** (2024) improved function but did not consistently outperform good-quality usual care, indicating that **frequency and consistency of therapy matter as much as intensity**.
- **Indian relevance**: deconditioning is aggravated by high nurse-to-patient ratios, dependence on family attendants who often do everything for the patient, limited inpatient physiotherapy, and low protein intake. Practical measures — **training the family attendant** as a mobility coach, early removal of catheters, and daily chair transfer targets — are the highest-yield interventions in Indian wards.

Current Practice Update

Issue	Textbook (Pathy 6e / Kane)	Current Evidence (2023–present)
Concept	Physical functional decline from immobility	**Whole-body and mind syndrome** including **cognitive deconditioning** and **acute sarcopenia**
Speed of loss	Described qualitatively; 10 days of bed rest harmful	Quantified: **2–5% muscle loss per day**, up to **20% strength loss in a week**, starting within **48 hours**
Measurement	ADL scales at discharge	**Accelerometry** and direct observation of sedentary time as ward quality metrics
Intervention model	Individual nursing and therapy measures; HELP; geriatric units	**Bundled, ward-culture interventions** — CHERISH/Eat Walk Engage, Frailty Care Bundle, End PJ Paralysis
Timing	Prevention during admission	**Prehabilitation before** admission and structured rehabilitation **after** discharge
Setting	Acute ward with restraint reduction	**Hospital-at-home and early supported discharge** to avoid the ward entirely where feasible

Exam One-Liners

- Deconditioning = **loss of ability to perform basic ADLs** due to immobilisation.
- Occurs in **one-third to one-half** of hospitalised older patients; **only half recover by 3 months**.
- Muscle loss = **2–5% per day** of inactivity; strength loss up to **20% in one week**.
- Immobility causes **negative nitrogen and calcium balance** and **hypercalcaemia**.
- Key hospital risk factors: **age 75+, cognitive impairment, prior IADL dependence, pressure sore, low social**

activity.

- Bed and toilet seat height should equal the patient's **lower leg height**.
- Prevention programmes: **HELP, ACE units, CHERISH/Eat Walk Engage, Frailty Care Bundle, End PJ Paralysis**.

Q6. Theories of Ageing

15–20 High-Yield Points (Textbook)

A. General Principles

1. Ageing is now accepted to be a **multifactorial process**; extended longevity is consistently associated with **enhanced metabolic capacity and stress response**.
2. Genetics matters — each species has a characteristic longevity — but **heritability accounts for only about 35% or less of the variance in lifespan**, while **environmental factors account for more than 65%**.
3. **Genes specifically selected to promote ageing are unlikely to exist**; ageing is not a programmed adaptive trait.
4. Kane groups all theories into **three categories**: (i) **accumulation of damage to informational molecules**, (ii) **regulation of specific genes**, and (iii) **depletion of stem cells**.

B. Damage-Based (Stochastic) Theories

5. **Somatic mutation theory**: greater susceptibility to mutagenesis or defective DNA repair drives ageing. Support: **species longevity correlates positively with DNA repair enzyme capacity**. Against: the human spontaneous mutation rate is **too low** to account for the changes required, and there is no evidence of a general failure of repair systems — though limited maintenance may allow somatic damage to accumulate.
6. **Error catastrophe theory (Orgel)**: errors in DNA, RNA and protein synthesis each amplify the others until an error catastrophe occurs; translation was thought the likeliest source. Against: increased translational errors have **not** been found in vivo or in vitro, and **amino acid substitutions do not increase with age**, although post-translational changes such as glycosylation may alter enzyme activity.
7. **Free radical / oxidative damage theory (Harman)**: superoxide radicals, the major by-product of oxidative metabolism, react with DNA, RNA, proteins and lipids to cause cellular damage. Scavenging enzymes and vitamins C and E are protective.
8. Evidence **against** a simple free radical theory: there is **no significant loss of scavenging enzymes with age**, and vitamins C and E do **not** increase longevity in experimental animals.
9. Evidence **for**: overexpression of antioxidative enzymes retards age-related oxidative damage and extends maximum lifespan in **transgenic fruit flies**, and **caloric restriction lowers oxidative stress and extends maximum lifespan in rodents**.
10. **Loss of molecular fidelity (Hayflick)**: genes do not drive ageing — the general loss of molecular fidelity does. Every molecule becomes subject to **thermodynamic instability**. This distinguishes ageing from disease, because ageing occurs in every multicellular animal that reaches a fixed size at reproductive maturity, occurs after reproductive maturity in all species, and has the same universal molecular aetiology.

C. Programmed and Genetic Theories

11. **Evolutionary theory (Darwin)**: current organisms result from an incrementally accumulative evolutionary process — "survival of the fittest" — organisms surviving longer had more opportunity to breed and propagate their design.
12. **Gene regulation theory**: specific genes regulate ageing. Genes extending lifespan have been found in **yeast, nematodes and fruit flies**, and they reinforce the importance of **metabolic capacity and stress response**. DNA microarray work found relatively few genes changed in ageing human fibroblasts, with **downregulation of mitosis-control genes** proposed as a general cause — but the pattern is not reproduced in other tissues, suggesting different mechanisms in different tissues.
13. **Replicative senescence (Hayflick limit)**: cells in culture divide a fixed number of times and then enter **replicative senescence**. Human fibroblasts manage about **50 doublings** (most cell types 60–70); replicative life in vitro correlates inversely with donor age. Replicative senescence is believed to have **evolved to protect higher organisms from cancer**.
14. **Telomere theory**: with each division a portion of the terminal chromosome (the **telomere**) is not replicated and shortens — about **50–100 base pairs are lost per replication**. **Telomerase** adds bases back; transfection of its catalytic component into senescent cells extends telomeres and replicative lifespan and restores a young pattern of gene expression.
15. Telomere caveats: relevance in vivo remains unknown; **studies of the oldest old show no relationship between telomere length and mortality**, though healthy centenarians had significantly longer telomeres than unhealthy ones. **Telomerase inhibitors are potential anticancer therapies**. **Stress-induced premature senescence** shows that oxidative stress and oncogene activation can produce an identical senescent phenotype without telomere shortening.
16. **Stem cell depletion**: convergence of the above mechanisms reduces proliferative potential. Cells are of three types — continuously replicating (epidermal, gastrointestinal, haematopoietic), replicating on challenge (liver), and non-replicating (neurons, cardiac and skeletal muscle) — and in non-replicating tissues, cell loss produces a **permanent deficit** (for example, loss of dopaminergic neurons affecting gait and balance).

D. Systemic and Integrative Theories

17. **Neuroendocrine theory (Dilman)**: age-related hypothalamic functional decline from **reduced sensitivity to feedback**. Controversial, because pituitary secretion also depends on sex, body composition, stress, illness, comorbidity, medication, frailty, caloric intake, immune status, activity and cognition.
18. **Immunological theory**: thymic involution after puberty with falling **thymosins**, reduced **IL-2** production and receptor expression, and diminished lymphocyte proliferative response — predisposing to infection, autoimmunity and cancer.

19. **Network and remodelling theories (Franceschi)**: ageing is **active remodelling**, not simple decline, with **inflammaging** at its centre. The **"seven pillars"** of ageing and age-related disease are adaptation to stress, loss of proteostasis, stem cell exhaustion, metabolic derangement, macromolecular damage, epigenetic modification and inflammation.
20. **Hormesis**: stress is not necessarily deleterious — **low-intensity stimulation activates maintenance and repair mechanisms** and improves health, whereas stress exceeding the organism's adaptive capacity causes disease. This is the theoretical basis for exercise, caloric restriction and the Mediterranean diet.
21. **Nine mechanisms are given as the fundaments of ageing** in Pathy: **genomic instability, telomere attrition, epigenetic modifications, loss of proteostasis, deregulated nutrient sensing, mitochondrial dysfunction, cellular senescence, stem cell exhaustion and altered intercellular communication**.
22. **Caloric restriction** and mutations in nutrient and growth signalling pathways extend lifespan by **30–50%** in preclinical models; in humans, long-term caloric restriction and intermittent fasting produce favourable metabolic changes but are not feasible for most people and may have adverse effects.
23. **Cautions from the textbook**: the reductionist single-marker approach fails because a living system is **homeodynamic**, with tolerance, compensation and adaptation — illustrated by failed beta-amyloid trials in Alzheimer's disease and by the unproven benefit of lengthening telomeres. The "hormonal fountain of youth", from Brown-Séquard's testicular extract onward, has been a consistent disappointment.

Summary Table (Kane Table 1-2)

Theory category	Mechanisms	Manifestations
Accumulation of damage to informational molecules	Spontaneous mutagenesis; failure of DNA repair; errors in DNA, RNA and protein synthesis; superoxide radicals and loss of scavenging enzymes	Copying errors; error catastrophe; oxidative cellular damage
Regulation of specific genes	Appearance of specific protein(s)	Genetically programmed senescence
Depletion of stem cells	Convergence of the above mechanisms	Proliferative potential decreases

Latest Updates (2023–Present)

- **"Hallmarks of Ageing: An Expanding Universe" (López-Otín, Blasco, Partridge, Serrano, Kroemer, *Cell*, January 2023)** expanded the original nine hallmarks to **twelve**, adding **disabled macroautophagy, chronic inflammation and dysbiosis**. The three defining criteria remain: the hallmark must manifest with age, accentuating it must accelerate ageing, and targeting it must decelerate, stop or reverse ageing.
- The twelve are grouped as **primary** (genomic instability, telomere attrition, epigenetic alterations, loss of proteostasis, disabled macroautophagy), **antagonistic** (deregulated nutrient sensing, mitochondrial dysfunction, cellular senescence) and **integrative** (stem cell exhaustion, altered intercellular communication, chronic inflammation, dysbiosis).
- The **2022 Copenhagen ageing meeting** proposed additional candidates — **altered mechanical properties and splicing dysregulation** — giving up to fourteen hallmarks in some frameworks.
- **Geroscience hypothesis**: ageing is the common upstream driver of the major chronic diseases, so targeting ageing biology should compress morbidity across many diseases simultaneously — the rationale for trials such as **TAME (Targeting Aging with Metformin)**.
- **Epigenetic clocks** (Horvath, Hannum, PhenoAge, GrimAge, DunedinPACE) are the most developed biological age measures, but the textbook's caution holds: a study cited in Pathy found the **frailty index outperformed DNA methylation age** as an indicator of biological age, and frailty is associated with the epigenetic clock but **not with telomere length**.
- **Senolytics** (dasatinib plus quercetin, fisetin) and **senomorphics** are in early human trials; newer approaches include **senolytic CAR-T cells**, **SGLT2 inhibitors such as canagliflozin** (senescent cell clearance via AMPK activation) and **anti-PD-1 antibodies** that clear PD-L1-positive senescent cells.
- **Partial cellular reprogramming** with Yamanaka factors reverses age-associated features in animal models, and **mTOR inhibition** (rapalogs) improves immune function in older humans — the two most-cited proofs of principle that ageing is modifiable.
- Nothing has yet translated into clinical geriatrics: the **only interventions with proven benefit for healthspan remain physical activity, adequate protein and micronutrient intake, avoidance of smoking and excess alcohol, management of vascular risk, sleep, and social engagement** — framed globally by the **WHO Decade of Healthy Ageing 2021–2030** and operationalised through **ICOPE**.

Current Practice Update

Issue	Textbook (Pathy 6e / Kane)	Current Evidence (2023–present)
Framework	Three categories (damage, gene regulation, stem cell depletion); nine ageing mechanisms	**Twelve hallmarks (López-Otín 2023)**, adding disabled macroautophagy, chronic inflammation and dysbiosis
Inflammation	Discussed under network/remodelling theory	Now a **formal hallmark** in its own right ("inflammaging")
Microbiome	Not included	**Dysbiosis** recognised as a hallmark of ageing
Biological age measurement	Telomere length; noted to be unreliable	**Epigenetic clocks** dominate research; still **not clinically validated**, and the frailty index remains a better practical marker
Interventions	Caloric restriction, scepticism about hormones	**Senolytics, senomorphics, mTOR inhibitors, metformin (TAME), partial reprogramming** — all investigational
Clinical translation	Warns against reductionism and false promise	Position unchanged — **lifestyle remains the only proven healthspan intervention**

Exam One-Liners

- Heritability of lifespan = **≤35%**; environment = **>65%**.
- Three theory categories = **damage to informational molecules, gene regulation, stem cell depletion**.
- **Hayflick limit** = about **50 doublings** for human fibroblasts; **50–100 base pairs** of telomere lost per replication.
- Hayflick's own view: ageing is **loss of molecular fidelity / thermodynamic instability**, not genes.
- Vitamins C and E do **not** extend lifespan; **caloric restriction** does in rodents (**30–50%**).
- Original hallmarks = **9 (2013)**; current = **12 (2023)**, adding **disabled macroautophagy, chronic inflammation, dysbiosis**.
- **Hormesis** = low-dose stress is beneficial; the theoretical basis of exercise and caloric restriction.
- **Frailty index outperforms DNA methylation age** as an indicator of biological age.

Q7. Immunology and Ageing — Innate and Adaptive Immune Systems; Immunosenescence and Cancer

Note on sources: Pathy's 6th edition does not carry a single dedicated chapter on the ageing immune system. The textbook content below is assembled from the chapters where it is covered — Cancer and Ageing, Geriatric Dermatology, Modern Use of Biomarkers in Geriatric Medicine, Centenarians, and Kane's chapter on the clinical implications of the ageing process.

15–20 High-Yield Points (Textbook)

A. General Concept

1. The immune system is one of the most important **master regulatory systems** in humans; its relationship with ageing has been re-evaluated away from a purely senescent, declining system to one that undergoes **remodelling**, with both **up- and downregulation** of immune functions.
2. **Both the innate and the adaptive immune systems become less effective with age**, leading to increased risk of infection and of malignancy.
3. **Healthy ageing** is believed to result from low pro-inflammatory activity combined with an efficient network capable of neutralising antigenic insults — that is, from successful **immune remodelling** rather than preserved youthful immunity.

B. Changes in the Adaptive Immune System

4. **Thymic involution** occurs after puberty; the **bone marrow diminishes in mass**, and levels of thymic hormones (**thymosins**) fall.
5. **T cells**: **reduced proliferation**, an **increased proportion of memory cells** and a **decrease in naive T cells**; T cells are also **less able to differentiate into specific subtypes**.
6. Lymphocytes from older adults show a **diminished proliferative response to mitogens**, due both to reduced lymphokine production and to reduced response to extracellular signals.
7. **Interleukin-2**: basal and stimulated production and responsiveness both fall, at least partly because of **decreased IL-2 receptor expression**. Conversely, **IL-6 levels rise**.
8. The T-cell defect appears to lie in the **transduction of extracellular signals to intracellular function**: the proliferative defect can be reversed in vitro by **calcium ionophores and activators of protein kinase C**, and some functions are restored by adding thymic hormones in vitro or administering them to aged animals.
9. **B cells**: production of B-cell precursors diminishes, and the ability to **isotype-switch** is altered, reducing the variety of antibodies and the diversity of functional B cells. **B-cell function is intact but dysregulated**, with increased **autoantibody formation** and increased **monoclonal protein production** (the basis of the high prevalence of MGUS in old age).
10. The **immune risk phenotype (IRP)**, defined in the Swedish NONA longitudinal study by Wikby, is a **CD4/CD8 ratio of less than 1**; it is associated with raised **IL-6** in the terminal decline stage and predicts mortality. The CD4/CD8 ratio is listed among the candidate immune biomarkers of frailty.

C. Changes in the Innate Immune System

11. **Neutrophils** show **decreased phagocytic ability**.
12. **Antigen-presenting cells**: there is a **reduction in Langerhans cells** in ageing skin, impairing cutaneous immune surveillance.
13. **Natural killer cells**: NK function is a clinically relevant variable — a prospective cohort study showed that individuals with **better NK cell function had lower rates of cancer ten years later**.
14. Impaired **innate, humoral and cell-mediated immunity within the skin** explains the increased risk of **herpes zoster** and **cutaneous candidiasis** in older people, and the delayed wound healing that follows reduced growth factor sensitivity of aged fibroblasts.

D. Inflammaging

15. **Chronic low-grade inflammation ("inflammaging")** is one of the major hallmarks of individual ageing. **Immunosenescence and inflammaging are described as two sides of the same coin**.
16. The three inflammatory markers most cited as biomarkers of frailty are **TNF-α (and its receptor TNFR2), IL-6 and CRP**; all three predict increased mortality among frail older people, and **pre-operative high IL-6** is a risk factor for post-operative delirium.
17. Franceschi's **network/remodelling theory** and the **hormesis** concept explain why some centenarians tolerate a high inflammatory load: they possess extraordinary **adaptive capacity and functional reserve**. Centenarians are classified as **"survivors", "delayers" and "escapers"**.

E. Immunosenescence and Cancer

18. **Immune dysregulation associated with ageing may contribute to the increased incidence of cancer with age** — reduced T-cell proliferation, fewer naive T cells, altered B-cell regulation, falling IL-2 and rising IL-6, and reduced NK function all impair **immune surveillance**.
19. Ageing and carcinogenesis **share molecular pathways**: DNA damage from random events or free radicals causes either cellular dysfunction and death (ageing) or mutations in proto-oncogenes and tumour suppressors (carcinogenesis). **DNA adduct formation, DNA hypomethylation, and chromosomal breakage and translocation** all increase with age and raise susceptibility to late-stage carcinogens.

20. Yet ageing and cancer are also **at odds**: cancer requires **limitless replicative potential**, whereas **finite replicative potential (replicative senescence) is a hallmark of ageing**. Normal cells manage 60–70 doublings and lose telomeres; malignant cells maintain telomeres through **telomerase** and replicate indefinitely.
21. **Stress-induced premature senescence** — triggered by oncogene mutation or by double-stranded DNA breaks from chemotherapy — drives clonal cells into permanent growth arrest and may be **as effective as apoptosis as an anticancer mechanism**.
22. Clinically, the probability of developing cancer over the age of 70 is **one in three in men and one in four in women**; cancer-specific death rates have fallen in the young while **rising in older individuals**, partly because of delayed diagnosis, incomplete evaluation, undertreatment and **under-representation of older people in clinical trials**.

Latest Updates (2023–Present)

- **Vaccination has become the main clinical application of immunosenescence.** Because standard vaccines respond poorly in older adults, **enhanced formulations** are now preferentially recommended for people aged 65 and above: **high-dose or adjuvanted (MF59) influenza vaccine**, and the **recombinant adjuvanted zoster vaccine (RZV, Shingrix)**, which retains over 90% efficacy in the very old, unlike the older live zoster vaccine.
- **RSV vaccines for older adults** (Arexvy, Abrysvo and the mRNA vaccine mResvia) were licensed from 2023; US ACIP guidance now recommends a single dose for **all adults aged 75 and above** and for adults **50–74 with risk factors** such as COPD, heart failure, diabetes, chronic kidney disease and immunocompromise.
- **Cytomegalovirus** is now recognised as a major driver of immunosenescence: chronic antigenic stimulation causes massive expansion of CMV-specific CD8 T cells with high **PD-1 and TIGIT** expression and reduced cytotoxicity, contributing to the immune risk phenotype and to **poorer vaccine antibody responses** in CMV-seropositive elders.
- The mechanistic picture has been refined to include **reduced dendritic cell phagocytosis, impaired T-cell receptor signalling, accumulation of terminally differentiated CD28-negative T cells, and accumulation of myeloid-derived suppressor cells**, forming a self-reinforcing **immunosenescence–inflammaging loop**.
- **Interventions being tested to rejuvenate immunity**: **mTOR inhibitors** (Mannick's trials showed improved influenza vaccine responses in older adults), **senolytics** (dasatinib plus quercetin, fisetin, senolytic CAR-T cells), **SGLT2 inhibitors** such as canagliflozin, and **anti-PD-1 antibodies** that clear PD-L1-positive senescent cells.
- **Cancer immunotherapy in older adults**: overall efficacy of immune checkpoint inhibitors appears broadly preserved in older patients in pooled analyses, but immunosenescence, inflammaging and cancer cachexia modify response, and older patients remain under-represented in trials. Geriatric assessment (SIOG/ASCO) before immunotherapy is recommended rather than age-based exclusion.
- **Dysbiosis** was added as a formal hallmark of ageing in 2023, linking the gut microbiome to inflammaging and to vaccine responsiveness — a plausible future intervention target.
- **Indian context**: adult immunisation uptake in older Indians remains very low. Association of Physicians of India and Geriatric Society of India recommendations cover annual **influenza**, **pneumococcal (PCV followed by PPSV23)**, **recombinant zoster**, **Tdap**, and COVID-19 boosters for those aged 60 and above; cost is the principal barrier (RZV and RSV vaccines are largely self-financed and not covered by the national immunisation programme). Practical alternatives with proven benefit remain **correction of undernutrition and vitamin D deficiency, control of diabetes, smoking cessation and physical activity**, all of which improve immune competence.

Current Practice Update

Issue	Textbook (Pathy 6e / Kane)	Current Evidence (2023–present)
Core concept	Age-related decline in immune function, with a note that remodelling is a better description	**Immune remodelling** is the accepted model — simultaneous loss of adaptive competence and gain of inflammatory tone
Driver of T-cell change	Thymic involution and reduced IL-2 signalling	Thymic involution plus **chronic CMV-driven antigenic stimulation** and accumulation of **CD28-negative, PD-1/TIGIT-high exhausted T cells**
Vaccination	Not addressed in detail	**Enhanced vaccines mandatory in the old**: high-dose/adjuvanted influenza, recombinant zoster, and new **RSV vaccines for 75+ and at-risk 50–74**
Inflammaging	Described within network/remodelling theory	A **formal hallmark of ageing (2023)**, alongside **dysbiosis**
Cancer immunity	Reduced surveillance; NK function predicts cancer risk	Same principles, plus recognition that **immunosenescence modifies checkpoint inhibitor response**; geriatric assessment rather than age-based exclusion
Reversibility	Some functions restorable with thymic hormones in vitro	**mTOR inhibitors and senolytics** show partial immune rejuvenation in early human trials

> **Exam One-Liners**

- Adaptive changes: **↓ naive T cells, ↑ memory T cells, ↓ proliferation, ↓ IL-2, ↓ isotype switching, ↑ autoantibodies, ↑ monoclonal proteins**.
- Innate changes: **↓ neutrophil phagocytosis, ↓ Langerhans cells, altered NK function, ↑ IL-6**.
- **Immune risk phenotype = CD4/CD8 ratio <1** (Wikby, NONA study) — predicts mortality.
- Immunosenescence and inflammaging = **"two sides of the same coin"**.
- Better **NK cell function** predicts **lower cancer incidence ten years later**.
- Cancer needs **limitless replication**; ageing shows **finite replication** — telomerase is the difference.
- Clinical translation = **high-dose/adjuvanted influenza, recombinant zoster and RSV vaccines** in the old.

Q8. Immunosenescence

This overlaps with Question 7; the answer below is written as a focused, self-contained account of the entity itself — definition, mechanisms, measurement, consequences and interventions.

15–20 High-Yield Points (Textbook)

1. **Immunosenescence** is the age-associated remodelling and functional deterioration of the immune system, affecting **both innate and adaptive arms**, and resulting in reduced protection against infection and malignancy with simultaneous **increased autoreactivity**.
2. It is best understood not as simple decline but as **remodelling** — some functions are downregulated and others upregulated — and it is listed among the mechanisms of ageing alongside cellular senescence and inflammaging.
3. **Structural basis**: **thymic involution** after puberty with falling **thymosin** levels, and reduction in **bone marrow mass**, which together restrict output of new naive lymphocytes.
4. **T-cell changes**: fall in **naive T cells**, rise in the proportion of **memory T cells**, impaired differentiation into specific subtypes, and reduced proliferative response to mitogens.
5. **Signalling defect**: reduced **IL-2** production and responsiveness with decreased IL-2 receptor expression; the defect lies in **signal transduction**, since it is reversible in vitro by calcium ionophores and protein kinase C activators.
6. **B-cell changes**: reduced B-cell precursor production, altered **isotype switching**, reduced antibody variety and B-cell diversity; function is preserved but **dysregulated**, with more **autoantibodies** and more **monoclonal protein**.
7. **Innate changes**: reduced **neutrophil phagocytosis**, fewer **Langerhans cells**, and variable NK cell function.
8. **Cytokine shift**: **IL-2 falls while IL-6 rises** — the biochemical signature of the shift from adaptive competence to inflammatory tone.
9. **Inflammaging** is the inseparable companion of immunosenescence — the two are described as **"two sides of the same coin"**, with chronic inflammation a major hallmark of individual ageing.
10. **Immune risk phenotype (IRP)**: defined by a **CD4/CD8 ratio below 1**, associated with raised IL-6 in the terminal decline stage and predictive of mortality in the Swedish NONA longitudinal study.
11. The **CD4/CD8 ratio** is the immune-system biomarker listed for frailty in Pathy's biomarker chapter; inflammatory markers (**TNF-α, IL-6, CRP**) are listed for both frailty and intrinsic capacity.
12. **Clinical consequences — infection**: increased incidence and severity of pneumonia, urinary tract infection, tuberculosis reactivation, **herpes zoster** and cutaneous candidiasis; **atypical presentation** with blunted fever and leucocyte response.
13. **Clinical consequences — vaccination**: reduced antibody titres, faster waning of protection and lower vaccine effectiveness in older adults.
14. **Clinical consequences — malignancy**: impaired immune surveillance contributes to the rise in cancer incidence with age; better **NK cell function predicts lower cancer rates ten years later**.
15. **Clinical consequences — autoimmunity and paraproteins**: rising autoantibody titres and monoclonal gammopathy of undetermined significance.
16. **Clinical consequences — delayed wound healing and impaired skin barrier**, with increased risk of skin infection and cutaneous malignancy compounded by ultraviolet exposure.
17. **Link to frailty**: frailty, inflammation and immunosenescence are explicitly grouped together; the frail phenotype is associated with raised inflammatory and immune markers in the Singapore Longitudinal Ageing Studies and elsewhere.
18. **Successful ageing**: centenarians appear to combine **low pro-inflammatory activity** with an efficient neutralising network, and possess exceptional adaptive capacity and functional reserve.

Summary of Changes with Ageing

Component	Change with ageing	Clinical consequence
Thymus / marrow	Involution; fall in thymosins; reduced marrow mass	Reduced output of naive lymphocytes
Naive T cells	Decreased	Poor response to **new** antigens and new vaccines
Memory T cells	Increased proportion; terminally differentiated	Narrowed repertoire; reactivation of latent infection (zoster, TB)
IL-2	Reduced production and receptor expression	Reduced clonal expansion; poor delayed hypersensitivity
B cells	Fewer precursors; impaired isotype switching	Lower-affinity antibody; poorer vaccine response; **MGUS**, autoantibodies
Neutrophils	Reduced phagocytosis	Bacterial infection risk
Langerhans cells	Reduced in skin	Skin infection and cutaneous malignancy
NK cells	Function variable, often impaired	Reduced tumour surveillance
IL-6 / TNF-α / CRP	Chronically raised (**inflammaging**)	Frailty, sarcopenia, anaemia, atherosclerosis, delirium risk, mortality
CD4/CD8 ratio	Falls below 1 in a subset (**IRP**)	Predicts mortality

Latest Updates (2023–Present)

- **Chronic inflammation** and **dysbiosis** were formally added to the hallmarks of ageing in the 2023 *Cell* update, placing inflammaging and the gut microbiome at the centre of immune ageing.
- **Cytomegalovirus** is now viewed as a principal accelerant: persistent antigenic stimulation drives massive expansion of CMV-specific CD8 T cells expressing **PD-1 and TIGIT** with reduced cytotoxicity, raises intracellular TNF-α in B cells, reduces switched memory B cells, and correlates with **poorer vaccine antibody responses** in seropositive older adults.
- The current mechanistic model adds **loss of CD28 expression on T cells**, **impaired T-cell receptor signalling**, **reduced dendritic cell phagocytic activity**, and **accumulation of myeloid-derived suppressor cells**, creating a self-amplifying **immunosenescence–inflammaging feedback loop** (2024–2025 reviews).
- **Vaccine strategy has been redesigned around immunosenescence**: adjuvanted and high-dose formulations, higher antigen content, recombinant platforms and mRNA platforms — exemplified by **high-dose and MF59-adjuvanted influenza vaccines**, the **AS01B-adjuvanted recombinant zoster vaccine**, and the **adjuvanted and mRNA RSV vaccines** licensed from 2023 for older adults.
- **Pharmacological reversal is being tested**: **mTOR inhibitors** improved influenza vaccine responses in older adults (Mannick trials); **senolytic therapy** (dasatinib plus quercetin, fisetin, senolytic CAR-T cells), **canagliflozin** via AMPK activation, and **anti-PD-1 antibodies** clearing PD-L1-positive senescent cells all reduce senescent cell burden and, in principle, restore vaccine immunogenicity.
- **Practical, presently available measures** that improve immune competence in older people remain **correction of protein-energy undernutrition, correction of vitamin D and zinc deficiency, glycaemic control, regular physical activity, adequate sleep, smoking cessation and age-appropriate vaccination**.
- **Indian relevance**: high background prevalence of latent tuberculosis, near-universal CMV seropositivity, high burden of diabetes and undernutrition, and very low adult vaccination coverage make immunosenescence clinically more consequential; the affordable, high-impact interventions are **influenza and pneumococcal vaccination, nutrition and diabetes control** rather than the newer costly vaccines.

Current Practice Update

Issue	Textbook (Pathy 6e / Kane)	Current Evidence (2023–present)
Terminology	Immunosenescence as decline, with a note that remodelling is more accurate	**Immune remodelling / immunobiography** — the individual's lifetime antigen exposure determines the phenotype
Main external driver	Not emphasised	**Chronic CMV infection** is a principal driver of the immune risk phenotype
Inflammaging	Discussed as an association	**Formal hallmark of ageing**; bidirectional loop with immunosenescence
Microbiome	Not covered	**Dysbiosis** recognised as a hallmark and a modifier of vaccine response
Clinical response	No specific recommendations	**Enhanced vaccine platforms** (high-dose, adjuvanted, recombinant, mRNA) are now standard for older adults
Reversal	Thymic hormones restore some function in vitro	**mTOR inhibitors and senolytics** in early human trials; not yet clinical practice

Exam One-Liners

- Immunosenescence = **age-related remodelling** of innate and adaptive immunity, not pure decline.
- Hallmark cellular change = **↓ naive T cells, ↑ memory T cells** from **thymic involution**.
- Cytokine signature = **IL-2 down, IL-6 up**.
- **Immune risk phenotype = CD4/CD8 <1** — predicts mortality.
- Immunosenescence + inflammaging = **"two sides of the same coin"**.
- B cells: **impaired isotype switching** → poor vaccine response, **↑ autoantibodies and MGUS**.
- Commonest clinical manifestations = **herpes zoster, pneumonia, TB reactivation, poor vaccine response, ↑ cancer**.
- Practical answer to immunosenescence today = **enhanced vaccines + nutrition + vitamin D + exercise + glycaemic control**.

Cross-Question Linking Points (useful for any of these eight answers)

- **One inflammatory axis runs through the whole set**: TNF-α and IL-6 explain sarcopenia, frailty, anaemia, anorexia of ageing, delirium risk and immunosenescence. Naming this axis links five answers together.
- **Sarcopenia is the biological substrate of physical frailty**; **deconditioning is acute sarcopenia**; **immunosenescence and inflammaging are the immunological expression of the same hallmarks of ageing**.
- The single intervention that appears in every answer is **progressive resistance exercise with adequate protein** — it improves frailty, sarcopenia, deconditioning and immune competence.
- For any Indian answer, quote **LASI** for epidemiology, **NPHCE** and the **Ayushman Vay Vandana Card** for service delivery, and **AWGS 2019** cut-offs rather than European cut-offs for muscle measurements.

Falls, Gait, Syncope & Dizziness

MD Geriatric Medicine — High-Yield 15-Mark Answers

Questions 1–14

Primary source: Pathy's Principles and Practice of Geriatric Medicine, 6th edition
(Ch. 75 Disorders of the Vestibular System; Ch. 78 Gait, Balance and Falls; Ch. 79 Hip Fracture and Orthogeriatrics)
Supplementary textbook: Kane's Essentials of Clinical Geriatrics (Ch. 9 Falls; Ch. 10 Immobility)

Latest Updates sections drawn from guidelines and peer-reviewed evidence, 2023–present
Indian epidemiology, units and programme context integrated throughout

Contents

1. Approach to recurrent falls in the elderly — prevention and management
2. 60-year-old diabetic, hypertensive, CAD and depression on multiple drugs with recurrent falls over one week — evaluate and manage
3. Intrinsic risk factors for falls
4. Post-fall syndrome
5. Long lie
6. Balance and gait assessment
7. Phases in human walking (gait cycle) with diagrams
8. Postural hypotension — evaluation and management
9. Syncope
10. Hypersensitive carotid sinus syndrome
11. Benign paroxysmal positional vertigo — diagnosis and management
12. Approach to a patient with dizziness
13. Evaluation of dizziness / drop attacks
14. Vestibular rehabilitation

> **How to use this set.** Every answer is written to the same shape: numbered high-yield points under labelled subheadings, then **Latest Updates (2023–present)**, then a **Current Practice Update** table wherever textbook and current evidence differ, then **Exam One-Liners**. Textbook content and post-textbook evidence are kept in separate blocks. Where a topic is not covered by name in the uploaded textbooks, this is stated openly at the start of that answer.

Q1. Approach to recurrent falls in the elderly, with emphasis on prevention and management

15–20 High-Yield Points (Textbook)

A. Definition and Burden

1. A **fall** is defined as an **"unexpected event in which the patient comes to rest on the ground, floor, or lower level"**. **Recurrent falls** means two or more falls in twelve months.
2. About **one-third of community-dwelling adults aged 65 and above fall every year**. Among **nursing home residents, one in two falls each year**.
3. **One in five falls causes serious injury** such as a **fracture or head trauma**. In the United States falls cause 3 million emergency visits and 800,000 hospitalisations yearly.
4. Common injuries are fractures of the **hip, femur, humerus, wrist and ribs**, soft-tissue injury, and **subdural haematoma**. Neurological signs appearing days to weeks after a fall should raise suspicion of subdural haematoma.
5. Even without injury, falls cause disability through **fear of falling, loss of self-confidence and restricted walking**, and are an important reason for **institutionalisation**.

B. Why Older People Fall

6. Falls are a **geriatric syndrome**. There is usually an **acute trigger acting on multiple chronic impairments** that have destroyed the person's ability to compensate. **A fall is rarely due to a single factor.**
7. Causes are divided into **intrinsic** (ageing neuromuscular physiology, disease) and **extrinsic** (medications, environment) factors. Risk rises **exponentially as factors accumulate**.
8. The mnemonic **AGAIN I'VE FALLEN** is used to check risk factors systematically: **A**gain fallen, **G**ait and balance problems, **A**DL loss, **I**mpaired mental status, **N**umber and type of drugs, **I**llness, **V**estibular disorders, **E**yes/Ears, **F**oot disorders, **A**lcohol, **L**ower extremity weakness, **L**ow blood pressure, **E**nvironmental hazards, **N**eurological disorders.
9. **Medication is the commonest and the most modifiable risk factor.** High-risk classes are **benzodiazepines, non-benzodiazepine hypnotics, antipsychotics, all antidepressants (TCA, SSRI, SNRI, trazodone, bupropion), antihistamines and anticholinergics, opioids and NSAIDs, muscle relaxants, alpha blockers, digoxin, amiodarone, antihypertensives and diuretics, insulin, Parkinson drugs and cholinesterase inhibitors**.
10. Risk is **highest in the days to weeks immediately after a drug is started or its dose is changed**. New drugs should ideally be started during periods of increased supervision.
11. In hospital, **delirium is the leading cause of falls**. In the community, environmental triggers are wet floors, poor lighting, uneven surfaces, rugs, steps and unstable furniture.

C. Evaluation

12. The **first step in history is to decide whether the event was a fall, a syncope or a seizure**, since management differs completely.
13. The CDC **STEADI algorithm** (Stopping Elderly Accidents, Deaths & Injuries) is the recommended step-wise approach. Ask every older adult about falls in the past year, or screen with the **Stay Independent 12-item questionnaire** or the **three key questions**.
14. Those who screen positive get a risk assessment using three performance tests: **Timed Up and Go Test (TUGT)**, **30-second chair stand** and **four-stage balance test**.
15. **There is no routine need for laboratory or imaging tests** in a fall assessment. **MRI brain** only if a treatable neurological cause is suspected (infarcts, demyelination, posterior fossa malformation, cerebellar degeneration, hydrocephalus). **EEG is rarely helpful** and only if seizure is strongly suspected.
16. The **minimum investigations in a patient who falls are lying and standing blood pressure and a 12-lead ECG**. A medical cause must be actively sought: **postural hypotension, vasovagal syncope, carotid sinus hypersensitivity**.

D. Management and Prevention

17. **Principles of management**: (i) assess and treat physical injury, (ii) treat the underlying conditions, (iii) prevent future falls by physiotherapy and education — gait and balance retraining, muscle strengthening, ambulation aids, properly fitted shoes, adaptive behaviours, and (iv) alter the environment — safe furniture of correct height, removal of loose rugs and obstacles, proper lighting, rails on stairs and in the bathroom.
18. **Exercise is the single most effective intervention.** In the Cochrane review by Sherrington, **balance and functional exercise reduced the rate of falls by 24%** (RR 0.76), **multiple exercise types (balance plus resistance) by 34%** (RR 0.66), and **tai chi by 19%** (RR 0.81). Walking alone or resistance training alone without balance work has uncertain benefit.
19. **Vision**: routine screening alone did not help, but **assessment and treatment of vision impairment combined with exercise reduces falls**. **Cataract surgery reduces falls in older women** (second-eye surgery does not give the same benefit). Replacing multifocal with single-vision lenses for outdoor use helps.
20. **Home hazard assessment and modification reduces falls**, especially with severe visual impairment, and is **more effective when done by an occupational therapist** — grab bars, better lighting on stairs, removal of rugs, socket covers, cupboard locks.
21. **Reducing high-risk medication, particularly withdrawal of psychotropics, is validated as effective. Vitamin D** does not help all older adults but benefits those with **low baseline levels** and **nursing home residents**.
22. What does **not** work: **physical restraints** (they worsen delirium, pressure ulcers and aspiration), **chemical restraints**, **bed rails** and **bed alarms**. **Hip protectors** give a small reduction in hip fracture in care homes (RR 0.82) but **no clear benefit in the community**; adherence is the main barrier.
23. **Cardiac pacing** is an effective single-factor intervention in patients with **carotid sinus hypersensitivity**.

Latest Updates (2023–Present)

- **World Falls Guidelines 2022 (Age and Ageing)** is now the global reference and has replaced the AGS/BGS 2011 and NICE 2013 framework quoted in the textbook. It uses a **three-tier risk stratification — low, intermediate and high**. Low risk: no fall in the past year, or one fall without gait/balance impairment → education and exercise advice. Intermediate risk: one non-injurious fall with impaired gait and balance → tailored balance and strength exercise. **High risk**: **two or more falls in 12 months, or one fall with injury requiring medical treatment, frailty, a long lie of one hour or more, or suspected transient loss of consciousness** → comprehensive multifactorial assessment and personalised multidomain intervention.
- World Falls Guidelines make a **strong recommendation (GRADE 1B)** that multifactorial assessment must include a **cardiovascular assessment — cardiac history, auscultation, lying and standing blood pressure, and a surface 12-lead ECG**. If this is normal, **no further cardiovascular testing is needed unless syncope is suspected** (witnessed syncope/pre-syncope or recurrent unexplained falls) (GRADE 1C).
- The guidelines formally add **concerns about falling (fear of falling)** to the multifactorial assessment, and emphasise **e-health, opportunistic case finding** and **applicability to low- and middle-income countries** — directly relevant to Indian practice.
- **USPSTF 2024 recommendation statement (JAMA)** for community-dwelling adults 65 years and above at increased risk of falls: **exercise interventions — Grade B** (moderate net benefit); **multifactorial interventions — Grade C**, decision to be individualised because net benefit is small. Vitamin D was removed from the scope of this statement.
- **Indian data (LASI wave-1)**: self-reported falls in **11.6–12.5% of adults aged 60 and above**, higher in women and rising with age; **injurious falls in 5.7%** and **dizziness in 14.5%**. Determinants include female sex, underweight BMI, tobacco and alcohol use, chronic joint disease, prior fracture and sensory impairment. **Multimorbidity independently raises fall risk (AOR 1.29)**. **Frailty prevalence is about 30%** and frail elders have higher falls (15.4% vs 11.9%) and multiple falls (7.7% vs 5.3%).
- **Indian service context**: falls prevention is delivered through **NPHCE** geriatric clinics and district geriatric units; assistive and mobility devices through **Rashtriya Vayoshri Yojana** and the **ADIP scheme (ALIMCO)**; hospitalisation for fall-related fracture covered under **AB-PMJAY** and the **Ayushman Vay Vandana Card** for those aged 70 and above; **Elder Line 14567** for elders living alone.

Current Practice Update

Issue	Textbook position (Pathy 6e / Kane)	Current position (2023-present)
Guideline framework	AGS/BGS 2011 and NICE 2013; CDC STEADI screen-assess-intervene	**World Falls Guidelines 2022** three-tier low/intermediate/high risk stratification is the global standard; STEADI retained as a screening tool
Cardiovascular workup	Minimum — lying/standing BP and 12-lead ECG	Same core, now a **strong graded recommendation (1B)**, with an explicit **stopping rule**: no further cardiac testing if initial assessment normal and no suspected syncope
Multifactorial intervention	Recommended broadly for all fallers	**USPSTF 2024 downgraded to Grade C** — individualise; **exercise remains Grade B** and is the strongest single component
Fear of falling	Described as a risk factor and consequence	Now a **formal mandatory domain** of multifactorial assessment (World Falls Guidelines 2022)
Long lie	Not named as a risk category	**Inability to rise for ≥1 hour is an explicit high-risk criterion**
Vitamin D	Kane advises at least 800 IU/day for all	Targeted only — deficiency and care-home residents; **removed from USPSTF falls statement**; high intermittent bolus doses avoided

Exam One-Liners

- Fall = unexpected event coming to rest on ground, floor or lower level. Recurrent = ≥2 falls in 12 months.
- One-third of community elders and one-half of nursing home residents fall each year; 1 in 5 falls injures.
- India (LASI): falls 11.6–12.5%; injurious falls 5.7%; frailty ~30%.
- First question in history: fall, syncope or seizure?
- Minimum tests: lying/standing BP + 12-lead ECG. No routine bloods or imaging.
- Cochrane: balance+functional exercise ↓24%, multiple exercise types ↓34%, tai chi ↓19%.
- High risk (WFG 2022): ≥2 falls, injurious fall, frailty, long lie ≥1 h, or TLoC.
- Restraints, bed rails and bed alarms do NOT prevent falls.

Q2. A 60-year-old diabetic, hypertensive, with CAD and depression on multiple drugs, presents with recurrent falls over one week — evaluate and manage

> **Key insight for the examiner:** falls clustering over **one week** in a previously stable patient indicate an **acute precipitant superimposed on chronic risk factors** — this is a "**premonitory fall**" pattern (Kane) and demands a search for acute illness, delirium or a recent drug change, not merely a routine falls assessment.

15–20 High-Yield Points (Textbook)

A. Framing the Problem

1. This patient has **four independent fall risk domains**: **diabetes** (neuropathy, retinopathy, hypoglycaemia, autonomic dysfunction), **hypertension** (drug-induced hypotension; orthostatic hypotension is commonest in uncontrolled hypertension), **CAD** (arrhythmia, low output, nitrates), and **depression** (antidepressants, poor concentration, deconditioning).
2. **Polypharmacy is the dominant modifiable factor.** More than four drugs, and use of psychotropic, analgesic, diuretic and antihypertensive drugs, are explicit fall risk factors.
3. **An abrupt cluster of falls over one week signals a new event**: acute illness ("premonitory fall"), delirium, a recent drug initiation or dose change, or a new cardiac or neurological event.

B. History

4. Take a **detailed history of each fall** — location and time of day, activity, whether alone or witnessed, and whether the patient was **aware of the impending fall**, tripped or slipped, or the fall was totally unexpected.
5. Ask about **relationship to change of posture, turning of the head, cough, urination, a meal, or medication intake** — each points to a different mechanism (orthostatic, carotid sinus, situational, postprandial hypotension, drug effect).
6. Ask for **premonitory and associated symptoms**: light-headedness, dizziness, vertigo, palpitations, chest pain, breathlessness, focal neurological symptoms (weakness, sensory disturbance, dysarthria, ataxia, aphasia), aura, incontinence of urine or stool.
7. Ask specifically about **loss of consciousness**, what is remembered immediately after the fall, **whether the patient could get up and how long it took**, and whether any witness can confirm loss of consciousness. Loss of consciousness suggests a **cardiac event or seizure**.
8. Obtain a **complete drug chart including doses, recent changes, over-the-counter drugs and alcohol**, and note any drug started or up-titrated in the preceding two weeks.

C. Examination

9. **Vital signs first.** Fever, tachypnoea, tachycardia or hypotension should prompt a search for **acute illness — pneumonia or sepsis, myocardial infarction, pulmonary embolism, gastrointestinal bleeding**.
10. **Postural blood pressure and pulse supine, sitting, and after 1 and 3 minutes of standing** are critical. A fall of **more than 20 mmHg systolic** is significant. Also check for **postprandial hypotension** if events occur within two hours of a meal.
11. If the history suggests carotid sinus sensitivity, **gently massage the carotid for 5 seconds** and observe for **profound bradycardia (50% reduction in heart rate) or a long pause**.
12. **Cardiovascular examination** for arrhythmia and **aortic stenosis**. Both are treatable, serious, and hard to detect clinically — refer for **continuous monitoring and echocardiography** if suspected. Syncope in aortic stenosis is an indication to evaluate for valve replacement.
13. **Neurological examination**: mental status, focal signs, muscle weakness, rigidity or spasticity, **peripheral neuropathy especially posterior column signs (position and vibration sense)** given diabetes, cerebellar signs (heel-shin, heel tapping), and features of Parkinson's disease.
14. **Visual acuity** for uncorrected refractive error, cataract and diabetic retinopathy. Examine the **feet** for deformity, painful lesions (calluses, bunions, ulcers), neuropathic changes and **poorly fitting or worn-out footwear**.
15. Examine **skin, extremities and painful soft-tissue areas for injury**, and specifically consider **subdural haematoma** if there is head injury or new neurological signs days to weeks later.
16. Perform **gait and balance assessment** — more useful for finding remediable problems than the standard neuromuscular examination. Use the **Get Up and Go test**, and the STEADI trio of **TUGT, 30-second chair stand and four-stage balance test**.

D. Investigations

17. Directed, not routine: **capillary and fasting glucose, HbA1c**; **haemoglobin g/dL** (occult GI bleed); **sodium and potassium mEq/L**; **blood urea and creatinine mg/dL**; **calcium mg/dL**; **vitamin D**; **TSH**; urine routine and culture; **12-lead ECG**. Continuous cardiac monitoring and echocardiography only if clinically indicated; the arrhythmia must be **temporally associated with symptoms** before it is blamed.

E. Management

18. **Treat the acute precipitant first** — infection, hypoglycaemia, dehydration, anaemia, electrolyte disturbance, delirium, ischaemia.
19. **Deprescribe and rationalise**: withdraw or reduce sedatives and hypnotics; review antidepressant choice and dose; reduce or space antihypertensives and diuretics if postural drop is present; review nitrates and alpha blockers; avoid sulfonylureas and reduce insulin to **relax glycaemic targets in a faller**; stop anticholinergics.
20. **Correct the specific causes found**: support stockings, leg elevation, slow staged rising and, if needed,

mineralocorticoid or midodrine for postural hypotension; podiatry and correct footwear; ophthalmology referral; pacemaker if cardio-inhibitory carotid sinus hypersensitivity; antiparkinsonian drugs if indicated.

21. **Rehabilitation and environment**: refer to physiotherapy for **gait and balance retraining and lower limb strengthening**, and to occupational therapy for **home hazard assessment**. Prescribe and train in the correct use of a walking aid. Assess osteoporosis and start calcium and vitamin D; consider bone-specific therapy.
22. **Ensure safety in this recurrent faller by urgent intervention**, review response at short interval, and educate patient and family about the heightened risk in the days after any drug change.

Latest Updates (2023–Present)

- By **World Falls Guidelines 2022** criteria this patient is **high risk** (≥2 falls in 12 months) and must receive a **comprehensive multifactorial falls risk assessment with co-designed, personalised multidomain intervention** — not a single-issue fix.
- The mandated **cardiovascular assessment (GRADE 1B)** — cardiac history, auscultation, lying and standing BP, 12-lead ECG — is particularly important here because of CAD; further cardiac investigation is justified only if syncope is suspected or falls remain unexplained.
- **Fall-Risk-Increasing Drugs (FRIDs)** is now the standard framework (EuGMS Task and Finish Group). The **STOPPFall tool** gives structured deprescribing steps for psychotropics and cardiovascular drugs, and is a better exam answer than "review medications". Note that the **OPTIMISE trial** showed antihypertensive deprescribing in older people is feasible but does not automatically improve outcomes — deprescribing must be individualised and monitored.
- **Glycaemic targets in a frail faller**: current ADA and IDF/ISPAD-aligned geriatric guidance supports relaxing to **HbA1c 7.5–8.0%** (and up to 8.5% in very frail or limited life expectancy), avoiding sulfonylureas and prandial insulin, and preferring **metformin, DPP-4 inhibitors or SGLT2/GLP-1 agents** where affordable and appropriate — because hypoglycaemia is a direct precipitant of falls.
- **Blood pressure targets**: the **2024 ESC hypertension guideline** pushes systolic to 120–129 mmHg but explicitly exempts patients with **symptomatic orthostatic hypotension, frailty and limited life expectancy**. **STRIDE (2020)** showed that a pragmatic multifactorial fall-injury intervention did not reduce serious fall injuries, reinforcing that **exercise plus targeted correction of specific deficits beats a generic checklist**.
- **India-specific**: use the **NPHCE district geriatric clinic** for follow-up, **AB-PMJAY / Ayushman Vay Vandana Card** for fracture care, **Rashtriya Vayoshri Yojana / ADIP (ALIMCO)** for free walking aids and spectacles, and note the high burden of **vitamin D deficiency and undiagnosed osteoporosis** in Indian elders.

Current Practice Update

Issue	Textbook position	Current position
Medication review	Withdraw psychotropics; list of offending classes	**FRIDs concept** with the structured **STOPPFall** deprescribing tool; monitored, individualised withdrawal
Diabetes control	Caution with hypoglycaemia-inducing agents	Explicit **relaxed targets (HbA1c 7.5–8.5%)** in frail fallers; avoid sulfonylureas and prandial insulin
BP target	Antihypertensives increase fall risk in new users	Intensive targets (SBP 120–129) recommended generally but **explicitly relaxed in symptomatic orthostatic hypotension and frailty**
Multifactorial package	Broadly recommended	STRIDE showed generic packages fail; **exercise plus targeted correction of identified deficits** is what works

Exam One-Liners

- Falls clustering within a week = look for an acute precipitant (premonitory fall).
- Check vitals, postural BP at 1 and 3 minutes, and a 12-lead ECG in every case.
- Loss of consciousness → suspect cardiac cause or seizure, not a simple fall.
- Carotid massage 5 s: positive if 50% fall in heart rate or a long pause.
- Deprescribing framework = FRIDs + STOPPFall.
- Relax HbA1c to 7.5–8.5% and avoid sulfonylureas in a frail faller.
- Refer to PT for gait/balance and OT for home hazards; assess osteoporosis.

Q3. Intrinsic risk factors for falls

15–20 High-Yield Points (Textbook)

A. Concept

1. Falls result from an interaction between **intrinsic factors** (arising within the patient) and **extrinsic factors** (medications, environment, assistive devices). **Rarely is a fall due to a single factor**, and risk rises **exponentially as intrinsic factors accumulate**.
2. **Intrinsic factors** comprise three groups: (i) **normal age-related physiological change**, (ii) **disease-related impairment**, and (iii) **psychological factors** such as fear of falling.

B. Age-Related Physiological Changes

3. **Diminished proprioception** from reduced conduction velocity in sensory and motor nerves. Peripheral sensation is the most critical afferent control of standing balance in healthy older people.
4. **Slower righting reflexes and slowed reaction time**, so the person cannot recover after a trip.
5. **Increased postural sway**, mainly anterior-posterior; both magnitude and velocity of sway increase with age.
6. **Loss of muscle mass (10–40%)** with preferential loss of **type II (fast twitch) fibres**, and prolonged contraction and relaxation time — the basis of **sarcopenia**.
7. **Stiffer joint capsules and ligaments** from cross-linking and loss of elastic fibres in articular cartilage.
8. **Gait changes**: gait speed falls **12–16% per decade** (up to 20% for maximum gait speed), with **reduced step length** and **increased time in double limb support**. Older men develop a flexed, wide-based, short-stepped gait; older women a narrow-based, waddling gait. Feet are not picked up as high, increasing tripping.
9. **Reduced baroreflex sensitivity** leading to **orthostatic hypotension** (about 20% of older persons) and **postprandial hypotension**.
10. **Impaired dual-tasking.** Institutionalised older adults who cannot hold a conversation while walking have a high risk of falls.

C. Disease-Related Intrinsic Factors

11. **Neurological**: stroke with residual deficit, **Parkinson's disease** (rigidity, freezing, slow gait), cerebellar disease, **peripheral neuropathy** (diabetes), cervical and lumbar spondylosis with cord or root compression, **normal-pressure hydrocephalus**, seizure disorder, subdural haematoma or tumour.
12. **Cognitive impairment and dementia.** Older adults with cognitive impairment fall at **two to eight times** the rate of cognitively intact peers, and recurrent falls may be the **first presentation of dementia**. Delirium is the strongest inpatient association.
13. **Cardiovascular**: arrhythmias, **aortic stenosis**, **carotid sinus hypersensitivity**, orthostatic and postprandial hypotension, and low cardiac output from heart failure or hypovolaemia.
14. **Musculoskeletal**: degenerative joint disease of neck, lumbosacral spine and lower limbs; healed hip and femur fractures causing an unsteady gait; **lower extremity weakness** and deconditioning from disuse.
15. **Sensory**: impaired visual acuity (cataract, glaucoma, retinopathy, poor near vision), **impaired hearing**, and **vestibular disorders**. Vision becomes more important as other inputs are lost, so visual loss is disproportionately disabling.
16. **Foot disorders**: bunions, calluses, nail disease and joint deformities are **common and correctable** causes of instability.
17. **Genitourinary**: overactive bladder, urge incontinence and **nocturia**.
18. **Chronic musculoskeletal pain** causes antalgic gait and acts as a cognitive distractor forcing dual-tasking; it is associated with falls **even after adjusting for analgesic use**.

D. Psychological

19. **Fear of falling (FoF)** is the lack of self-confidence that normal activities can be performed without falling. It is an **independent predictor of future falls**, and it acts by making the person restrict activity (leading to further deconditioning) and by consuming the cognitive resources needed for gait and balance control.
20. Also intrinsic in effect are **alcohol use**, **loss of ADL function**, **low baseline physical activity** and **previous falls** — a previous fall is the single strongest predictor of the next one.

Latest Updates (2023–Present)

- **World Falls Guidelines 2022** require that every multifactorial assessment covers, as a minimum, **gait and balance, muscle strength, orthostatic blood pressure and cardiovascular status, vision, cognition, continence, foot and footwear, nutrition and vitamin D, medication review, and concerns about falling**.
- **Concerns about falling** have been formally added as an assessment domain; a 2025 systematic review and meta-analysis applying Bradford Hill criteria confirmed that concerns about falling independently predict future falls, though the effect size is modest.
- **Sarcopenia** is now the preferred intrinsic construct for muscle-related fall risk, using **EWGSOP2 / AWGS 2019** criteria (low grip strength, low muscle mass, low gait speed), with **SARC-F** as the practical Indian screening tool.
- **Indian evidence (LASI wave-1)** identifies as significant intrinsic determinants: **female sex, advancing age, underweight BMI, chronic joint disease, previous injury or fracture, sensory impairment, depressive symptoms, poor grip strength and slow gait speed**. **Frailty (Fried phenotype, prevalence ~30%)** independently increases falls (aOR 1.24).
- **Orthostatic hypotension, cognitive impairment and reduced mobility** are now described as an interacting **"Bermuda triangle"** in older fallers, meaning these three should be looked for together rather than separately.

Current Practice Update

Issue	Textbook position	Current position
Muscle weakness	Described as age-related loss of type II fibres and deconditioning	Formalised as **sarcopenia** with operational criteria (EWGSOP2 / AWGS 2019, SARC-F screening)
Fear of falling	Recognised as a consequence and a predictor	**Mandatory assessment domain**; independent predictive value confirmed by 2025 meta-analysis
Frailty	Mentioned only in passing	**Independent high-risk criterion** in World Falls Guidelines 2022; ~30% prevalence in Indian elders

Exam One-Liners

- Intrinsic = age change + disease + psychological; Extrinsic = drugs + environment + devices.
- Peripheral sensation (proprioception) is the most critical afferent for standing balance.
- Gait speed declines 12–16% per decade; type II fibres lost preferentially.
- Orthostatic hypotension in ~20% of older persons.
- Cognitive impairment raises fall rate 2–8 fold; delirium is the main inpatient trigger.
- Previous fall is the strongest single predictor of a future fall.
- Fear of falling is itself an independent risk factor, not just a consequence.

Q4. Post-fall syndrome

> **Textbook coverage statement.** The term **"post-fall syndrome" is not used by name** in the uploaded chapters of Pathy's 6th edition or Kane's Essentials. However, **all of its components are described** — fear of falling, loss of self-confidence, restriction of ambulation (self-imposed or imposed by caregivers), disability and increased risk of institutionalisation and death. The definition and classical description below are therefore drawn from the wider geriatric literature (Murphy and Isaacs, 1982) and are clearly separated from textbook content.

15–20 High-Yield Points

A. Definition and Concept `supplementary`

1. **Post-fall syndrome** (also called **ptophobia**, or the post-fall anxiety syndrome) was described by **Murphy and Isaacs in 1982** as a **disabling psychomotor state occurring after a fall in an older person, characterised by fear of falling, loss of postural confidence and a stereotyped abnormal posture and gait, in the absence of any neurological lesion sufficient to explain it**.
2. It is a **geriatric emergency of function**: the disability is out of all proportion to the physical injury sustained, and it can convert an independent older person into a bed- or chair-bound one within days.
3. It typically follows a fall associated with a **long lie**, an injurious fall, or a fall witnessed by an anxious family.

B. Clinical Features `supplementary`

4. **Psychological component**: intense **fear of falling**, anxiety, loss of self-confidence, refusal to walk, and sometimes frank **depression**.
5. **Postural component (retropulsion)**: the trunk is **thrown backwards** when standing or transferring, with the centre of gravity displaced behind the base of support.
6. **Gait component**: **hesitancy in initiating gait**, short shuffling steps with the feet barely leaving the floor ("**marche à petits pas**"), a wide base, absent arm swing, and **clutching or grasping** at furniture and at the examiner.
7. **Failure of protective reactions**: the person does not put the arms out to save themselves and does not step to correct a perturbation.
8. It is sometimes described in three stages: **acute (immediate fear and refusal to stand)**, **sub-acute (retropulsion and clutching gait)** and **chronic (established immobility and dependency)**.

C. Consequences `textbook`

9. Kane records that **even when the fall does not cause serious injury, substantial disability may result from fear of falling, loss of self-confidence, and restricted ambulation — either self-imposed or imposed by caregivers**.
10. **Impaired mobility** leads to the full spectrum of **complications of immobility**: pressure ulcers, muscular deconditioning and atrophy, contractures, bone loss, cardiovascular deconditioning, **orthostatic hypotension**, venous thrombosis and embolism, reduced ventilation with atelectasis and aspiration pneumonia, constipation and faecal impaction, urinary retention and infection, negative nitrogen balance, and psychologically sensory deprivation, delirium and depression.
11. Reduced activity causes **further loss of musculoskeletal and neurological reserve**, so the risk of the next fall **increases** — a vicious cycle.
12. **Fear of falling reduces the cognitive resources available for gait and balance control** (impaired dual-tasking), which itself worsens gait.
13. End results are **loss of independence, caregiver burden, increased risk of institutionalisation and increased risk of death**.

D. Assessment

14. Recognise it clinically: a patient who **will not stand or walk after a fall despite adequate analgesia and no fracture** has post-fall syndrome until proved otherwise.
15. **Exclude organic causes** of the refusal — occult hip or pelvic fracture, **subdural haematoma** (neurological signs days to weeks after the fall), acute stroke, delirium, pain, and drug effect.
16. **Quantify fear of falling** with a scale: **Falls Efficacy Scale-International (FES-I)** or the short FES-I, or the Activities-specific Balance Confidence (ABC) scale. Quantify gait and balance with the **Tinetti POMA** and **TUG**.

E. Management

17. **Get the patient up early.** The single most important step is **early, supervised, graded mobilisation** — prolonged bed rest converts a reversible syndrome into permanent immobility.
18. **Physiotherapy**: progressive **balance and functional training, lower limb strengthening, transfer and gait retraining**, and specific practice of **backward disequilibrium correction** and protective stepping reactions.
19. **Teach the patient how to get up from the floor** (backward chaining) — this directly reduces the fear and prevents a future long lie.
20. **Cognitive-behavioural approaches and psychological support**, treatment of coexisting depression, and reassurance and education of the family, who are often the source of the restriction ("don't walk, you will fall again").
21. **Occupational therapy** for home hazard modification, grab bars and correct-height furniture; appropriate **walking aid** with training; well-fitted footwear.
22. **Provide a personal alarm or pendant** and arrange a check-in system for the elder living alone. Withdraw fall-risk-increasing drugs and treat the underlying cause of the index fall.

Latest Updates (2023–Present)

- **World Falls Guidelines 2022** make assessment of **"concerns about falling"** a formal component of the multifactorial falls risk assessment and recommend **FES-I / short FES-I** as the measurement instrument. The guidelines deliberately prefer the term "concerns about falling" over "fear of falling" because it is less stigmatising and better captures activity restriction.
- A **2025 systematic review and meta-analysis** (Age and Ageing) applying the Bradford Hill criteria confirmed that **baseline concerns about falling independently predict future falls**, supporting its inclusion as a treatment target and not merely an outcome.
- **Cognitive-behavioural therapy-based programmes** such as **"A Matter of Balance"** and CBT delivered by trained lay leaders reduce fear of falling and activity restriction; Cochrane evidence supports **exercise (especially tai chi) plus CBT** as the most effective combination.
- **Teaching floor-rise technique (backward chaining)** is now explicitly recommended by World Falls Guidelines and by the British Geriatrics Society for anyone at risk of a long lie, as both a physical and a psychological intervention.
- **Indian context**: activity restriction after a fall is reinforced by family protectiveness and by the cultural expectation that elders should "rest". Practical measures include family education under **NPHCE** counselling, provision of a walking aid through **Rashtriya Vayoshri Yojana**, home-based physiotherapy where available, and the **Elder Line 14567** for elders living alone.

Current Practice Update

Issue	Textbook position	Current position
Terminology	Term "post-fall syndrome" not used; components described as fear of falling and restricted ambulation	Recognised entity; preferred assessment term is **"concerns about falling"** (World Falls Guidelines 2022)
Measurement	No named scale	**FES-I / short FES-I** recommended as standard instrument
Treatment	Physical therapy, education, environmental change	**Exercise (tai chi) plus CBT**; explicit teaching of **floor-rise / backward chaining**

Exam One-Liners

- Post-fall syndrome = fear + loss of postural confidence + retropulsion + hesitant clutching gait, with no adequate neurological lesion.
- Described by Murphy and Isaacs, 1982; also called ptophobia.
- Disability is out of proportion to the physical injury.
- Measure with FES-I; treat with early mobilisation + physiotherapy + CBT.
- Teach floor-rise (backward chaining) — prevents both fear and future long lie.
- Prolonged bed rest converts a reversible syndrome into permanent immobility.

Q5. Long lie

> **Textbook coverage statement.** The term **"long lie" is not used by name** in the uploaded chapters of Pathy's 6th edition or Kane's Essentials. Kane does, however, make it a key history point — "**Could the patient get up, and if so, how long did it take?**" — and Pathy notes the older person's fear of "**being incapacitated at home, unable to reach help**". The definition and consequences below come from the wider geriatric literature and current guidelines and are clearly separated.

15–20 High-Yield Points

A. Definition `supplementary`

1. A **long lie** is defined as **remaining on the floor or ground for one hour or more after a fall, because the person is unable to get up unaided**.
2. It is a **marker of severity, not a diagnosis** — it indicates that the faller has neither the muscle strength nor the balance nor the help required to rise.
3. Roughly **half of older people who fall cannot get up without help**, and a long lie occurs in about **10–20% of falls** in community-dwelling elders. It is far commoner in those who **live alone**, have **dementia**, are **frail or sarcopenic**, or have **no personal alarm**.

B. Why It Matters

4. It is an **independent predictor of death**: classically about **half of those who lie for more than an hour die within six months**, even if the fall itself caused no serious injury.
5. It is also a strong predictor of **hospital admission and permanent institutionalisation**.

C. Complications

6. **Dehydration** and **hypovolaemia** from prolonged immobility without fluid intake.
7. **Rhabdomyolysis** from sustained pressure on muscle, causing raised creatine kinase, **myoglobinuria** and **acute kidney injury**; monitor **creatinine mg/dL** and **potassium mEq/L**.
8. **Hyperkalaemia, hyperphosphataemia, hypocalcaemia (calcium mg/dL)** and metabolic acidosis from muscle breakdown.
9. **Pressure injury** over the sacrum, heels, trochanters and elbows — pressure damage can begin within two hours of unrelieved loading.
10. **Hypothermia**, which in Indian practice is a real risk in northern winters and in poorly heated homes.
11. **Aspiration pneumonia and hypostatic pneumonia**, and **compartment syndrome** of a dependent limb.
12. **Delirium**, and worsening of any underlying acute illness that caused the fall.
13. **Post-fall syndrome** — the long lie is the classical trigger for profound fear of falling and activity restriction.

D. Management of the Patient Found After a Long Lie

14. Treat as a **medical emergency**. Assess airway, breathing, circulation and **core temperature**; rewarm gradually if hypothermic.
15. **Investigate**: haemoglobin g/dL, sodium and potassium mEq/L, urea and creatinine mg/dL, calcium mg/dL, **creatine kinase**, arterial blood gas, urine for myoglobin, blood glucose, ECG (hyperkalaemic changes), chest X-ray, and imaging of any painful area for fracture.
16. **Aggressive intravenous fluid resuscitation** to prevent or treat AKI from rhabdomyolysis; monitor urine output, potassium and renal function; treat hyperkalaemia.
17. **Full skin inspection** and immediate pressure injury care with redistribution surfaces; treat infection; provide analgesia and nutritional support.
18. **Find and treat the cause of the fall** — including a search for syncope, arrhythmia, stroke, sepsis, hypoglycaemia and drug effects.

E. Prevention

19. **Teach floor-rise technique (backward chaining)** to every at-risk older person and their carer — this is the single most useful preventive skill.
20. Provide a **personal alarm, pendant or wearable fall detector**, keep a **charged mobile phone within reach at floor level**, arrange **daily check-in calls or visits**, and consider a key-safe so help can enter.
21. **Strength and balance exercise** to maintain the ability to rise; treat sarcopenia; keep a low bed and clear floor space; avoid a locked bathroom door.

Latest Updates (2023–Present)

- **World Falls Guidelines 2022** make the long lie an explicit part of risk stratification: an adult who **"was laying on the floor unable to rise independently for at least one hour"** is to be regarded as at **high risk of future falls**, alongside injurious falls, recurrent falls, frailty and suspected transient loss of consciousness. This has moved the long lie from a descriptive observation to a formal **triage criterion**.
- The guidelines also recommend directly asking about the **ability to get up from the floor** as part of the fall history, and teaching floor-rise strategies.
- **Technology**: wearable and ambient fall-detection systems (accelerometer-based pendants, smartwatch fall detection, radar and depth-camera ambient sensors, smart-home floor sensors) are the focus of current research to shorten the time on the floor. Evidence to date shows they **shorten time-to-help but have not yet been shown to reduce mortality**, and false alarms and non-wearing remain limiting; guidelines therefore describe them as **promising adjuncts, not**

replacements for exercise and floor-rise training.

- **Ambulance and emergency service pathways** increasingly include a “fallen and unable to get up” response with lifting equipment and an on-scene falls assessment rather than automatic conveyance, with referral to a falls service.
- **Indian relevance**: with rising numbers of elders living alone or with children abroad, long lie is under-recognised. Practical measures are a **charged mobile phone kept at floor level**, ASHA/ANM home visits under **NPHCE**, neighbourhood senior citizen associations, police senior-citizen registration schemes, and the national helpline **Elder Line 14567**.

Current Practice Update

Issue	Textbook position	Current position
Status of long lie	Not named; time taken to get up is a history question only	**Formal high-risk triage criterion (≥1 hour) in World Falls Guidelines 2022**
Floor-rise training	Not mentioned	**Explicitly recommended** for all at-risk older adults
Detection	Not addressed	Wearable and ambient **fall-detection technology** recommended as an adjunct; shortens time-to-help

Exam One-Liners

- Long lie = on the floor ≥1 hour, unable to rise unaided.
- Around half of fallers cannot get up unaided; ~50% of those with a long lie die within 6 months.
- Complications: dehydration, rhabdomyolysis → AKI, hyperkalaemia, pressure injury, hypothermia, pneumonia, delirium, post-fall syndrome.
- Always check creatine kinase, potassium mEq/L and creatinine mg/dL; give aggressive IV fluids.
- Prevention = floor-rise (backward chaining) + personal alarm + phone at floor level + strength training.
- WFG 2022: long lie ≥1 hour = automatic high fall risk.

Q6. Balance and gait assessment

15–20 High-Yield Points (Textbook)

A. Definitions and Physiology

1. **Balance (equilibrium)** is the ability to maintain the **centre of mass of the body within the base of support with minimal postural sway**.
2. Balance depends on the integration of **proprioception, vestibular input, visual input, central coordination and neuromuscular responses**.
3. When standing, changes in orientation are first perceived by **cutaneous proprioceptive sensors in the feet**; the **vestibular apparatus** detects linear and angular acceleration of the head; **vision** detects motion of the visual field. **On a fixed level surface proprioception predominates; when the surface is irregular or moving, vestibular input becomes essential.**
4. **Gait** is the pattern of trunk and limb movement during locomotion over a solid surface — the "manner of walking" or "sequence of foot movements".
5. **Gait assessment is a critical part of the comprehensive geriatric assessment.** At least **one-fifth of community-dwelling older adults report difficulty walking**, rising to **one-half in those aged 85 and above**.

B. Tools for Assessing Balance

6. **30-second chair stand** — number of chair stands completed in 30 seconds with arms crossed on the chest. The most practical tool in a busy clinic; measures both **balance and strength**. It is one component of the **Short Physical Performance Battery (SPPB)**.
7. **Short Physical Performance Battery (SPPB)** — repeated chair stands plus balance testing with **side-by-side, semitandem and tandem stands**, plus gait speed.
8. **4-Stage Balance Test** — side-by-side, semitandem, tandem and one-foot stand. **Normal balance = holding each position for 10 seconds.**
9. **Berg Balance Scale** — a **14-item** test of everyday tasks (sitting unsupported, sit-to-stand, stand-to-sit, transfers, standing unsupported, eyes closed, feet together, tandem standing, one-leg stand, turning the trunk, retrieving objects from the floor, turning 360 degrees, stool stepping, reaching forward). Total score **0–56**. Interpretation: **0–20 wheelchair bound, 21–40 walks with assistance, 41–56 independent**. Validated originally in older stroke patients; predicts functional and motor performance and future falls.
10. **Functional reach test** — dynamic standing balance. The subject stands behind a line and reaches the arm as far forward as possible without stepping. **Fall risk is suspected if reach is less than 10 inches.** It correlates with other balance measures and **predicts future decline in ADLs**, though it may not reliably predict falls.

C. Tools for Assessing Gait

11. **Gait speed** is the simplest and most standardised global measure of health and function. Measured over a **pre-measured 4 metre distance**. **0.6–0.8 m/sec identifies a high-risk population and 0.6 m/sec is the generally accepted cut-off for abnormal gait speed.**
12. **Get Up and Go Test (GUGT) / Timed Up and Go (TUGT)** — rise from a chair, walk a set distance, turn, walk back and sit down. In the inpatient setting sensitivity is **91%** but specificity only **22%**. Even an **untimed** GUGT is clinically useful for identifying specific balance and gait problems. Kane recommends a simple "get up and go" test in **all** patients who have fallen.
13. **Tinetti's Performance-Oriented Mobility Assessment (POMA)** — 14 performance tasks split into balance and gait. **Maximum balance score 16, maximum gait score 12, total 28. Score below 19 = high risk of falls; 19–24 = at risk.** Mean sensitivity 82%, specificity 65%. Takes 10–15 minutes, so it is harder to use in a busy clinic. Interrater agreement is over 85%.
14. The Tinetti **balance** items are: sitting balance, arises, attempts to arise, immediate standing balance (first 5 seconds), standing balance, nudged, eyes closed, turning 360 degrees, sitting down. The **gait** items are: initiation of gait, step length and height, step symmetry, step continuity, path, trunk, and walking stance.
15. **Kane's performance-based assessment** grades each manoeuvre as **normal, adaptive or abnormal**: sitting balance, arising from chair, immediate standing balance (first 3–5 seconds), standing balance, **Romberg**, **sternal nudge** (examiner pushes with light even pressure over the sternum three times), walking, turning balance through 360 degrees, **neck turning**, and **back extension**. This is **more useful than the standard neuromuscular examination for identifying remediable problems**.
16. **Dual-task testing** — walking while counting backwards from 100 by sevens, GUGT while holding a full glass of water, or one-leg stand with eyes closed. Institutionalised older adults who **cannot keep up a conversation while walking have a high risk of falls**. Both young and old prioritise gait over the secondary cognitive task. Clinical utility is still unclear because of variability across studies.

D. Age-Related Findings to Look For

17. Expect **slower gait, reduced step length, increased time in double limb support, reduced range of motion, and increased anterior-posterior postural sway**. Gait speed falls **12–16% per decade**.
18. On perturbation testing, younger people abduct the arms uphill and hinge at hips and trunk to keep the centre of mass away from the tilt; **older subjects have reduced compensatory trunk movement and stretch the arms in the direction of the fall**.
19. Recognise disease-specific gaits: **Parkinson's disease** — gait initiation failure and freezing, better with external visual cues; **post-stroke** — decreased hip and knee flexion during the swing phase; **cerebellar disease** — broad-based ataxic

gait; **vestibular lesion** — veering to the ipsilateral side when walking with eyes closed.

20. **Where to place it in practice**: gait, strength and balance assessment forms the core of the CDC **STEADI** risk assessment using **TUGT, 30-second chair stand and 4-stage balance test**.

Latest Updates (2023–Present)

- **World Falls Guidelines 2022** retain gait and balance assessment as the core of risk stratification and specifically recommend **timed up and go, gait speed, and a balance test such as the 4-stage or Berg scale**. Crucially, the guidelines position gait and balance impairment as the factor that separates **intermediate** from **low** risk in a person with a single fall.
- **Gait speed** is now used as a vital sign and as a core criterion for **sarcopenia** in **EWGSOP2** (≤0.8 m/s) and **AWGS 2019** (<1.0 m/s for Asians — the more appropriate cut-off for Indian patients) and for **frailty** in the Fried phenotype.
- **WHO ICOPE (2nd edition, 2025)** uses the **chair rise test** as the primary screening item for the locomotor capacity domain in integrated care for older people, making it the most relevant tool for Indian primary and community-level screening under NPHCE.
- **Dual-task gait assessment** and **gait variability** (stride time variability) are increasingly validated as markers of **cognitive-motor risk**. The **Motoric Cognitive Risk (MCR) syndrome** — slow gait plus subjective cognitive complaint without dementia — predicts incident dementia and falls, and can be diagnosed without any neuropsychological testing.
- **Instrumented and digital gait assessment** using wearable inertial sensors and smartphone applications gives spatiotemporal parameters (variability, asymmetry, turning) with better fall prediction than stopwatch measures; these are still research or specialist tools, and guidelines caution that no instrumented tool has yet replaced clinical assessment.

Current Practice Update

Issue	Textbook position	Current position
Gait speed cut-off	0.6 m/s abnormal; 0.6–0.8 m/s high risk	Retained for falls, but **≤0.8 m/s (EWGSOP2)** and **<1.0 m/s (AWGS 2019, Asian)** are used for sarcopenia — use the **AWGS cut-off in Indian patients**
Screening tool of choice	STEADI trio: TUGT, 30-s chair stand, 4-stage balance	Same trio endorsed; **WHO ICOPE 2025 uses the chair rise test** as the single locomotor screen for community settings
Dual tasking	Proposed screen, clinical utility unclear	Now linked to **Motoric Cognitive Risk syndrome** and used as a cognitive-motor risk marker
Technology	Not addressed	**Wearable inertial sensors / smartphone gait analysis** improve prediction but do not yet replace clinical tests

Exam One-Liners

- Balance = keeping centre of mass within base of support with minimal sway.
- Fixed level surface → proprioception dominant; uneven or moving surface → vestibular dominant.
- Berg Balance Scale: 14 items, 0–56; ≤20 wheelchair, 21–40 assisted, 41–56 independent.
- Functional reach <10 inches = fall risk.
- 4-stage balance test: hold each position 10 seconds.
- Gait speed over 4 m; abnormal <0.6 m/s (use <1.0 m/s AWGS for Asian sarcopenia).
- Tinetti POMA: balance 16 + gait 12 = 28; <19 high risk, 19–24 at risk.
- TUGT: sensitivity 91%, specificity 22% in inpatients.

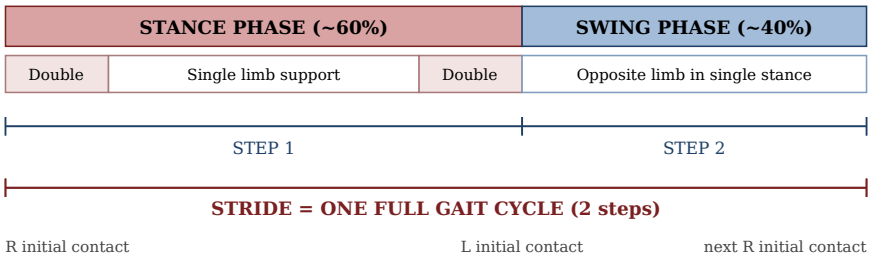
Q7. Phases in human walking (gait cycle), with diagrams

15–20 High-Yield Points

A. Basic Definitions textbook

1. The **gait cycle** is the sequence of events between one event of one limb and the recurrence of the same event in the same limb. It has two phases: **stance phase** and **swing phase**.
2. **Stance phase** — the period when the foot is in contact with the ground. During **double stance** the weight is transferred from one foot to the other.
3. **Swing phase** — during **single stance** the centre of mass of the body passes over the supporting foot in preparation for shifting to the other limb, while the opposite limb swings forward.
4. **Step** is the period from **initial contact of one limb to the initial contact of the contralateral limb**.
5. **Stride** is the period from the **initial contact of a particular limb to the point of initial contact of the same limb**. **A stride equals one gait cycle, and there are two steps in each stride.**

Figure 1. The walking gait cycle (one stride = one gait cycle)



B. Sub-phases of the Gait Cycle supplementary

> Pathy's Figure 78.4 divides the cycle only into **stance, swing, step and stride**. The eight sub-phases below are the standard Rancho Los Amigos description used in rehabilitation and are given here as supplementary content because they are frequently asked in examinations.

6. **Stance phase (approximately 60% of the cycle)** has five sub-phases: **initial contact (heel strike, 0–2%)**, **loading response (0–10%)**, **mid-stance (10–30%)**, **terminal stance (heel off, 30–50%)** and **pre-swing (toe off, 50–60%)**.
7. **Swing phase (approximately 40% of the cycle)** has three sub-phases: **initial swing (acceleration, 60–73%)**, **mid-swing (73–87%)** and **terminal swing (deceleration, 87–100%)**.
8. **Double support occupies about 20% of the cycle** and occurs twice, at the beginning and end of stance. **Double support disappears in running** — this is the defining difference between walking and running.
9. Functional tasks: **weight acceptance** (initial contact and loading response), **single limb support** (mid-stance and terminal stance), and **limb advancement** (pre-swing to terminal swing).

Phase	Sub-phase	% of cycle	Key event
Stance (~60%)	Initial contact (heel strike)	0–2%	Heel touches ground; limb prepares to accept weight
	Loading response	0–10%	Double support; shock absorption; knee flexes slightly
	Mid-stance	10–30%	Single limb support; body passes over the fixed foot
	Terminal stance (heel off)	30–50%	Heel rises; body advances ahead of the supporting foot
	Pre-swing (toe off)	50–60%	Second double support; push-off; limb unloaded
Swing (~40%)	Initial swing (acceleration)	60–73%	Toe leaves ground; hip and knee flex to clear the foot
	Mid-swing	73–87%	Swinging limb passes the stance limb; ankle dorsiflexes
	Terminal swing (deceleration)	87–100%	Knee extends; limb decelerates ready for next initial contact

C. Neural Control of Walking textbook

10. Human locomotion depends on **higher command and control centres in the brainstem and forebrain**, and the pathway used depends on whether the stimulus is **internal or external**.
11. In **normal, internally cued walking**, the **supplementary motor area (SMA)** of the frontal cortex fires significantly **just before gait initiation**, and this preparatory activity is sent to the **primary motor cortex (M1)**.
12. SMA activity is then **switched off by phasic activity from the basal ganglia (BG)**, which provides a non-specific cue that both triggers the sub-movement and instructs the SMA to prepare the next signal. This **BG–SMA interaction ensures smooth running of predictable, well-learned, automatic movement sequences**.
13. When movement is **externally cued**, the BG–SMA pathway is **bypassed** and sensory information feeds directly into the **pre-motor area (PMA)** through visual, auditory and proprioceptive pathways.
14. The motor cortex, basal ganglia and cerebellum then pass input to the **pedunculopontine nucleus (PPN)**, which projects to the **brainstem reticular nuclei**, from which instructions for gait pass along **non-pyramidal pathways into the ventral spinal cord**.
15. **Clinical correlation — Parkinson's disease**: disordered cueing from the basal ganglia disturbs internal rhythm formation and impairs feedback to the SMA, producing **bradykinesia, gait initiation failure and freezing**. PD patients

therefore rely on intact PMA (external cue) pathways — gait improves with **external cues such as stepping over coloured patterns on the ground**. **Vascular dementia and stroke** show a similar compensatory dependence on PMA pathways.

D. Ageing Changes in the Gait Cycle textbook

16. **Gait speed declines 12–16% per decade**, and maximum gait speed by up to **20%**.
17. **Step length is reduced** and **time spent in double limb support is increased** — the body's way of buying stability at the cost of speed.
18. Gait becomes **slower, more uncertain and uncoordinated, and lacks a full range of motion**, from stiffer joint capsules and ligaments and loss of muscle mass.
19. Feet are **not picked up as high**, increasing the tendency to trip. Older men develop a **wide-based, short-stepped gait**; older women a **narrow-based, waddling gait**. These changes correlate with **white matter changes on MRI** and with subsequent cognitive impairment.
20. After acute stroke, patients may show **decreased hip and knee flexion during the swing phase**, causing foot drag and tripping.

Latest Updates (2023–Present)

- **Gait variability** — particularly **stride time variability** and **swing time variability** — is now recognised as a better predictor of falls than mean gait speed alone, because it reflects the rhythmic control that fails early in ageing and neurodegeneration.
- **Instrumented gait analysis** using body-worn inertial measurement units (usually lower back and shanks) now permits routine measurement of the sub-phases — double support time, swing time asymmetry, turning duration and number of steps to turn — outside a gait laboratory. Prolonged **double support time** and increased **turn duration** are consistent markers of fall risk.
- **Gait is treated as a cognitive task.** Current evidence positions **dual-task gait cost** and **Motoric Cognitive Risk syndrome** (slow gait plus subjective cognitive complaint) as predictors of incident dementia, reinforcing the textbook's BG–SMA and PPN model with clinical evidence.
- **External cueing** for Parkinson's disease freezing — laser-line walking sticks, rhythmic auditory cueing and metronome apps — is now standard rehabilitation practice and is the direct clinical application of the PMA bypass pathway described in the textbook.
- In Indian practice, **barefoot walking, squatting and floor-level activities, uneven flooring and thresholds** materially alter gait mechanics and tripping risk, and should be addressed specifically in gait retraining and home assessment.

> **Exam One-Liners**
> - Gait cycle = stance (~60%) + swing (~40%). Double support ~20%, occurs twice.
> - Step = one limb's contact to the other limb's contact. Stride = same limb to same limb = one gait cycle = 2 steps.
> - Stance sub-phases: initial contact, loading response, mid-stance, terminal stance, pre-swing.
> - Swing sub-phases: initial (acceleration), mid, terminal (deceleration).
> - Running is defined by loss of the double support period.
> - Internal cue pathway: SMA → M1, switched off by basal ganglia → PPN → reticular nuclei → ventral cord.
> - External cue pathway bypasses BG–SMA and goes through the pre-motor area — the basis of visual cueing in Parkinson's disease.
> - Ageing: gait speed ↓12–16% per decade, shorter step, longer double support.

Q8. Postural (orthostatic) hypotension — evaluation and management

15–20 High-Yield Points (Textbook)

A. Definition and Epidemiology

1. **Orthostatic (postural) hypotension** is defined as a **drop in systolic blood pressure of 20 mmHg or more on moving from a lying to a standing position**.
2. It occurs in about **20% of older persons**. **Many are asymptomatic**, but the impaired physiological response still causes instability and precipitates falls.
3. It is more common in older adults with **uncontrolled hypertension**, and it is **specifically in that context that it most strongly predicts falls**.
4. Among **nursing home residents it is common even when asymptomatic and is an independent risk factor for recurrent falls**.
5. **Postprandial hypotension** is a separate entity: a fall in blood pressure after a meal, which worsens when the person then stands. Both orthostatic and postprandial hypotension are **more common in the morning**.

B. Pathophysiology and Causes

6. The ageing basis is **reduced baroreflex sensitivity** together with reduced vascular compliance, reduced renal sodium conservation, and blunted heart rate response.
7. **Low cardiac output** from heart failure or **hypovolaemia** (dehydration, acute blood loss, occult gastrointestinal bleeding).
8. **Overtreatment with cardiovascular medication** — diuretics, antihypertensives, nitrates, alpha blockers.
9. **Autonomic dysfunction** — **diabetes mellitus** and **Parkinson's disease**; also multiple system atrophy, where fludrocortisone or midodrine may be needed for symptomatic postural hypotension. Note the textbook point that **Parkinson's disease medication itself can cause orthostatic hypotension**.
10. **Impaired venous return** — venous insufficiency, varicose veins.
11. **Prolonged bed rest with deconditioning** of muscles and reflexes — a listed complication of immobility.
12. Drug groups to suspect in a faller: **diuretics (hypovolaemia), antihypertensives (hypotension), antidepressants (postural hypotension), antipsychotics (sedation, rigidity and postural hypotension), sedatives, hypoglycaemics and alcohol**. Combinations greatly increase risk. Alpha blockers are specifically associated with hip and femur fracture.
13. Autonomic dysfunction causing repeated falls, syncope and transient loss of consciousness may occur **early in dementia with Lewy bodies**, along with urinary incontinence, constipation, impotence and swallowing difficulty.

C. Evaluation

14. **Correct measurement technique is the whole examination.** Take blood pressure and pulse rate **supine, after 1 minute sitting, and after 1 and 3 minutes of standing**. Pathy's assessment scale states that in older people all blood pressures need to be measured at a **minimum of sitting and standing**, with a check for postural hypotension **at one and three minutes after standing**.
15. Check for **postprandial hypotension** if the person falls, is dizzy, is syncopal, or has a stroke or myocardial infarction **within two hours of a meal**.
16. **Look at the pulse response.** A rise in heart rate suggests volume depletion or drug effect; **failure of the heart rate to rise suggests autonomic failure** or beta blockade.
17. Other blood-pressure pitfalls in older people: **pseudohypertension** from arteriosclerotic vessels (screen with the **Osler manoeuvre**, though sensitivity and specificity are poor); **inter-arm difference** — always treat the higher blood pressure; **white-coat hypertension** — always obtain home readings; and a **wide pulse pressure carries a poor prognosis** in older people.
18. **Investigate for the cause**: haemoglobin g/dL, sodium and potassium mEq/L, urea and creatinine mg/dL, blood glucose, ECG, and where indicated echocardiography and autonomic function testing. Evaluate for occult blood loss if indicated.

D. Management

19. **Non-pharmacological first. Eliminate the offending drugs** or reduce the dose. **Rehydrate as appropriate** and evaluate for blood loss. **Support (compression) stockings**, **leg elevation**, and **adaptive behaviours — pausing and getting up slowly, rising in stages**. Pathy adds that **compression stockings and tools around the home to aid slow rising** are helpful for adults with postural hypotension.
20. **Pharmacological** for autonomic dysfunction or idiopathic cases: **mineralocorticoids (fludrocortisone)** and **midodrine hydrochloride**, in addition to support stockings and adaptive behaviour.
21. Additional practical measures: **head-up tilt of the bed by 15–20%**, a glass of water before rising (kept at the bedside overnight), **liberalised salt and fluid intake** where not contraindicated, **small frequent low-carbohydrate meals** for postprandial hypotension (acarbose is listed as useful), avoiding hot baths, large meals and alcohol, and physical counter-manoeuvres.
22. **Do not chase the numbers.** If the drop is mild and the person can understand and use non-pharmacological measures, reassurance and explanation focused on **managing symptoms rather than absolute values** may be all that is required.

Latest Updates (2023–Present)

- The **consensus definition** used by current guidelines is a fall of **≥20 mmHg systolic or ≥10 mmHg diastolic within 3 minutes of standing** (or head-up tilt to at least 60 degrees). Three patterns are now distinguished: **initial OH**

(transient drop >40/20 mmHg within 15 seconds — needs beat-to-beat monitoring), **classical OH** (within 3 minutes) and **delayed OH** (beyond 3 minutes, often an early marker of autonomic failure).

- **World Falls Guidelines 2022** make **lying and standing blood pressure a mandatory element (GRADE 1B)** of multifactorial falls assessment in every older faller, alongside cardiac history, auscultation and a 12-lead ECG.
- **Active standing at the bedside is preferred to tilt-table testing** for routine diagnosis. **Tilt-table testing (with or without carotid sinus massage) is reserved** for ongoing unexplained recurrent syncope refractory to initial management where reflex or delayed orthostatic hypotension is suspected and the result would change management (ESC syncope guidance).
- **Supine hypertension coexists with orthostatic hypotension in a large proportion of patients** and is now managed actively — avoid supine daytime rest, elevate the head of the bed at night, and use short-acting agents at night if needed. The **2024 ESC hypertension guideline**, while lowering targets to systolic 120–129 mmHg generally, explicitly exempts patients with **symptomatic orthostatic hypotension and frailty**.
- **Droxidopa** (a norepinephrine prodrug) is an additional approved option for neurogenic orthostatic hypotension in some countries; it is not widely available in India, where **fludrocortisone and midodrine remain the mainstays** and are both inexpensive and available. Pyridostigmine is a further option that raises standing blood pressure without worsening supine hypertension.
- **Orthostatic hypotension, cognitive impairment and reduced mobility** are described as an interacting triad in older fallers, and orthostatic hypotension is now also recognised as a risk marker for **incident dementia, stroke and cardiovascular events**, not merely for falls.
- **Indian relevance**: high prevalence of **diabetic autonomic neuropathy**, hot climate with **dehydration and heat-related vasodilatation**, and widespread use of diuretics and alpha blockers make orthostatic hypotension particularly common; salt liberalisation must be balanced against heart failure and hypertension.

Current Practice Update

Issue	Textbook position	Current position
Definition	Fall of >20 mmHg systolic on standing	**≥20 mmHg systolic OR ≥10 mmHg diastolic within 3 minutes**; sub-types **initial, classical, delayed**
Measurement	Supine, 1 min sitting, 1 and 3 min standing	Same, now a **mandatory graded recommendation (1B)** in every older faller; active stand preferred over tilt
Supine hypertension	Not addressed	**Actively managed** — head-up bed tilt, avoid daytime supine rest, short-acting nocturnal agents
Drug therapy	Fludrocortisone and midodrine	Same first line; add **droxidopa** and **pyridostigmine** where available; droxidopa not widely available in India
BP targets	Not linked to OH	2024 ESC targets (SBP 120–129) **explicitly relaxed** in symptomatic OH and frailty
Prognosis	Framed as a fall risk factor	Also an independent risk marker for **dementia, stroke and cardiovascular events**

Exam One-Liners

- OH = SBP fall ≥20 mmHg (or DBP ≥10 mmHg) within 3 minutes of standing.
- Present in ~20% of older persons; often asymptomatic but still causes falls.
- Measure supine, 1 min sitting, then 1 and 3 min standing.
- No rise in heart rate with the BP drop = neurogenic (autonomic failure).
- Check postprandial hypotension if events occur within 2 hours of a meal.
- Osler manoeuvre screens for pseudohypertension; always treat the higher arm.
- Treatment ladder: stop culprit drugs → hydration, salt, stockings, staged rising, head-up bed → fludrocortisone / midodrine.
- Manage supine hypertension at the same time; relax BP targets in symptomatic OH.

Q9. Syncope

> **Textbook coverage statement.** Pathy's 6th edition covers this in **Chapter 52 – Syncope and Non-Epileptic Attacks (pp. 664–673)** and **Chapter 33 – Hypotension (pp. 419–426)**. The uploaded files contain **Chapters 75, 78 and 79 in full plus the index**; the text of Chapters 52 and 33 is **not present** in the uploaded material. The textbook content below is therefore taken from **Kane's Chapter 9** and from the cross-references within Pathy Chapters 78 and 79, and post-textbook evidence is clearly separated.

15–20 High-Yield Points

A. Definition and Importance textbook

1. **Syncope** is **sudden, transient loss of consciousness** with loss of postural tone and spontaneous complete recovery. In the falls setting, **if there is a clear history of loss of consciousness, a cause for true syncope must be sought**.
2. **Distinguishing a fall from syncope and from a seizure is the first step** in evaluating any older person who has fallen — it changes the entire diagnostic pathway.
3. Syncope in the older person is difficult to diagnose because falls are often **unwitnessed**, the patient may not recall the event, and the causes (arrhythmia, TIA, postural hypotension) are **transient**. **A precise cause remains unidentified in 40–60% of older patients.**
4. A history of loss of consciousness after a fall should raise suspicion of a **cardiac event (transient arrhythmia or heart block)** causing syncope or near-syncope, or a **seizure** (especially if there has been incontinence).

B. Causes in Older People textbook

5. **Reflex (neurally mediated)**: **vasovagal responses**; **carotid sinus hypersensitivity**; and **situational** syncope from reflex increase in vagal tone caused by **cough, straining at stool or micturition**, leading to bradycardia and hypotension.
6. **Cardiovascular**: **bradycardia and heart block, tachyarrhythmias, and aortic stenosis**. **Syncope is a symptom of aortic stenosis and is an indication to evaluate the patient for valve replacement.**
7. **Orthostatic**: postural hypotension from hypovolaemia, low cardiac output, autonomic dysfunction, impaired venous return, prolonged bed rest, drugs, and **postprandial hypotension**.
8. **Acute neurological events**: TIA, stroke, seizure. Note that a history of transient vertigo alone is not enough to diagnose a posterior circulation TIA — **one or more other symptoms (visual field cut, dysarthria, ataxia, limb weakness) must accompany the vertigo**.
9. **Other**: **pulmonary embolism** and **metabolic disturbances such as hypoxia and hypoglycaemia**.
10. **Drugs** are a major contributor: antihypertensives, diuretics, antidepressants, antipsychotics, hypoglycaemics and alcohol. **Acetylcholinesterase inhibitors are specifically associated with syncope.**
11. **Dementia with Lewy bodies** is an important cause — **syncope and other transient episodes of unresponsiveness, repeated falls and severe autonomic dysfunction are supportive diagnostic features**.

C. Evaluation textbook

12. The **history is the single most valuable investigation**. Record: circumstances (location, time, activity, whether alone), relationship to **posture change, head turning, cough, micturition, a meal or drug intake**, **premonitory symptoms** (light-headedness, palpitations, chest pain, breathlessness, focal neurology, aura, incontinence), what is remembered immediately after, and whether a witness can confirm loss of consciousness.
13. **Vital signs** to detect an acute illness. **Postural blood pressure and pulse supine, sitting, and at 1 and 3 minutes standing.**
14. If carotid sinus sensitivity is suspected, perform **gentle carotid sinus massage for 5 seconds** and observe for **profound bradycardia (50% reduction in heart rate) or a long pause**.
15. **Cardiovascular examination** for arrhythmia and aortic stenosis. Because both are serious, treatable and difficult to detect clinically, refer for **continuous cardiac monitoring and echocardiography** when suspected. **Echocardiography should be considered in all patients with a suggestive history and a systolic murmur, or a delayed carotid upstroke.**
16. **Cardiac monitoring must document a temporal association between a specific arrhythmia and symptoms before that arrhythmia is diagnosed and treated as the cause** — asymptomatic ectopy is common in older people and must not be over-interpreted.
17. **Do not send all patients for extensive diagnostic studies or cardiac monitoring.** CT or MRI should be reserved for patients with a high suspicion of an intracranial lesion or seizure disorder. Non-invasive vascular studies if the history suggests anterior circulation TIA.
18. The **minimum investigations in a patient who falls** are **lying and standing blood pressure and a 12-lead ECG**.

D. Management textbook

19. Treat the identified cause: **antiarrhythmics for tachyarrhythmias, pacemaker for bradyarrhythmias, valve surgery for aortic stenosis with syncope** — noting that antiarrhythmic and pacing treatment is indicated **only if the cardiac disturbance is clearly related to symptoms**.
20. **Cardiac pacing in carotid sinus hypersensitivity** is one of the single-factor interventions with demonstrated efficacy in fall prevention.
21. Withdraw or reduce culprit drugs, correct volume depletion and anaemia, treat postural hypotension, and give **education on prodrome recognition and safe positioning**.

Latest Updates (2023–Present)

- **Classification (ESC 2018 syncope guidelines, still current)** divides syncope into three groups: **reflex (neurally mediated) — vasovagal, situational, carotid sinus syndrome**; **syncope due to orthostatic hypotension**; and **cardiac syncope — arrhythmic or structural**. Syncope sits within the broader concept of **transient loss of consciousness (TLoC)**, which has four defining characteristics: **short duration, abnormal motor control, loss of responsiveness, and amnesia for the period of loss of consciousness**.
- A **detailed clinical history alone distinguishes syncope from other forms of TLoC in about 60% of cases**. The **initial evaluation** is history, physical examination including orthostatic blood pressure, and a 12-lead ECG.
- **Risk stratification has replaced blanket admission. Low-risk** patients with clear reflex, situational or orthostatic features need **no further tests in the emergency department** — reassurance and counselling suffice. **High-risk** patients need an intensive diagnostic approach, monitoring in a resuscitation-capable area (usually up to 6 hours in the ED and 24 hours in hospital) and expert syncope review, which can often be delivered in an **outpatient syncope unit**. This is reinforced by the **2024 European Society for Emergency Medicine consensus statement**.
- **Tilt-table testing has fallen sharply out of favour.** It is now used only where there is genuine diagnostic uncertainty — suspected reflex syncope, orthostatic hypotension, POTS or psychogenic pseudosyncope — and there is a class III recommendation against using it to select or assess drug treatment.
- **Prolonged rhythm monitoring has become central.** The **implantable loop recorder (ILR)** is recommended early in recurrent unexplained syncope with high-risk features or a likely arrhythmic mechanism, and detects a diagnosis far more often than conventional Holter monitoring. Smartphone-linked and patch monitors are increasingly used where an ILR is not affordable — relevant to Indian practice.
- **Reflex syncope treatment** now emphasises **education and reassurance, adequate fluid and salt intake, physical counter-pressure manoeuvres, and withdrawal of hypotensive drugs**. **Pacing is reserved for older patients with documented spontaneous asystolic pauses** (the ISSUE-3 and BioSync CLS evidence) and is used with caution.
- **Overlap with falls is now formally recognised. World Falls Guidelines 2022** require cardiovascular assessment in every faller and state that further cardiac work-up is needed **only** when syncope is described or witnessed, or falls are recurrent and unexplained — an explicit attempt to stop over-investigation.
- **Indian context**: syncope evaluation is limited by cost and access to ILR and tilt testing; a rigorous history, orthostatic blood pressure, 12-lead ECG, echocardiography and ambulatory ECG will resolve most cases. **Anaemia, dehydration in hot climates, tuberculosis-related autonomic disturbance, and polypharmacy from multiple prescribers** are common contributors, and cardiac procedures are covered under **AB-PMJAY**.

Current Practice Update

Issue	Textbook position	Current position
Framework	List of causes of syncope in older people	**Three-group ESC classification** (reflex / orthostatic / cardiac) within the concept of **TLoC**
Who gets investigated	Do not investigate everyone; monitoring must correlate with symptoms	Formal **low-risk vs high-risk stratification**; low risk discharged from ED without tests; high risk monitored and referred to a **syncope unit**
Tilt-table testing	Not emphasised	**Markedly restricted** — only for genuine diagnostic uncertainty; not for selecting therapy
Rhythm monitoring	Continuous monitoring if arrhythmia suspected	**Implantable loop recorder used early** in recurrent unexplained syncope with suspected arrhythmia
Reflex syncope therapy	Not detailed	**Education, salt and fluid, counter-pressure manoeuvres, drug withdrawal**; pacing only for documented spontaneous asystole

Exam One-Liners

- Syncope = transient LOC from global cerebral hypoperfusion, with spontaneous complete recovery.
- Three ESC groups: reflex, orthostatic hypotension, cardiac.
- Cause remains unidentified in 40–60% of older patients.
- Initial evaluation = history + examination with orthostatic BP + 12-lead ECG; history alone diagnoses ~60%.
- Syncope in aortic stenosis = indication to assess for valve replacement.
- Arrhythmia must be temporally linked to symptoms before it is blamed.
- ILR early for recurrent unexplained syncope; tilt testing now rarely indicated.
- Incontinence after the event and a prolonged post-ictal phase point to seizure, not syncope.

Q10. Hypersensitive carotid sinus syndrome

> **Textbook coverage statement.** Pathy's 6th edition covers carotid sinus hypersensitivity in **Chapter 33 – Hypotension (pp. 419, 423–424)**, with sections on definition and measurement, pathophysiology, prevalence, symptoms, diagnostic criteria, prevention, and non-pharmacological and pharmacological treatment, and in **Chapter 52 (pp. 665, 668)** as carotid sinus syndrome. **The text of these chapters is not present in the uploaded files** — only the index entries. The textbook content below is taken from **Kane's Chapter 9** and from the cross-references in **Pathy Chapters 78 and 79**; the remainder is clearly flagged as supplementary.

15–20 High-Yield Points

A. Definition and Terminology

1. **Carotid sinus hypersensitivity (CSH)** is an **exaggerated reflex response to carotid sinus stimulation**. **Carotid sinus syndrome (CSS)** is the term used when this abnormal response **reproduces the patient's spontaneous symptoms** — syncope, pre-syncope or unexplained falls. **CSH is a finding; CSS is a diagnosis.**
2. It is a form of **reflex (neurally mediated) syncope** and is a recognised **cardiovascular cause of falls** in older people, listed alongside arrhythmias and aortic stenosis.
3. **Some older individuals have sensitive carotid baroreceptors and are susceptible to syncope resulting from a reflex increase in vagal tone** caused by **cough, straining at stool or micturition**, leading to **bradycardia and hypotension**.

B. Pathophysiology supplementary

4. The **carotid sinus baroreceptor** at the carotid bifurcation sends afferents through the **glossopharyngeal nerve (Hering's nerve)** to the **nucleus tractus solitarius** in the medulla, producing **vagal efferent bradycardia** and **sympathetic withdrawal causing vasodilatation**.
5. In CSH this reflex arc is exaggerated. Current thinking is that the abnormality lies **centrally and in the efferent limb** rather than in the sinus itself, with proposed contributions from **sternocleidomastoid muscle denervation and altered central neurotransmission**; it is associated with **ageing, hypertension, coronary artery disease and neurodegenerative disease**.

C. Types

6. **Cardio-inhibitory type** (about two-thirds): predominant **asystole or profound bradycardia**.
7. **Vasodepressor type**: predominant **fall in blood pressure** without significant bradycardia.
8. **Mixed type**: both components present. **This classification determines treatment** — only the cardio-inhibitory and mixed forms respond to pacing.

D. Clinical Features

9. **Syncope, pre-syncope, dizziness and — characteristically in older people — unexplained falls with amnesia for the event**. Many older patients deny loss of consciousness and describe only "my legs gave way".
10. Symptoms are typically **triggered by neck movement** — turning or extending the head, looking up, a tight collar, shaving, or a sudden turn while walking.
11. It is **under-diagnosed** because the events are unwitnessed, brief and often not remembered, and because it is not looked for.

E. Diagnosis — Carotid Sinus Massage

12. **Kane's bedside technique**: if the history suggests carotid sinus sensitivity, **the carotid can be gently massaged for 5 seconds** to observe whether this precipitates **profound bradycardia (a 50% reduction in heart rate) or a long pause**.
13. **Standard formal criteria** supplementary: massage the carotid sinus at the level of the cricoid cartilage, just medial to the sternocleidomastoid, for **5–10 seconds**, with **continuous ECG and beat-to-beat blood pressure monitoring**, right side first, then the left, and repeated **both supine and at 70 degrees head-up tilt** if the supine test is negative. A positive test is **ventricular pause ≥3 seconds (cardio-inhibitory)** and/or a **fall in systolic blood pressure ≥50 mmHg (vasodepressor)**.
14. **Diagnosis of carotid sinus syndrome requires reproduction of the patient's spontaneous symptoms**, not merely an abnormal haemodynamic response.
15. **Contraindications** supplementary: **myocardial infarction, TIA or stroke within the preceding 3 months**, a **carotid bruit** (unless significant stenosis has been excluded by Doppler), and previous ventricular tachycardia or ventricular fibrillation. Complications are uncommon (transient neurological deficit in well under 1%), but resuscitation facilities and atropine should be available.

F. Management

16. **Remove precipitants and aggravating drugs**: avoid tight collars and sudden neck movements; withdraw or reduce **beta blockers, digoxin, alpha blockers, nitrates, diuretics, and cholinesterase inhibitors**.
17. **Non-pharmacological measures for the vasodepressor component**: adequate salt and fluid intake, **compression stockings**, staged rising, and physical counter-pressure manoeuvres.
18. **Cardiac pacing is the definitive treatment for the cardio-inhibitory and mixed forms.** Pathy lists **"pacemaker implants (cardiac pacing) in patients with carotid sinus hypersensitivity" among the single-factor interventions that have demonstrated efficacy in preventing falls**. **Dual-chamber (DDD) pacing is used** because ventricular pacing alone can provoke pacemaker syndrome.
19. **The vasodepressor component does not respond to pacing**, and residual symptoms after pacing usually mean an

unrecognised vasodepressor element — this is the commonest reason for “pacing failure”.

20. Because carotid sinus hypersensitivity is a **correctable cause of falls**, it must be actively sought in any older person with **recurrent unexplained falls**, alongside postural hypotension and vasovagal syncope, with lying and standing blood pressure and a 12-lead ECG as the minimum work-up.

Latest Updates (2023–Present)

- **The evidence for pacing in fallers is genuinely conflicting, and this is the key discussion point.** The landmark **SAFE PACE trial (Kenny et al., JACC 2001)** randomised older patients attending an emergency department after a non-accidental fall who had cardio-inhibitory CSH to dual-chamber pacing or standard care: **falls fell by about two-thirds (odds ratio 0.42)** and syncope by half. However, the trial was **unblinded**. The subsequent **randomised, double-blind, placebo-controlled crossover trial (pacemaker programmed on DDD/RDR versus off ODO, Ryan et al., 2010)** in recurrent fallers with CSH **did not confirm a benefit**, indicating a substantial **placebo and reporting effect** in the unblinded studies.
- Current guidance therefore restricts pacing to patients with a **documented cardio-inhibitory response that reproduces symptoms**, particularly **recurrent unpredictable syncope**, and is far more cautious about pacing for falls alone.
- **World Falls Guidelines 2022** reflect this restraint: cardiovascular assessment (history, auscultation, lying and standing BP, 12-lead ECG) is mandatory in every faller, but **further cardiovascular investigation — including carotid sinus massage — is reserved for those with described or witnessed syncope or pre-syncope, or recurrent unexplained falls**. Routine carotid sinus massage in all fallers is no longer recommended.
- The **high prevalence of asymptomatic carotid sinus hypersensitivity in healthy older people** (rising with age and reaching a substantial proportion of those over 80) is now emphasised. This means a positive test in an asymptomatic person is a **frequent incidental finding**, and the requirement for **symptom reproduction** before diagnosing carotid sinus syndrome has become stricter.
- **Associations with neurodegeneration** — particularly **dementia with Lewy bodies and Parkinson’s disease** — are increasingly recognised, so CSH in an older faller should prompt assessment for cognitive impairment and autonomic failure rather than being treated as an isolated cardiac problem.
- **Indian context**: carotid sinus massage is a **free, bedside, zero-cost test** and is under-used in Indian falls practice; it should be performed with ECG monitoring in any older patient with recurrent unexplained falls or syncope. Permanent pacemaker implantation is covered under **AB-PMJAY**, but given the conflicting trial evidence, pacing should be restricted to documented symptomatic cardio-inhibitory disease.

Current Practice Update

Issue	Textbook position	Current position
Positive test threshold	Kane: 50% fall in heart rate or a long pause on 5 seconds of massage	Formalised as **pause ≥3 seconds and/or systolic fall ≥50 mmHg**, tested **supine and at 70° head-up tilt**
CSH vs CSS	Terms used loosely	**Strict distinction** — carotid sinus syndrome requires **reproduction of spontaneous symptoms**; asymptomatic CSH is common in the very old and should not be treated
Pacing for falls	Listed as an intervention with demonstrated efficacy in fall prevention (SAFE PACE)	**Contested** — the double-blind crossover trial was negative; pacing now restricted to **documented symptomatic cardio-inhibitory disease**, chiefly recurrent unpredictable syncope
Who gets carotid massage	Perform if history suggests it	**Only** with syncope/pre-syncope or recurrent unexplained falls (WFG 2022); not routine in all fallers

Exam One-Liners

- CSH = abnormal response to carotid sinus massage; CSS = abnormal response PLUS reproduction of symptoms.
- Types: cardio-inhibitory (~two-thirds), vasodepressor, mixed.
- Positive test: pause ≥3 seconds and/or systolic BP fall ≥50 mmHg.
- Massage 5–10 seconds, right then left, supine then 70° head-up tilt, with ECG and BP monitoring.
- Contraindicated within 3 months of MI, TIA or stroke; and with a carotid bruit.
- Dual-chamber pacing for the cardio-inhibitory and mixed forms only; vasodepressor component does not respond.
- SAFE PACE (unblinded) showed a two-thirds fall reduction; the double-blind crossover trial was negative.
- Triggers: neck turning or extension, tight collar, shaving, cough, micturition, straining.

Q11. Benign paroxysmal positional vertigo — diagnosis and management

15–20 High-Yield Points (Textbook)

A. Definition and Cause

1. **BPPV** is characterised by **momentary, short-lived episodes of vertigo occurring on assuming specific head positions**, particularly with **neck extension**, and typically appearing when **lying down on one ear, bending forward, or looking up**.
2. It is **the most common clinical syndrome after head injury**, but is also seen in older people as an **idiopathic disorder** or **secondary to vestibular neuritis**. Even **trivial head injury** can cause it, with onset days or weeks later.
3. **Canalithiasis** is the accepted mechanism and explains most of the features: **free-floating particles (otoconia) within a semicircular canal**. With a change of head position the cluster of particles moves in the canal and has a **"plunger" effect within the narrow canal, deflecting the cupula** and causing a brief paroxysm of vertigo and nystagmus.
4. **Cupulolithiasis** is the alternative mechanism, in which the free-floating particles **adhere to the cupula** of a semicircular canal.
5. Although BPPV can affect **each of the semicircular canals, posterior canal involvement is the most common**.

B. Clinical Features in the Older Patient

6. The **vertigo is particularly severe**, and the older patient is **frequently extremely afraid**, because the attacks are very sudden and may cause **a drop to the ground and vomiting**.
7. This leads to **anxiety** — partly fear of embarrassment if an attack happens in a public place, and partly fear of being **incapacitated at home, unable to reach help**.
8. **The diagnosis is frequently overlooked.** The clinician often sees only an extremely anxious older patient who finds it difficult to explain such brief yet severe symptoms.
9. **Frequently the only abnormal clinical sign is benign positional nystagmus of paroxysmal type on the Dix-Hallpike manoeuvre.**

C. The Dix-Hallpike Manoeuvre

10. **Technique**: the patient sits close to the top end of a flat examination couch. The head is held firmly between the examiner's hands and **turned 30–45 degrees to the right or left**. The patient is then **carried rapidly backward with the head over the edge of the couch**, and the eyes are carefully observed. If nystagmus develops it is observed until it disappears, or for two to three minutes until it is clear that it is persistent. The patient is returned upright and the procedure repeated on the other side.
11. **The manoeuvre must be performed briskly and correctly at the first attempt**, because symptoms and signs **adapt and fatigue on repeated testing**.
12. **Peripheral (benign paroxysmal) findings**: after a **latent period of a few seconds** in the head-back position, **severe vertigo develops lasting less than one minute**, during which the patient may be extremely distressed and nauseated. The **nystagmus is rotatory and directed toward the undermost ear**.

Feature	Benign paroxysmal type (peripheral)	Central type
Latent period	**2–20 seconds**	None
Adaptation	**Disappears in 50 seconds**	Persists
Fatigability	**Disappears on repetition**	Persists
Vertigo	**Always present**	Typically absent
Direction of nystagmus	**To the undermost ear**	Variable
Incidence	Relatively common	Relatively uncommon

Cases that do not fit either category, and all "central" patterns, should be investigated for neurological disease.

D. Management

13. **Positional (particle repositioning) manoeuvres** are the treatment. Specific movements of the head allow the **offending debris in the semicircular canal to be moved by gravitation into the utricle**.
14. The **Epley manoeuvre** is the most commonly used manoeuvre for **posterior canal BPPV**. It is **effective in 80–85% of patients at the first attempt and in a further 10% at a second attempt**.
15. **Semont's manoeuvre** is another specific manoeuvre for posterior canal BPPV.
16. **Pre-treatment sedation is not required** except for the most anxious of patients, and **the manoeuvre is as effective in older people as in younger people**.
17. **Relapses may occur, and the manoeuvre should then simply be repeated.**
18. In the small percentage in whom particle repositioning fails, and in **intractable cases with severe disabling symptoms**, **plugging of the posterior semicircular canal, or section of the posterior and ampullary (singular) nerve**, can be considered.
19. **A combination of canalith repositioning manoeuvre and vestibular rehabilitation improves benign positional vertigo in the elderly.** Although the repositioning manoeuvre is the most effective treatment, **vestibular rehabilitation can be added to improve results, particularly given the high recurrence rate** of positional vertigo.
20. **Drugs have a very limited role.** Vestibular sedatives such as **cinnarizine** are of value in acute vertigo but have a very limited role in chronic vestibular syndromes. **Prochlorperazine should be avoided** because of the rare but irreversible syndrome of **extrapyramidal dysfunction**. Psychotropic drugs should be given only for specific psychiatric indications,

as they **interfere with vestibular compensation**.

21. Because the condition causes drop attacks, vomiting and profound anxiety in older people, **a clear explanation of the benign nature of the condition is an essential part of treatment**.

Latest Updates (2023–Present)

- The **AAO-HNSF Clinical Practice Guideline: BPPV (Update)** remains the reference standard and its key action statements are examinable: **diagnose posterior canal BPPV with the Dix-Hallpike test**; if Dix-Hallpike is negative but BPPV is suspected, **perform the supine roll test for lateral (horizontal) canal BPPV** — here the nystagmus is **predominantly horizontal**, geotropic or apogeotropic, in contrast to the **upbeating torsional** nystagmus of posterior canal BPPV.
- Guideline recommendations that differ from older teaching: **do not routinely order imaging or vestibular testing** when the diagnosis is clear; **treat posterior canal BPPV with a canalith repositioning procedure**; **do not impose post-procedural postural restrictions**; **do not routinely use vestibular suppressants such as antihistamines and benzodiazepines**; **observation with follow-up is an acceptable initial option**; **vestibular rehabilitation may be offered**; and **reassess within one month** to confirm resolution.
- The guideline also mandates **assessment for factors that modify management — impaired mobility or balance, CNS disorders, lack of home support and increased risk of falling** — which is precisely the older Indian patient.
- **Canal conversion** is now formally described: lateral canal BPPV may appear **after** an Epley manoeuvre performed for posterior canal BPPV, as debris migrates from the posterior into the lateral canal. Recognise it rather than assuming treatment failure.
- A **2025 network meta-analysis** of 20 randomised trials (2,089 patients) for posterior canal BPPV ranked the **Epley manoeuvre highest for overall effectiveness (SUCRA 97.84%)**, significantly better than **Semont**, **Brandt-Daroff** and control. Epley and Semont were both superior to other interventions for cure rate. **Brandt-Daroff exercises are inferior and should not be first-line.**
- **Modified Epley technique**: a 2023 randomised trial found that **prolonging the healthy-side lying and final head-bowed positions to about 5 minutes** raised first-attempt success from 63% to 85%, achieved 100% success after two attempts and prevented canal switching; a 2024 simulation study supported longer hold times and a modified rotation angle.
- **Recurrence and bone health**: BPPV recurs in about **10–30% within a year**, more with older age, head trauma, osteoporosis and low vitamin D. A 2024 systematic review and meta-analysis confirmed a **higher prevalence of osteoporosis in BPPV patients**, and randomised evidence supports **vitamin D and calcium supplementation in patients with recurrent BPPV and low serum vitamin D** to reduce recurrence. This links BPPV directly to fracture prevention in the older Indian patient, in whom vitamin D deficiency is near-universal.
- **Practical Indian relevance**: BPPV is the commonest treatable cause of dizziness in older adults, needs **no equipment and no investigation**, and is curable in a single outpatient visit — making Dix-Hallpike and Epley among the highest-yield skills in a resource-limited geriatric clinic.

Current Practice Update

Issue	Textbook position	Current position
Canal involved	Posterior canal most common; Dix-Hallpike is the test	Same, plus mandatory **supine roll test** for lateral canal if Dix-Hallpike negative; recognise **canal conversion** after Epley
Post-procedure restrictions	Not addressed	**Explicitly not recommended** — no need to sleep upright or avoid head movement
Drugs	Cinnarizine for acute vertigo only; avoid prochlorperazine	Stronger: **do not routinely use vestibular suppressants at all in BPPV** — they delay compensation and increase falls
Choice of manoeuvre	Epley most common; Semont an alternative	2025 network meta-analysis: **Epley ranks first (SUCRA 97.8%)**; **Brandt-Daroff clearly inferior**. **Modified Epley** with 5-minute holds improves first-attempt success
Recurrence prevention	Repeat the manoeuvre; add vestibular rehabilitation	Same, plus **check and correct vitamin D and calcium**, and assess for osteoporosis
Follow-up	Not specified	**Reassess within one month** to document resolution or persistence

Exam One-Liners

- Mechanism: canalithiasis (free otoconia) or cupulolithiasis (otoconia adherent to cupula); posterior canal commonest.
- Dix-Hallpike: head turned 30–45°, rapid backward to head-hanging position.
- Peripheral nystagmus: latency 2–20 s, lasts <1 min, adapts in 50 s, fatigues, rotatory towards the undermost ear, vertigo always present.
- Central: no latency, persists, does not fatigue, variable direction, vertigo typically absent.
- Epley cures 80–85% at first attempt, a further 10% at second attempt; equally effective in the elderly.
- No sedation needed; no post-procedure postural restrictions; no vestibular suppressants.
- Lateral canal BPPV: supine roll test, horizontal nystagmus.
- Intractable cases: posterior canal plugging or singular neurectomy.

- Correct vitamin D to reduce recurrence; reassess at one month.

Q12. Approach to a patient with dizziness

15–20 High-Yield Points (Textbook)

A. Terminology and Principle

1. **Vertigo** is defined as **“an hallucination of movement”** and is the cardinal manifestation of a disordered vestibular system. **Dizziness** is a lay term meaning “a feeling of being in a whirl or in a daze or as if about to fall”, and is associated with a **multiplicity of general medical disorders**.
2. **This semantic distinction is rarely volunteered by the older patient**, who more often complains of feeling **faint, swimmy or light-headed**. Therefore **all complaints of disorientation are best considered within a single diagnostic approach**.
3. Dizziness in older people is **usually multifactorial** — the result of **central pathology and/or multiple sensory deficits** (uncorrectable visual impairment, neuropathy, vestibular deficits, cervical spondylosis, orthopaedic disorders interfering with joint mechanoreceptors) rather than vestibular degeneration alone. Vestibular degenerative change tends to occur **symmetrically**, and symptoms arise from **asymmetry** of afferent input.
4. **Do not attribute vertigo to ‘age’.** A thorough history and examination will often give a clear direction as to diagnosis, and correct diagnosis allows treatment of many peripheral and central vestibular disorders.

B. History

5. The three key axes are the **character, time course and associated symptoms** of the dizziness.
6. **Character**: **peripheral labyrinthine** disease classically gives **acute, unprecipitated, short-lived attacks of rotational disequilibrium with nausea and vomiting** (rarely diarrhoea); **central vestibular** disease gives a **more insidious, protracted sense of instability**.
7. **Exceptions to remember**: short-lived attacks may also occur in **epilepsy and vertebrobasilar artery ischaemia**; protracted instability may also occur with **uncompensated peripheral vestibular disorders, bilateral vestibular failure and psychogenic disequilibrium**.
8. Other typical vestibular symptoms are a sensation of **being pulled downward or sideways, of the room tilting, and of swimming, floating and light-headedness**. **A clear description of subjective or objective motion raises suspicion of vestibular pathology**, whereas light-headedness, swimminess or faintness suggests a **general medical or neurological disorder**.
9. Always take a full **drug history** (ototoxic and vestibulotoxic drugs, antihypertensives, sedatives), and ask about **hearing loss, tinnitus and aural fullness**, **headache**, and **relationship to posture, head position, meals, cough or micturition**.

C. Causes of Dizziness in the Elderly (Pathy Table 75.1)

10. **General medical — haematological**: anaemia, polycythaemia, hyperviscosity syndromes.
11. **General medical — cardiovascular**: **postural hypotension, carotid sinus syndrome, dysrhythmias, mechanical dysfunction, shock**.
12. **General medical — metabolic/endocrine**: hypo- and hyperglycaemia, thyroid disease, chronic renal failure, alcohol.
13. **Neurological — supratentorial**: trauma, neoplasia, epilepsy, cerebrovascular disease, syncope, psychogenic.
14. **Neurological — infratentorial**: vertebrobasilar insufficiency, subclavian steal, **Wallenberg’s syndrome**, anterior inferior cerebellar artery syndrome, degenerative disorders including neuropathy, tumours including those of the vestibulocochlear nerve, infective disorders (Ramsay-Hunt, neurosyphilis, tuberculosis), foramen magnum abnormalities, cerebellar degeneration, basal ganglion disease, demyelinating disease.
15. **Otological**: drug-induced/ototoxic, degenerative (**BPPV**), post-traumatic syndrome, infection, vascular, tumours, **Menière’s disease**, otosclerosis and Paget’s disease, autoimmune disorders.
16. **Others**: **migraine** and **multisensory dizziness syndrome**.

D. Examination

17. **Otological examination**, with audiological testing where symptoms suggest vertigo — this helps differentiate inner ear from central nervous system involvement.
18. **Vestibulo-ocular assessment**: look for spontaneous nystagmus; perform the **head thrust (head impulse) test** — on a rapid head thrust to one side the eyes move with the head and lose the target, and **corrective saccades** are then required to bring the visual focus back, indicating **vestibular hypofunction on that side**.
19. **The presence of positional nystagmus is a most valuable and most frequently overlooked sign** and should be sought by a **briskly performed Dix-Hallpike manoeuvre** in every dizzy older patient.
20. **Vestibulospinal assessment**: **Romberg** and postural sway; note that older patients with unilateral vestibular hypofunction show **markedly increased postural sway** compared with younger patients with the same disorder. **Gait testing** — ask the patient to walk toward a fixed point with eyes closed; a tendency to **veer to one side** usually indicates an **ipsilateral peripheral vestibular disturbance** but may occasionally be cerebellar. **Broad-based ataxic gait** suggests cerebellar disease.
21. **Anxious older patients tend to fall backward “like a wooden soldier”**, which indicates a non-organic component — but this is **almost always seen in the presence of an underlying abnormality** that will be identified on full examination.
22. **Screening in the CGA**: ask “**Do you get dizzy, or does your head spin around?**” If yes, **check for postural hypotension, perform the Dix-Hallpike manoeuvre for BPPV, check haemoglobin, and apply the Geriatric Depression Scale**.

Latest Updates (2023–Present)

- **The "what do you mean by dizzy?" question has been abandoned.** Symptom quality (vertigo vs light-headedness vs imbalance) is unreliable and inconsistent when patients are re-questioned. The current standard is the **ATTEST / TiTrATE approach**, which classifies by **Timing and Triggers** instead: **Acute Vestibular Syndrome (AVS)**, **Triggered Episodic Vestibular Syndrome (t-EVS)**, **Spontaneous Episodic Vestibular Syndrome (s-EVS)** and **chronic vestibular syndrome**.
- **t-EVS** (seconds, triggered by position change) → test with **Dix-Hallpike and supine roll** → BPPV or orthostatic hypotension. **s-EVS** (minutes to hours, spontaneous) → vestibular migraine, Menière's disease, TIA, arrhythmia, panic. **AVS** (continuous, days, with nausea, nystagmus and gait unsteadiness) → vestibular neuritis versus posterior circulation stroke.
- **HINTS examination** (**H**ead **I**mpulse, **N**ystagmus, **T**est of **S**kew) is used **only in acute vestibular syndrome**. A **central (dangerous) pattern** is a **normal head impulse test, direction-changing gaze-evoked nystagmus, or skew deviation**. **HINTS performed by a trained examiner is more sensitive than early MRI** for posterior circulation stroke in the first 48 hours, since early diffusion-weighted MRI misses up to 15–20% of small posterior fossa infarcts. Adding new hearing loss gives **HINTS-plus**. Applying HINTS to an episodic or chronic presentation is a common and dangerous error.
- **Bárány Society consensus definitions** now standardise the major diagnoses — BPPV, vestibular neuritis, Menière's disease, **vestibular migraine**, **persistent postural-perceptual dizziness (PPPD)** and **presbyvestibulopathy**. Two are particularly relevant here: **vestibular migraine** is now recognised as one of the commonest causes of episodic vertigo at all ages, and **PPPD** explains chronic non-spinning dizziness with unsteadiness worsened by upright posture, motion and complex visual environments — treated with vestibular rehabilitation, SSRIs and CBT, not with vestibular sedatives.
- **Presbyvestibulopathy** is a defined entity for age-related bilateral vestibular hypofunction with chronic imbalance and mild bilateral VOR deficits — it validates the textbook's "multisensory dizziness syndrome" but gives it operational criteria.
- **Video head impulse testing (vHIT)** now allows objective, quantitative bedside measurement of the VOR gain in all six semicircular canals and detects covert saccades missed by the naked eye.
- **Indian context**: dizziness is reported by **14.5% of Indian adults aged 60 and above (LASI wave-1)** and is a major driver of falls and activity restriction. The commonest treatable causes in an Indian geriatric clinic are **BPPV, orthostatic hypotension, anaemia (haemoglobin g/dL), drug effects, hypoglycaemia and vitamin B12 deficiency** — all diagnosable at the bedside or with basic tests.

Current Practice Update

Issue	Textbook position	Current position
Classification approach	Character (vertigo vs light-headedness) plus time course and associated symptoms	**Timing and triggers (ATTEST/TiTrATE)** — symptom quality is unreliable; classify as AVS, t-EVS, s-EVS or chronic
Distinguishing stroke from neuritis	Vertigo alone is not enough to diagnose posterior circulation TIA; needs other symptoms	**HINTS / HINTS-plus in acute vestibular syndrome**, more sensitive than early MRI; retains the principle but operationalises it
Head thrust test	Bedside test for vestibular hypofunction	Same, now quantifiable by **video head impulse testing (vHIT)** across all six canals
Chronic non-spinning dizziness	Attributed to anxiety plus underlying abnormality; multisensory dizziness syndrome	Defined as **PPPD** and **presbyvestibulopathy** with Bárány criteria; treated with vestibular rehabilitation, SSRIs and CBT
Episodic vertigo	Menière's, BPPV, vertebrobasilar ischaemia, migraine mentioned	**Vestibular migraine** now a leading and formally defined cause of spontaneous episodic vertigo

Exam One-Liners

- Vertigo = hallucination of movement; dizziness = lay term with many general medical causes.
- Never attribute dizziness in an older person to age alone.
- Dizziness in the elderly is usually multifactorial — central pathology plus multiple sensory deficits.
- Ask about timing and triggers, not "what kind of dizzy" (ATTEST/TiTrATE).
- Dix-Hallpike in every dizzy older patient — positional nystagmus is the most overlooked sign.
- Head impulse test: corrective saccades = ipsilateral vestibular hypofunction.
- HINTS is only for acute vestibular syndrome; normal head impulse + direction-changing nystagmus + skew = central.
- India (LASI): dizziness in 14.5% of those aged 60 and above.

Q13. Evaluation of dizziness / drop attacks

> The dizziness component overlaps with Q12; this answer therefore concentrates on the **practical evaluation algorithm** and on **drop attacks**, which are examined separately.

15–20 High-Yield Points (Textbook)

A. Drop Attacks — Definition and Concept

1. A **drop attack** is described as **sudden leg weakness causing a fall without loss of consciousness**. It is listed as one of the causes of falls in older persons, alongside accidents, syncope and dizziness/vertigo.
2. Drop attacks are **often attributed to vertebrobasilar insufficiency and are often precipitated by a change in head position**.
3. **Only a small proportion of older people who fall have truly had a drop attack.** The **underlying pathophysiology is poorly understood, and care should be taken to rule out other causes.**
4. **Vertebrobasilar insufficiency and mechanical compression of the vertebral arteries by cervical osteophytes when the head is turned are both poorly documented, probably over-diagnosed, and should not be used as the cause of a fall simply because nothing else can be found.** This is a high-yield examination statement.
5. The **essential distinction** is: **syncope = sudden loss of consciousness**; **drop attack = sudden fall with preserved consciousness**; **seizure = loss of consciousness with abnormal motor activity, often with incontinence and a post-ictal phase**.

B. Differential Diagnosis of Drop Attacks

6. **Vertebrobasilar ischaemia** and posterior circulation TIA — but the diagnosis of a posterior circulation TIA requires **one or more other symptoms (visual field cut, dysarthria, ataxia, limb weakness, which can be bilateral) in association with vertigo**; transient vertigo alone is not sufficient.
7. **Carotid sinus hypersensitivity** — a treatable cause of unexplained falls, often precipitated by head turning, in which the patient may deny loss of consciousness.
8. **Cardiac** — transient arrhythmia, heart block, aortic stenosis, causing near-syncope with a fall.
9. **Otolithic crisis of Tumarkin** in **Menière's disease** supplementary — sudden violent falls without loss of consciousness. Pathy notes that in **BPPV** too, attacks are very sudden and **may cause a drop to the ground**.
10. **Neurological** — atonic (astatic) seizure; **normal-pressure hydrocephalus**; cervical or lumbar spondylosis with cord or root compression; cerebellar disease; Parkinson's disease with freezing; **cataplexy** associated with narcolepsy supplementary; and **subclavian steal syndrome**.
11. **Musculoskeletal** — sudden knee buckling from quadriceps weakness or arthritis, which patients frequently describe as a drop attack.

C. Evaluation — Structured Approach

12. **Step 1: Characterise the event.** Was consciousness lost? Was there an aura, incontinence, tongue biting or a post-ictal phase? Could a **witness** confirm loss of consciousness? What is remembered immediately after? **Could the patient get up, and how long did it take?**
13. **Step 2: Establish the trigger.** Relationship to **change of posture, turning of the head, cough, micturition, a meal, or drug intake**. Note location, time of day, activity and whether alone.
14. **Step 3: Search for premonitory symptoms** — light-headedness, dizziness, vertigo, palpitations, chest pain, breathlessness, sudden focal neurological symptoms.
15. **Step 4: Vital signs and postural blood pressure** supine, sitting, and at 1 and 3 minutes standing; look for fever, tachypnoea, tachycardia and hypotension indicating acute illness.
16. **Step 5: Targeted bedside tests** — **carotid sinus massage for 5 seconds** under monitoring if the history suggests it; **Dix-Hallpike manoeuvre**; **head impulse test**; **Romberg** and eyes-closed gait testing; cardiovascular examination for arrhythmia and aortic stenosis.
17. **Step 6: Neurological and musculoskeletal examination** — mental status, focal signs, muscle weakness, rigidity or spasticity, peripheral neuropathy with posterior column signs, cerebellar tests (heel-shin, heel tapping), parkinsonian signs; and examine the feet and footwear.
18. **Step 7: Investigations, kept targeted. 12-lead ECG and lying/standing blood pressure are the minimum. Echocardiography** in all patients with a suggestive history and a systolic murmur or a delayed carotid upstroke. **Continuous cardiac monitoring** only where an arrhythmia can be temporally correlated with symptoms. **Non-invasive vascular studies** if anterior circulation TIA is suspected. **CT or MRI reserved** for a high suspicion of an intracranial lesion or seizure disorder. **EEG only** where there is a high degree of clinical suspicion of seizure. Basic bloods: haemoglobin g/dL, glucose, sodium and potassium mEq/L, creatinine mg/dL, calcium mg/dL.
19. **Step 8: In parallel, evaluate the dizziness itself** — character, time course and associated symptoms, with the full differential of general medical, neurological and otological causes, and screen with the CGA question "do you get dizzy, or does your head spin around?" followed by **postural blood pressure, Dix-Hallpike, haemoglobin and the Geriatric Depression Scale**.
20. **Step 9: Treat what you find** — pacemaker for symptomatic cardio-inhibitory carotid sinus hypersensitivity; Epley for BPPV; drug withdrawal and volume repletion for orthostatic hypotension; valve surgery for aortic stenosis with syncope; shunt for normal-pressure hydrocephalus; anticonvulsants for seizure; physiotherapy, strengthening and gait retraining for musculoskeletal causes; and **do not label the event vertebrobasilar insufficiency by exclusion**.

Latest Updates (2023–Present)

- The term **“drop attack” is being retired as a diagnosis** and treated as a **symptom description requiring a mechanism**. Current practice is to reclassify every drop attack as **syncopal (unrecognised transient loss of consciousness), vestibular (Tumarkin otolithic crisis, BPPV), neurological (atonic seizure, cataplexy, NPH, myelopathy) or musculoskeletal (knee buckling)**. **Vertebrobasilar insufficiency as a default explanation is explicitly discouraged.**
- **Unwitnessed falls with amnesia are now assumed to be syncopal until proved otherwise** in older people. **World Falls Guidelines 2022** class **suspected transient loss of consciousness** as an automatic **high-risk** criterion, and mandate cardiovascular assessment — cardiac history, auscultation, lying and standing blood pressure and a 12-lead ECG — in every faller, with further cardiac work-up reserved for suspected syncope or recurrent unexplained falls.
- **Implantable loop recorders** in older adults with recurrent unexplained falls detect clinically significant bradyarrhythmias in a meaningful proportion, supporting the view that many “drop attacks” are unrecognised arrhythmic syncope. Availability and cost limit this in India, where **prolonged Holter or patch monitoring** is the practical alternative.
- **Timing- and trigger-based classification (ATTEST/TiTrATE)** and the **HINTS examination in acute vestibular syndrome** have replaced symptom-quality-based evaluation of the dizzy patient, with HINTS by a trained examiner outperforming early MRI for posterior circulation stroke.
- **Tumarkin’s otolithic crisis** is now formally recognised within the **Bárány Society criteria for Menière’s disease** and is a specific indication for escalating treatment because of the injury risk.
- **Indian practice point**: in a resource-limited clinic, the highest-yield sequence in a patient with drop attacks is a **careful witness history, lying and standing blood pressure, 12-lead ECG, carotid sinus massage, Dix-Hallpike, haemoglobin and blood glucose** — almost all of which are free or very low cost, and which together will identify the mechanism in the majority.

Current Practice Update

Issue	Textbook position	Current position
Status of “drop attack”	A listed cause of falls, pathophysiology poorly understood, often attributed to vertebrobasilar insufficiency	**A description, not a diagnosis** — must be reclassified into a mechanism; vertebrobasilar insufficiency by exclusion is discouraged
Unwitnessed falls	Loss of consciousness often impossible to document	**Assume syncope until proved otherwise**; suspected TLoC is an automatic high-risk criterion
Rhythm monitoring	Only if arrhythmia can be temporally correlated	**Implantable loop recorder** in selected recurrent unexplained fallers; patch/prolonged Holter as the affordable alternative
Dizziness evaluation	Character, time course, associated symptoms	**Timing and triggers (ATTEST/TiTrATE)**, with **HINTS** restricted to acute vestibular syndrome

> **Exam One-Liners**
> - Drop attack = sudden fall from leg weakness WITHOUT loss of consciousness.
> - Only a small proportion of falls are true drop attacks; the mechanism is poorly understood.
> - Never diagnose vertebrobasilar insufficiency simply because nothing else is found.
> - Posterior circulation TIA needs another symptom besides vertigo (visual field cut, dysarthria, ataxia, limb weakness).
> - Tumarkin otolithic crisis = sudden violent fall in Menière’s disease without LOC.
> - Minimum work-up: witness history, lying/standing BP, 12-lead ECG, carotid sinus massage, Dix-Hallpike.
> - EEG only with high clinical suspicion of seizure; imaging only with high suspicion of an intracranial lesion.
> - Unwitnessed fall with amnesia in an older person = syncope until proved otherwise.

Q14. Vestibular rehabilitation

15–20 High-Yield Points (Textbook)

A. Principle — Cerebral Compensation

1. After an acute unilateral vestibular upset the patient experiences vertigo, but symptoms are usually **short-lived and resolve in 6–12 weeks** by processes collectively known as **cerebral compensation**. **Vestibular rehabilitation exists to drive and accelerate this compensation.**
2. Restoration of balance involves **reducing or abolishing the asymmetry in postural and ocular motor tone** and **recalibrating the gain of dynamic vestibular reflexes** to ensure symmetrical vestibulospinal and vestibulo-ocular reflex action during head and body movement.
3. Compensation depends on **brainstem, cerebellar and cortical structures together with sensory inputs including vision, somatosensory afferents and the remaining labyrinthine input**.
4. In the older patient **the plasticity of the CNS is compromised, so recovery is often slower and is rarely complete**. Contributing factors to failed compensation or intermittent decompensation include **cerebellar damage, impaired proprioception, visual impairment, mild cerebral dysfunction and psychological disorders**.
5. **Decompensation** can be triggered by an **intercurrent illness or a psychological upset such as bereavement**, and then presents as vague floating, rocking or depersonalisation rather than true rotational vertigo.

B. Cawthorne–Cooksey Exercises

6. **Physical exercise regimes were introduced as early as the mid-1940s (the Cawthorne–Cooksey exercises)** as a means of **expediting recovery from peripheral vestibular disorders**.
7. They are a **graduated series of exercises aimed at encouraging head and eye movements, which systematically provoke dizziness and facilitate vestibular compensation**. The therapeutic principle is **deliberate, controlled provocation of symptoms**, not avoidance.
8. **The exercises are not an endurance test.** In the older patient it is important to **modify the regime within the limits of the patient's physical abilities**.
9. Progression follows a standard order supplementary: **eye movements alone (up-down, side-to-side, focusing on a finger), then head movements with eyes open and then closed, then movements while sitting (shoulder shrugging, bending forward to pick objects), then standing (sit-to-stand with eyes open and closed, throwing a ball), then moving about (walking across the room with eyes open and closed, walking up and down a slope and steps, ball games, and activities requiring bending and stretching)**.

C. Evidence and Effects

10. **The passage of time has supported the efficacy of these exercises.**
11. **Successful vestibular rehabilitation improves activities of daily living and reduces fall risk** — the key statement linking this topic to the falls syllabus.
12. **Significant improvement has been shown in patients with peripheral vestibular dysfunction and also in patients with central balance disorders.**
13. **There is no evidence that age is a negative prognostic factor.** Older patients should therefore be offered vestibular rehabilitation on exactly the same basis as younger patients.
14. **"Customised" exercises tailored to the individual patient are equally effective** as the standard regime.
15. **Introduce vestibular rehabilitation and gait strategy exercises in the elderly early and emphatically** — this is one of the chapter's key points.

D. Relationship with Repositioning Manoeuvres

16. **Specific positional manoeuvres for benign positional vertigo, such as the Epley manoeuvre, form an important element of vestibular management** and are distinct from vestibular rehabilitation.
17. **A combination of canalith repositioning manoeuvre and vestibular rehabilitation improves benign positional vertigo in the elderly.** Although the **repositioning manoeuvre is the most effective treatment**, **vestibular rehabilitation can be added to improve results, particularly given the high recurrence of positional vertigo**.

E. Drugs and Vestibular Rehabilitation

18. **Vestibular sedatives such as cinnarizine are of value in managing acute vertigo but have a very limited role in managing chronic vestibular syndromes**, because they suppress the very asymmetry signal that drives compensation.
19. **Anti-emetics such as prochlorperazine should be avoided** because of the **rare but irreversible syndrome of extrapyramidal dysfunction** — a particularly important warning in older patients.
20. **Psychotropic drugs should be administered only for specific psychiatric indications, as such medication may interfere with compensatory mechanisms** for peripheral vestibular disorders.
21. **Surgery**: in the elderly the indications for otological surgery must be carefully weighed against the general medical state and the likely extent of postoperative recovery. **Destructive surgical procedures should not be considered until detailed neuro-otological investigations have been performed and medical management has been tried exhaustively**, and **the risk of persisting imbalance after vestibular destruction must be borne in mind**.

Latest Updates (2023–Present)

- The **updated Clinical Practice Guideline on Vestibular Rehabilitation for Peripheral Vestibular Hypofunction** (Academy of Neurologic Physical Therapy, APTA, 2022; a revision of the 2016 guideline) is now the reference standard and gives, for the first time, **explicit exercise dosing**.

- **Strong recommendation**: clinicians **should offer vestibular rehabilitation** to adults with unilateral and bilateral vestibular hypofunction who have impairments, activity limitations or participation restrictions, and **should offer supervised vestibular rehabilitation** rather than an unsupervised home programme alone.
- **Gaze stabilisation dosing**: at least **12 minutes daily, in 3 sessions, for acute or sub-acute unilateral vestibular hypofunction**; at least **20 minutes daily, in 3 to 5 sessions, for chronic unilateral hypofunction**; and a **higher total daily dose (typically 20–40 minutes) for bilateral vestibular hypofunction**, which also needs a longer progression.
- **Strong recommendation against**: do **not** use **voluntary saccadic or smooth-pursuit eye movements in isolation** (that is, without head movement) to promote gaze stability — the guideline judged harm to outweigh benefit. Retinal slip produced by **head movement** is the necessary error signal for VOR adaptation. This is an important update, because "eye exercises" are still widely prescribed.
- **Early initiation** of vestibular physical therapy, ideally **within 2 weeks of acute onset of unilateral vestibular hypofunction**, may optimise outcomes.
- **Balance and gait training** should accompany gaze stabilisation, using a variety of modalities: low-technology exercise, **virtual reality**, **optokinetic stimulation**, platform perturbations, **vibrotactile and augmented sensory feedback**, dual-task walking, obstacle negotiation, uneven surfaces and turning, and task-specific ADL practice (sit-to-stand, stairs, bed mobility, tight-space turning).
- The guideline gives **expanded recommendations on factors that modify outcome**, specifically **medications** (vestibular suppressants and psychotropics impair compensation) and **mild cognitive impairment** — which supports and updates the textbook's warning about drugs and about failed compensation in the older brain.
- **Persistent postural-perceptual dizziness (PPPD)** is now a defined Bárány Society entity treated with **vestibular rehabilitation plus SSRI/SNRI plus cognitive behavioural therapy**, not with vestibular sedatives — the modern equivalent of the "anxious elderly patient with chronic disequilibrium" described in the textbook.
- **Indian delivery**: formal vestibular physiotherapy services are scarce outside tertiary centres. Practical approaches are **therapist-taught, home-based customised Cawthorne–Cooksey and gaze stabilisation programmes with a written or illustrated schedule in the local language**, family-supervised practice, telerehabilitation, and integration with **NPHCE** geriatric clinics and district physiotherapy services. This is supported by the textbook's own finding that **customised exercises are equally effective** and that **age is not a negative prognostic factor**.

Current Practice Update

Issue	Textbook position	Current position
Exercise dose	Graduated exercises, modified to the patient's ability; not an endurance test	**Explicit dosing**: gaze stabilisation ≥12 min/day in 3 sessions (acute/subacute UVH); ≥20 min/day in 3–5 sessions (chronic UVH); 20–40 min/day (BVH)
Eye exercises	Head and eye movement exercises encouraged	**Strong recommendation against isolated saccadic or smooth-pursuit eye exercises without head movement**
Supervision	Customised exercises equally effective	**Supervised programmes recommended (strong evidence)**; customisation retained
Timing	Introduce early and emphatically	Quantified — **within 2 weeks of acute onset** may optimise outcomes
Modalities	Cawthorne–Cooksey exercises	Plus **virtual reality, optokinetic stimulation, vibrotactile and augmented sensory feedback, dual-task and gait training**
Drugs	Vestibular sedatives limited to acute vertigo; avoid prochlorperazine; psychotropics interfere with compensation	Unchanged and reinforced — medication effect is now a formal outcome-modifying factor in the guideline

Exam One-Liners

- Vestibular rehabilitation works by driving cerebral compensation — abolishing tone asymmetry and recalibrating VOR gain.
- Cawthorne–Cooksey exercises (mid-1940s): graduated head and eye movements that deliberately provoke dizziness.
- Progression: eyes → head → sitting → standing → moving about.
- Improves ADLs and reduces fall risk; effective in peripheral AND central balance disorders.
- Age is NOT a negative prognostic factor; customised exercises are equally effective.
- Dose (APTA 2022): ≥12 min/day acute UVH; ≥20 min/day chronic UVH; 20–40 min/day BVH.
- Do NOT prescribe isolated eye movements without head movement.
- Avoid vestibular sedatives in chronic vertigo; avoid prochlorperazine (extrapyramidal risk).
- Epley + vestibular rehabilitation together give the best outcome in elderly BPPV.

DELIRIUM / ACUTE CONFUSIONAL STATE IN THE ELDERLY

MD Geriatric Medicine — 15-Mark Exam Answers | Primary source: Pathy's Principles and Practice of Geriatric Medicine, 6th ed. (Ch. 61) with Kane's Essentials of Clinical Geriatrics (Ch. 6) | Updates to August 2026

RECURRING TOPIC ALERT. All five questions in this set are variations of a single core theme — **delirium**. Examiners repeat this topic because it is the commonest geriatric syndrome in hospital, the most under-diagnosed, and the most preventable. Master **one** framework (Predisposing + Precipitating → Screen → Treat cause → Non-pharmacologic bundle → Drugs only as last resort) and it will answer every version. Extra recall points are given at the end.

Q1. Aetiology, pathogenesis, differential diagnosis and management of acute confusional state in the elderly

18 High-Yield Points (Textbook)

1. **Definition:** Delirium (acute confusional state, "**acute brain failure**", altered mental status) is an **acute disturbance of attention, awareness and cognition**, developing over hours to days, **fluctuating** through the day, and caused by an underlying medical condition, drug or withdrawal.
2. **Key clinical features:** impaired consciousness, **global cognitive impairment of acute onset**, disorientation, **disturbed sleep–wake cycle**, altered psychomotor behaviour, illusions, visual hallucinations and paranoid delusions. It is typically **worse at night**, with lucid intervals in between.
3. **Rates (Pathy):** ICU **30–80%**; hospice care 26–44%; general medical and oncology wards **18–35%**; nursing home / post-acute care 16%. Surgical rates: cardiac and vascular surgery 29–50%, hepatobiliary and general surgery 17–24%, hip fracture repair 12–48%.
4. **Phenotypes:** classically **hyperactive, hypoactive and mixed**. Newer recognised phenotypes are ICU delirium (sedative, septic, hypoxic, metabolic subtypes), **postoperative delirium (POD)** vs **postoperative cognitive dysfunction (POCD)**, emergence delirium, delirium at end of life (replaces "terminal restlessness"), **subsyndromal delirium**, persistent/chronic delirium and **delirium superimposed on dementia (DSD)**.
5. **DSD is the hardest phenotype:** worse outcomes, longer stay, hardest to diagnose, and takes longer to resolve than other phenotypes.
6. **Pathogenesis — Systems Integration Failure Hypothesis (SIFH):** several interacting processes — **neuronal ageing, oxidative stress, neuroendocrine changes, diurnal dysregulation and neuroinflammation** — damage the neurotransmitter systems and break down **neuronal network connectivity**. This combination produces the outward picture of acute brain failure.
7. **Neurotransmitters involved:** dopaminergic, adrenergic, **muscarinic (cholinergic)**, histaminic and serotonergic pathways are all disturbed, but to an unmeasurable degree in any given patient.
8. **Why the pathogenesis matters in exams:** (a) it explains the very wide spectrum of presentations, and (b) it explains **why antipsychotic trials keep failing** — these drugs hit several receptors at once to unpredictable degrees, and correcting one pathway may disturb another.
9. **Vulnerability model:** a patient with many **predisposing** factors is highly vulnerable, so only a **tiny precipitant** (e.g. a single dose of oral diphenhydramine for insomnia) is needed to tip them into delirium. Many factors are both predisposing and precipitating.
10. **Aetiology — the Pathy mnemonic "DELIRIUM IS VERY PAINFUL"** covers the entire differential of causes and contributors (table below).
11. **Deliriogenic drugs (Pathy list):** antipsychotics, **benzodiazepines** (especially intravenous — paradoxical reactions), corticosteroids, **H2-receptor antagonists in renal impairment**, narcotics (especially **pethidine/meperidine** and IV opioids), muscle relaxants, **antimuscarinics for overactive bladder**, first-generation antihistamines, and non-benzodiazepine benzodiazepine-receptor agonist hypnotics such as **zolpidem**.
12. **Differential diagnosis:** dementia, delirium superimposed on dementia, depression (pseudodementia), late-onset psychosis or mania, **non-convulsive status epilepticus**, Wernicke's encephalopathy, and drug/alcohol/benzodiazepine withdrawal.
13. **Best discriminator from dementia:** focus on the **core criteria — attention, awareness and level of consciousness — not on the behaviour**. Behavioural symptoms are shared; inattention with acute fluctuating onset is not.
14. **Diagnosis: DSM-5 and ICD-10 remain the gold standard**. DSM-5 requires disturbed attention/awareness/cognition, acute onset, fluctuation, not better explained by a neurocognitive disorder, not in coma, and evidence of a medical cause, substance or multiple aetiologies. ICD-10 adds detail on perceptual distortion, psychomotor and sleep–wake disturbance.
15. **Bedside screening: CAM** (sensitivity 94%, specificity 89%), 3D-CAM, CAM-ICU, 4AT, bCAM. Screen **all hospitalised older adults at least daily**.
16. **Targeted investigation:** laboratory and imaging tests are guided by the clinical assessment. **Neuroimaging, lumbar puncture and EEG are NOT required in every patient.** CT/MRI only for new focal deficit, suspected stroke, fall or head trauma, unexplained reduced consciousness, or fever with suspected viral encephalitis; LP if meningitis/encephalitis suspected; EEG if seizure suspected.
17. **Management — four sequential steps:** (i) identify and treat every predisposing/precipitating factor; (ii) institute **multicomponent non-pharmacologic care as the mainstay**; (iii) use the **T-A-DA method** — **T**olerate behaviours that are not dangerous, **A**nticipate needs, **D**on't **A**gitate; (iv) drugs only if severe agitation endangers the patient or others.

18. **Drug rules: no drug is approved by the FDA or EMA for delirium. Benzodiazepines must not be used** (they precipitate and prolong delirium) — the **only two exceptions are benzodiazepine withdrawal and alcohol withdrawal**. Antipsychotics carry a **black-box warning** for increased mortality in dementia; AGS/ACS advise **lowest dose, shortest duration, only after non-pharmacologic measures fail**; PADIS advises against routine use in the ICU; and their use in delirium is listed under **Choosing Wisely — "Things We Do for No Reason"**.

Table 1. Causes and contributors — "DELIRIUM IS VERY PAINFUL" (Pathy Table 61.1)

	Factor	Key exam point
D	Drugs & drug withdrawal	Commonest reversible cause; high index of suspicion in the vulnerable brain
E	Eyes, Ears	Visual/hearing impairment — mainly *predisposing*
L	Low O_2 / low perfusion	MI, pulmonary embolism, stroke, cardiac or pulmonary failure, anaemia, anoxia
I	Infection	Even *mild* infection can precipitate delirium in the vulnerable; do not over-treat behaviour as "infection"
R	Retention of urine	**Cystocerebral syndrome** — sudden delirium, muteness, restlessness
I	Indwelling urinary catheter	Infection risk + a "one-point restraint" that impairs mobility
U	Underhydration	**BUN : creatinine ratio > 17:1** is associated with delirium and delirium risk
M	Metabolic	Hypercalcaemia, hypokalaemia, hyponatraemia, hypernatraemia, hyperammonaemia
I	Immobility & inactivity	Early mobilisation reduces delirium rate and duration
S	Sleep problems	Sleep deprivation, sleep–wake reversal, sleep apnoea
V	Vascular changes / volume loss on CT-MRI	Predisposing when interpreted with the history
E	Endocrinopathies	Thyroid and adrenal disorders, hyper- and **hypoglycaemia**
R	Restraints	Wrist restraints, IV lines, catheters, pneumatic compression devices, casts
Y	ps**Y**chiatric disorders	Anxiety, depression, schizophrenia, bipolar disorder — predisposing
P	Pain	Uncontrolled pain precipitates; but do **not** use opioids for behaviour. Regular paracetamol reduces opioid need
A	Attachments	IV lines, telemetry — anything that tethers or annoys the patient
I	Impaction	Severe constipation / faecal impaction
N	Neurological injury	Subdural haematoma, intracerebral bleed, head trauma, stroke, tumour, encephalitis, meningitis, seizures
F	Frailty	Major predisposing factor
U	Undernutrition	Contributor; alters protein binding of drugs
L	Lights, loud noises	Environmental; sensory input is misperceived

Table 2. Delirium vs Dementia (Kane's, Table 6-3)

Feature	Delirium	Dementia
Onset / course	Hours to days; days to weeks duration; marked **fluctuation**	Gradual over months to years; slowly progressive
Consciousness	**Altered and changing**	Unchanged until very late
Attention	**Short attention span (core deficit)**	Attention span preserved
Psychomotor	Marked change — lethargic or agitated	Baseline activity maintained
Speech	Often **incoherent**	Word-finding difficulty, aphasia
Hallucinations	Common, usually **visual**; illusions	Unusual until late (except Lewy body dementia)
Sleep–wake cycle	Disrupted	Sleep disturbed, but cycle less affected
CAM screen	Positive	Negative
Acute illness / drug	Usually evident	No evidence of acute illness

Latest Updates (2023–Present)

- **APA Practice Guideline for the Prevention and Treatment of Delirium (Sept 2025)** — the first full revision since 1999 (brief update 2004). It shifts the emphasis firmly to **prevention**, mandates a thorough medication and risk-factor history, restricts restraints to situations of **"imminent harm"**, and adds structured guidance on **transitions of care** (medication reconciliation, delirium follow-up, patient and caregiver psychoeducation).
- **NICE CG103 (updated January 2023)** — new recommendations on assessing and diagnosing delirium; the **4AT** is now the NICE- and SIGN-endorsed bedside tool.
- **SCCM PADIS Focused Update 2025** (Crit Care Med 2025) — suggests **dexmedetomidine over propofol** when light sedation and/or delirium reduction are priorities; suggests **enhanced mobilisation/rehabilitation** and **melatonin**; explicitly suggests **not** using haloperidol, atypical antipsychotics, dexmedetomidine, statins or ketamine **to prevent** delirium; and could issue **no recommendation for or against antipsychotics for treatment** because effects on outcomes are minimal.

- **AGS Beers Criteria 2023** — anticholinergics are now split into antihistamines, CNS antiparkinsonian agents and GI antispasmodics, with explicit warning that **combined anticholinergic burden** raises the risk of delirium, falls, cognitive decline and possibly dementia. The companion **AGS Beers Alternatives List (2025)** supplies safer substitutes for drugs used in insomnia, anxiety, delirium and chronic pain.
- **Epidemiology refresh:** a 2025 systematic review and meta-analysis of 35 studies (12,097 medically hospitalised older patients) found a **pooled prevalence of 23.6%** and a **pooled incidence of 13.5%**, with frailty, physical restraints, previous falls, illness severity, cognitive impairment and older age as the leading risk factors — validating the Pathy risk-factor table.
- **Ormseth et al. (JAMA Network Open 2023)** — large systematic review confirming that predisposing and precipitating factors are almost always **multifactorial** rather than single-cause.
- **Biomarkers:** genetic and proteomic risk-factor studies (Nature Aging 2026) are beginning to define a biological risk signature for delirium — not yet clinically usable.
- **Indian data:** tertiary-care studies continue to show marked under-recognition — delirium accounts for only about 0.4% of total inpatients when identified through psychiatry referral, but Indian ICU studies using CAM-ICU report **~30% prevalence in non-ventilated elderly ICU patients, with hypoactive delirium commonest (~45%)**. The gap between referral-based and screening-based figures is itself an exam point.

Current Practice Update

Issue	Pathy 6th edition (textbook)	Current practice (2023–2026)
Guideline anchor	AGS/ACS 2015 postoperative delirium statement; SCCM PADIS 2018	**APA 2025 delirium guideline** + **PADIS Focused Update 2025** + NICE CG103 (2023)
Antipsychotics for treatment	Evidence does not support use; reserve for severe agitation	Position hardened — PADIS 2025 could issue **no recommendation either way**; APA 2025 restricts to imminent harm
Antipsychotics for prevention	Not supported	**Explicitly recommended against**, along with statins and ketamine
Sedative of choice in ICU	Propofol or dexmedetomidine over benzodiazepines	**Dexmedetomidine preferred over propofol** when light sedation / delirium reduction are the priority (watch bradycardia)
Melatonin	Not recommended	**Conditionally suggested** in ICU patients (PADIS 2025)
Screening tool emphasis	CAM family emphasised	**4AT** endorsed by NICE and SIGN for rapid ward and ED use
Restraints	Avoid; no evidence of benefit	Restricted to **"imminent harm"** only (APA 2025)
Discharge	Comprehensive discharge planning	Formal **transition-of-care** block — medication reconciliation, delirium follow-up, caregiver education

Exam One-Liners — Q1

- Delirium = **acute brain failure**; the core deficit is **inattention**, not agitation.
- Pathogenesis = **Systems Integration Failure Hypothesis** (Maldonado).
- Mnemonic = **DELIRIUM IS VERY PAINFUL**.
- Cystocerebral syndrome = delirium from **urinary retention**.
- **BUN : creatinine > 17:1** = dehydration-associated delirium risk.
- CAM: sensitivity **94%**, specificity **89%**.
- Gold standard diagnosis = **DSM-5 / ICD-10**; CAM/4AT are screens.
- Mainstay of treatment = **multicomponent non-pharmacologic care**; behaviour tool = **T-A-DA**.
- **No drug is FDA/EMA approved** for delirium; benzodiazepines only for alcohol or benzodiazepine withdrawal.
- Up to **30–40% of delirium is preventable**.

Q2. A 75-year-old diabetic, hypertensive smoker presents with cough, fever and altered sensorium; he is incoherent and abusive — differential diagnosis, trigger factors, evaluation and ward management

> **One-line formulation:** This is **hyperactive delirium** in a multi-morbid older adult, most probably precipitated by **community-acquired pneumonia**. The abusive, incoherent behaviour is a **symptom of acute brain failure, not a psychiatric illness** — the task is to find and treat the precipitant, not to sedate the patient.

18 High-Yield Points

A. Recognising the syndrome

1. Acute onset over hours–days, incoherent speech, abusive and restless behaviour with fever indicates **delirium, hyperactive subtype**, until proven otherwise. Confirm with **CAM or 4AT** at the bedside: acute onset with fluctuation **plus inattention**, plus either disorganised thinking or altered consciousness.
2. Test inattention immediately — **months of the year backwards**, days of the week backwards, or digit span. Do not "make excuses" for a positive item because the patient is old, febrile or sleepless.
3. Remember the **hypoactive** phase may follow; delirium waxes and wanes, so a single normal assessment does not exclude it.

B. Differential diagnosis of altered sensorium in this patient

4. **Infective:** community-acquired pneumonia with delirium (most likely), **sepsis / septic encephalopathy**, aspiration pneumonia, **pulmonary tuberculosis or miliary TB** (important in India), influenza/COVID, meningitis, viral encephalitis, brain abscess, urinary sepsis with an incidental cough.
5. **Respiratory / hypoxic: hypoxaemia** and **hypercapnic encephalopathy** (smoker → COPD), pulmonary embolism, acute pulmonary oedema.
6. **Metabolic / endocrine: hypoglycaemia** (must be excluded within seconds), diabetic ketoacidosis, hyperosmolar hyperglycaemic state, **hyponatraemia** (SIADH accompanying pneumonia, or thiazide-induced), hypercalcaemia, uraemia, hepatic encephalopathy, thyroid disease.
7. **Neurological:** ischaemic stroke or intracerebral haemorrhage (hypertensive smoker), **subdural haematoma** after an unwitnessed fall, **non-convulsive status epilepticus**, hypertensive encephalopathy, **Wernicke's encephalopathy**.
8. **Drug-related:** anticholinergics, first-generation antihistamines in cough syrups, sedative-hypnotics, opioids, corticosteroids for wheeze, **fluoroquinolone neurotoxicity**, H2-blockers in renal impairment.
9. **Withdrawal states: alcohol withdrawal** (abusive behaviour, tremor, autonomic overactivity), benzodiazepine withdrawal, and **nicotine withdrawal** in a smoker admitted to a smoke-free ward.
10. **Simple bedside causes often missed: urinary retention** (cystocerebral syndrome), **faecal impaction**, uncontrolled pain.
11. **Psychiatric mimics (least likely):** late-onset psychosis, mania, agitated depression, and **delirium superimposed on dementia**. Abrupt onset with fever and inattention argues strongly against a primary psychiatric cause.

C. Trigger factors in this specific patient

12. Map him onto **DELIRIUM IS VERY PAINFUL**: **Infection** (pneumonia), **Low O_2** (smoker with hypoxaemia), **Endocrinopathy** (diabetes — hyper- and hypoglycaemia), **Drugs**, **Underhydration**, **Vascular change** (hypertension, smoking), plus hospital-generated triggers — **Restraints, Attachments, Indwelling catheter, Immobility, Sleep disruption, Lights and noise, Pain**.
13. Predisposing (non-modifiable) factors here: **age ≥ 75**, likely undetected cognitive impairment, frailty, sensory impairment, multimorbidity and polypharmacy. These make him so vulnerable that even a mild pneumonia can precipitate florid delirium.

D. Evaluation

14. **Within the first five minutes:** airway, breathing, circulation; **SpO_2**, respiratory rate, temperature, blood pressure, pulse; **capillary blood glucose**; GCS and RASS; pupils and focal neurological signs; look for neck stiffness; palpate the bladder; examine the perineum/rectum for impaction. Give **thiamine 100 mg IV before glucose** if alcohol use or malnutrition is suspected.
15. **Collateral history is mandatory** — baseline cognition and function, exact time course, complete drug and alcohol history, recent falls, and any drug newly started or stopped.
16. **Laboratory (Indian conventional units):** complete blood count with differential; blood glucose (mg/dL); serum sodium and potassium (mEq/L); calcium (mg/dL); blood urea and creatinine (mg/dL) with **BUN:creatinine ratio**; liver function tests; **arterial blood gas**; CRP or procalcitonin; blood cultures × 2; sputum Gram stain, culture, AFB and **CBNAAT**; urine routine and culture; HbA1c; serum TSH; vitamin B12 if cognition was already impaired; ECG (baseline **QTc before any antipsychotic**); chest radiograph.
17. **Targeted imaging only:** non-contrast CT head if there are new focal signs, suspected stroke, a fall or head injury, unexplained reduced consciousness, or fever with suspected encephalitis. **Lumbar puncture** if meningitis or encephalitis is suspected; **EEG** if non-convulsive seizures are suspected. Routine CT for every confused patient is not indicated.
18. Grade pneumonia severity (**CURB-65**) and screen for sepsis (qSOFA / NEWS2) to decide ward versus HDU care.

E. Ward management

Domain	Action

Treat the precipitant	Empirical antibiotics for CAP per severity and local antibiogram, started **within 1 hour if sepsis is present**; oxygen to target saturation (88–92% if COPD is suspected, otherwise 94–98%); chest physiotherapy; nebulised bronchodilators
Fluids & glucose	Correct dehydration and electrolytes; treat hyperglycaemia with **basal-bolus insulin, avoiding sliding scale alone**; target roughly **140–180 mg/dL**; **hypoglycaemia is more dangerous than modest hyperglycaemia** in delirium
Medication review	Stop every deliriogenic drug; apply **Beers / STOPP-START**; avoid anticholinergics, sedative-hypnotics, pethidine and H2-blockers; regular paracetamol for pain to reduce opioid load
Non-pharmacologic bundle *(the mainstay)*	Repeated **reorientation**; clock, calendar and familiar objects; **spectacles and hearing aids** restored; well-lit room by day, dark and quiet at night; **non-drug sleep protocol**; **early mobilisation three times daily**; oral hydration and feeding assistance; remove tethers — catheter, IV line, telemetry — as early as possible
Behaviour	**T-A-DA:** Tolerate non-dangerous behaviour, Anticipate needs (toilet, thirst, pain, position), Don't Agitate. Give the hands something to do (folding cloths) instead of pulling lines. **Avoid physical restraints** — they worsen delirium
Family	Keep a relative at the bedside for reassurance, orientation, feeding and mobilising. Explain that **fluctuation is expected** and that apparent improvement by day does not mean recovery. In Indian wards the attendant culture is a major asset — use it deliberately
Drugs (last resort)	Only if the patient is **severely agitated or distressed and dangerous to himself or others**, and only after non-pharmacologic measures have failed. **Haloperidol 0.25–0.5 mg** orally or IM, lowest dose and shortest duration, with ECG monitoring for **QTc**; avoid in Parkinson's disease and Lewy body dementia (use quetiapine). **Do not use benzodiazepines** unless this is alcohol or benzodiazepine withdrawal
Nicotine	Consider **nicotine replacement therapy** — withdrawal is a genuine, easily missed precipitant in an admitted smoker
Complication prevention	Aspiration precautions and swallow assessment; VTE prophylaxis; **pressure-injury prevention**; falls precautions with low bed and floor mats rather than restraints; bowel and bladder care
Monitoring & discharge	**Daily CAM/4AT screening**; document trajectory; medication reconciliation at discharge; cognitive reassessment at 4–8 weeks because delirium may unmask or accelerate dementia

Latest Updates (2023–Present)

- **APA 2025** guideline explicitly links delirium management to **medication history and deprescribing** as a first-line intervention, and confines restraints to situations of **imminent harm** — directly relevant to the "abusive" patient in this vignette.
- **NICE CG103 (2023)** names **benzodiazepines, opioids and anticholinergics** as the commonest pharmacological precipitants and recommends the **4AT** for rapid ward assessment.
- **AGS Beers 2023 / Alternatives List 2025** emphasise **cumulative anticholinergic burden** and offer non-drug alternatives for insomnia and agitation — useful when the ward wants "something for sleep".
- **Non-pharmacologic prevention remains the only intervention with consistent benefit**; a 2025 meta-analysis of **family involvement** in critically ill patients supports formally recruiting relatives into the delirium care plan.
- **Antipsychotic caution reinforced:** the **AID-ICU trial** and the **PADIS 2025 focused update** together show haloperidol has minimal or no effect on length of stay or duration of delirium — it is a behavioural rescue, not a treatment.
- **Indian relevance:** in a febrile elderly patient with cough and confusion, **CBNAAT for tuberculosis** should be sent early; TB remains a leading cause of subacute confusion with fever in Indian older adults and is easily missed when pneumonia is assumed.

Current Practice Update

Issue	Textbook position	Current practice
Sedating the abusive patient	Antipsychotic in lowest dose only if dangerous	Same, but with an explicit **"imminent harm"** threshold, mandatory **baseline ECG** and a documented stop date
Glycaemic target	Individualised control	Ward target roughly **140–180 mg/dL**; **avoid sliding-scale-only** insulin; hypoglycaemia treated as a delirium precipitant
Routine CT head	Not required in all	Unchanged and reinforced — targeted imaging only
Family role	Encouraged	A **formal, evidence-supported component** of the care bundle
Tuberculosis	Not emphasised	In India, **CBNAAT early** in any elderly patient with fever, cough and confusion

Exam One-Liners — Q2

- Abusive + incoherent + fever = **hyperactive delirium**, not psychosis.
- **Check glucose before anything else**; thiamine before glucose if alcohol is possible.
- Commonest precipitants in this patient: **infection, hypoxia, drugs, dehydration, retention**.
- CURB-65 for pneumonia severity; qSOFA/NEWS2 for sepsis.
- **Restraints worsen delirium** — low bed and a bedside relative are safer.
- Haloperidol **0.25–0.5 mg** is a rescue, not a treatment; check QTc.

- Don't forget **nicotine withdrawal** in an admitted smoker.
- Reassess cognition **4–8 weeks** after discharge.

Q3. A 65-year-old diabetic with chronic kidney disease presents with altered sensorium, fever, leucocytosis and hypotension — how will you evaluate and manage?

> **One-line formulation:** Fever + leucocytosis + hypotension + altered sensorium = **sepsis / septic shock with sepsis-associated delirium**, on a background of **diabetes and CKD** which both predispose to infection and to metabolic and drug-induced encephalopathy. This is a **time-critical emergency**: the altered sensorium is itself an organ-failure marker (SOFA/qSOFA).

18 High-Yield Points

A. Frame the problem

1. **Sepsis-3:** life-threatening organ dysfunction from a dysregulated host response to infection. **Altered mentation is one of the three qSOFA items** (altered mentation, systolic BP ≤ 100 mmHg, respiratory rate ≥ 22/min). **Septic shock** = vasopressor requirement to keep MAP ≥ 65 mmHg plus lactate > 2 mmol/L after adequate fluids.
2. In older adults, **delirium and falls may be the only presenting features of sepsis**; fever may be blunted or absent, and hypothermia carries a worse prognosis.
3. Delirium here is **multifactorial**: infection + hypoperfusion (Low O_2/low perfusion) + metabolic derangement + drug accumulation + ICU environment — the whole left column of **DELIRIUM IS VERY PAINFUL** operating at once.

B. Likely sources of sepsis in a diabetic with CKD

4. **Urinary tract:** acute pyelonephritis, **emphysematous pyelonephritis or cystitis**, perinephric abscess, obstructed infected system, catheter-associated UTI. **Diabetes plus CKD makes urosepsis the single commonest source.**
5. **Skin and soft tissue: diabetic foot infection**, cellulitis, necrotising fasciitis, pressure injury.
6. **Respiratory:** community-acquired or aspiration pneumonia, **tuberculosis**.
7. **Vascular access:** haemodialysis catheter-related bloodstream infection, arteriovenous graft infection, peritoneal dialysis peritonitis if on PD.
8. **Intra-abdominal:** cholangitis, emphysematous cholecystitis, diverticulitis, **rhino-orbito-cerebral mucormycosis** in poorly controlled diabetes.

C. Non-septic contributors to the altered sensorium (must be excluded in parallel)

9. **Uraemic encephalopathy**, metabolic acidosis, **hyperkalaemia**, **hyponatraemia**, hypocalcaemia, hyperphosphataemia.
10. **Hypoglycaemia** — CKD reduces renal insulin clearance and gluconeogenesis, so sulfonylureas and insulin accumulate; this is a classic and lethal miss.
11. **Hyperosmolar hyperglycaemic state** or euglycaemic ketoacidosis if on an SGLT2 inhibitor.
12. **Drug accumulation in CKD: cefepime and carbapenem neurotoxicity**, morphine metabolites, gabapentin/pregabalin, aciclovir, H2-receptor antagonists, metformin-associated lactic acidosis.
13. **Structural / other:** stroke, intracerebral bleed (uraemic platelet dysfunction, anticoagulation), **dialysis disequilibrium syndrome**, non-convulsive status epilepticus, Wernicke's encephalopathy.

D. Evaluation

14. **Immediate bedside:** ABC, airway protection if GCS is falling; large-bore access; **capillary glucose**; continuous SpO_2, BP, ECG; temperature; urine output via measurement (avoid an unnecessary indwelling catheter, but it is justified in shock); GCS and RASS; **full skin, foot, perineal and dialysis-access examination**; fundus and sinus examination if mucormycosis is possible.
15. **Investigations: serum lactate** (repeat if raised); **two sets of blood cultures before antibiotics** (do not delay antibiotics for them); complete blood count with differential; blood glucose (mg/dL); urea and creatinine (mg/dL) with baseline eGFR for comparison; sodium and potassium (mEq/L); calcium (mg/dL), phosphate, magnesium; arterial blood gas with anion gap; liver function tests; coagulation profile; CRP/procalcitonin; urine routine, microscopy and culture; sputum culture and CBNAAT; pus/wound culture; HbA1c; ECG; chest radiograph; **ultrasound KUB** for hydronephrosis, gas or abscess, with contrast-enhanced CT only when the benefit outweighs contrast risk. Add CT head, EEG or lumbar puncture only if focal signs, seizures or meningism are present.

E. Management

16. **Hour-1 sepsis bundle:** measure lactate, take blood cultures, give **broad-spectrum antibiotics within the first hour**, begin fluid resuscitation for hypotension or lactate ≥ 4 mmol/L, and start **noradrenaline** as the first-line vasopressor to keep **MAP ≥ 65 mmHg**. Add vasopressin if noradrenaline requirement rises; consider hydrocortisone in refractory shock.
17. **Fluids in CKD need judgement, not a fixed recipe.** The 30 mL/kg crystalloid figure is a starting point; in oligo-anuric CKD, established heart failure or dialysis dependence, give **smaller aliquots (250–500 mL) with reassessment after each** using passive leg raise, lactate clearance, capillary refill and lung ultrasound. Prefer **balanced crystalloids**; avoid starches; watch for hyperkalaemia with potassium-containing fluids.
18. **Antibiotics and the kidney:** give a **full loading dose**, then adjust the maintenance dose to renal function; avoid aminoglycosides and other nephrotoxins where possible; stop metformin, NSAIDs, ACE inhibitors/ARBs and SGLT2 inhibitors during the acute illness; de-escalate on culture results. Achieve **source control** within 6–12 hours — drain the abscess, relieve obstruction with a nephrostomy or stent, debride the foot, or remove the infected dialysis catheter.

F. Continuing ward/ICU care

Domain	Action

Glycaemia	Insulin infusion or basal-bolus; target roughly **140–180 mg/dL**; hourly monitoring during infusion; **stop sulfonylureas** (prolonged hypoglycaemia in CKD)
Renal support	Watch for AKI on CKD. RRT indications — **A**cidosis, **E**lectrolytes (refractory hyperkalaemia), **I**ntoxication, **O**verload (fluid), **U**raemic complications (encephalopathy, pericarditis). Continuous modalities are preferred in haemodynamic instability
Delirium care	**ABCDEF bundle** — Assess/prevent/manage pain; Both spontaneous awakening and breathing trials; Choice of sedation; Delirium assess, prevent and manage; Early mobility; Family engagement. Screen with **CAM-ICU** (or 4AT on the ward)
Sedation	Avoid benzodiazepines; if sedation is needed, **dexmedetomidine** is preferred when light sedation and delirium reduction are priorities
Drugs to stop	All deliriogenic and renally accumulating drugs — opioids (especially morphine), gabapentinoids, H2-blockers, anticholinergics; recheck antibiotic doses daily as renal function changes (**cefepime neurotoxicity** presents exactly like this)
Supportive	VTE prophylaxis, stress-ulcer prophylaxis where indicated, nutrition (protein without overloading urea), pressure-injury prevention, early mobilisation, bowel and bladder care, correction of anaemia
Goals of care	Assess pre-morbid frailty and function with **CGA**; discuss ceiling of care, dialysis initiation and resuscitation status early with family — frailty predicts outcome better than age

Latest Updates (2023–Present)

- **Surviving Sepsis Campaign 2026 guidelines** (Crit Care Med, April 2026) — the first full update since 2021, based on evidence to June 2025. Notable directions: **narrower empirical cover** (recommendation against anti-anaerobic agents in patients at low risk of anaerobic infection), acceptance in **low-resource settings that a single set of blood cultures** gives similar yield to multiple sets, stronger emphasis on taking cultures **before** antimicrobials (sensitivity falls by roughly a third within about 70 minutes of the first dose), and a new block on **post-sepsis recovery** — shared decision-making, structured discharge information and follow-up for new long-term impairments.
- **Post-sepsis syndrome** is now formally part of sepsis care: survivors need follow-up for new physical, cognitive and psychological impairment — especially relevant because delirium during sepsis predicts long-term cognitive decline.
- **PADIS Focused Update 2025:** dexmedetomidine over propofol for light sedation; enhanced mobilisation/rehabilitation over usual care; melatonin suggested; **no antipsychotic recommended for prevention or treatment**.
- **KDIGO-aligned practice** continues to stress full loading dose then renal dose adjustment, avoidance of nephrotoxins, and early nephrology involvement rather than fixed creatinine thresholds for RRT.
- **Indian context:** emphysematous pyelonephritis, diabetic foot sepsis, tuberculosis and mucormycosis are disproportionately common causes of septic shock in Indian diabetics. Access to continuous RRT is limited outside tertiary centres; the **Pradhan Mantri National Dialysis Programme** and **AB-PMJAY** cover much of the dialysis cost burden and should be mentioned in the social-care part of the answer.

Current Practice Update

Issue	Textbook / earlier position	Current practice (2023–2026)
Fluid resuscitation	Fixed 30 mL/kg crystalloid bolus	**Individualised, dynamically assessed** boluses — particularly in CKD and heart failure
Empirical antibiotics	Broadest possible cover	Broad but **targeted**; SSC 2026 advises against routine anti-anaerobic cover at low anaerobic risk; daily de-escalation review
Blood cultures	Two or more sets always	Still preferred, but **a single set is acceptable in low-resource settings**; taking them **before** antibiotics matters more than the number
Sedation choice	Propofol or dexmedetomidine over benzodiazepines	**Dexmedetomidine preferred over propofol** for light sedation and delirium reduction
Antipsychotics in ICU delirium	Recommended against routinely (PADIS 2018)	**No recommendation for or against treatment**; explicitly recommended **against for prevention**
After discharge	Little guidance	Formal **post-sepsis follow-up** for cognitive, physical and psychological sequelae

Exam One-Liners — Q3

- **Altered sensorium is an organ-failure sign** in sepsis (qSOFA item).
- Commonest source in a diabetic with CKD = **urinary tract**; think emphysematous pyelonephritis.
- **Cultures before antibiotics; antibiotics within 1 hour.**
- Noradrenaline first-line; target **MAP ≥ 65 mmHg**.
- In CKD — **load fully, then dose-adjust**; think **cefepime neurotoxicity**.
- **Source control within 6–12 hours.**
- RRT indications = **AEIOU**.
- Glucose target roughly **140–180 mg/dL**; stop sulfonylureas.
- Delirium care = **ABCDEF bundle** + CAM-ICU screening.
- Survivors need **post-sepsis follow-up** for cognitive decline.

Q4. Assessment of delirium

18 High-Yield Points

A. Principles

1. Assessment has **two distinct steps**: **screening** (a rapid bedside tool that flags possible delirium) and **diagnosis** (a full clinical evaluation against DSM-5 or ICD-10 criteria). A positive screen is not a diagnosis.
2. **Why delirium is missed:** the cognitive baseline is often unknown; there may be no reliable informant; symptoms **wax and wane**, so one assessment can look completely normal hours later; and **hypoactive delirium** is quiet and easily mistaken for tiredness, depression or "just being old".
3. **Anchor the assessment on the core features — attention, awareness and level of consciousness — not on behaviour.** This is the single most useful strategy when dementia coexists or when no informant is available.

B. Diagnostic criteria (gold standard)

4. **DSM-5:** (i) disturbance of **attention and awareness**; (ii) develops over a **short period**, a change from baseline, with **fluctuation** during the day; (iii) an additional disturbance in **cognition**; (iv) not better explained by a pre-existing, established or evolving neurocognitive disorder and not occurring in **coma**; (v) evidence of an underlying **medical condition, substance intoxication or withdrawal, or multiple aetiologies**.
5. **ICD-10** criteria are similar but add detail on **perceptual distortion (illusions, hallucinations), psychomotor disturbance, sleep–wake cycle disruption and emotional disturbance**.
6. **Kane's practical three questions** for any confused older patient: was the onset **acute** (hours to days)? Are there **physical factors** (illness, drugs, sensory deprivation)? Are there **psychological factors** (depression, psychosis) contributing?

C. Screening instruments (Pathy list)

7. **Confusion Assessment Method (CAM)** — the most widely used. Four features: **(i) acute onset and fluctuating course; (ii) inattention; (iii) disorganised thinking; (iv) altered level of consciousness.** Delirium is screened positive if **(i) AND (ii)** are present, **PLUS either (iii) OR (iv)**. **Sensitivity 94%, specificity 89%**, with high inter-rater reliability.
8. **Testing inattention at the bedside:** months of the year backwards, days of the week backwards, digit span 3 or 4 backwards; or simply observe that the patient is distracted or cannot follow the conversation.
9. **Testing disorganised thinking:** orientation to time and place, and observing an illogical flow of ideas.
10. **3D-CAM** — a structured **3-minute** interview with scripted questions. In patients **with dementia**: sensitivity 96%, specificity 86%. In patients **without dementia**: sensitivity 93%, specificity 96%.
11. **Ultra-brief screens:** the single best item is **"months of the year backwards"** (sensitivity 83%, specificity 69%); the best two-item screen is **"months of the year backwards" + "what is the day of the week?"** (sensitivity 93%, specificity 64%).
12. **CAM-ICU** — for intubated or non-verbal patients. Inattention is tested with the letter task ("squeeze my hand every time you hear the letter A" → **SAVEAHAART**); altered consciousness is any **RASS score other than 0**; disorganised thinking uses yes/no questions ("will a stone float on water?").
13. **Screening for delirium superimposed on dementia (DSD):** best single item = **"What day of the week is it?"**; best two items = **days of the week backwards + day of the week**; best three items add **"What type of place is this?"** and **"Does the patient appear sleepy?"**. Sensitivities are high (84%, 93%, 94%) but specificities low (41%, 30%, 42%) — so a **negative screen helps rule out** delirium, but a positive screen needs fuller evaluation.
14. **Other validated tools named in Pathy:** SPMSQ, Delirium Rating Scale and DRS-R-98, Delirium Symptom Interview, Delirium Assessment Scale, Cognitive Test for Delirium, NEECHAM Confusion Scale, Confusional State Evaluation, **Memorial Delirium Assessment Scale**, Delirium Index, Delirium Severity Scale, Delirium Detection Score, DDT-Pro, **bCAM**, **4AT**, and the Stanford Proxy Test for Delirium. **Prediction** tools: **E-PRE-DELIRIC** and Stanford's Algorithm for Predicting Delirium (SAPD).

D. Frequency and clinical rules

15. **Screening frequency:** hospitalised patients **at least daily**; post-acute care patients **weekly**; long-term care residents **monthly** — and at any time a clinician, carer or relative reports a change from baseline.
16. **Never explain away a positive item.** Do not attribute a failed test to being in hospital, being unwell, being old, having just had analgesia, or having slept badly — **sleep disturbance is itself part of delirium**.
17. **When in doubt, assume delirium.** If collateral history is unavailable or the patient is too drowsy to complete an interview, **delirium should be presumed until proven otherwise** because the adverse outcomes are so severe.
18. **After a positive screen**, proceed to a **comprehensive medical evaluation** — full history including collateral and drug history, examination, targeted investigations, and systematic review of every predisposing and precipitating factor in the DELIRIUM IS VERY PAINFUL table. Neuroimaging, LP and EEG only on specific indication.

Table 3. Choosing a tool

Tool	Time	Best used for	Performance / note
CAM	~5 min	General ward, the reference screen	Sens 94%, Spec 89%; needs the 4-feature algorithm
3D-CAM	3 min	Structured ward screening, including dementia	Sens 93–96%, Spec 86–96%
4AT	<2 min	Ward and emergency department; needs	NICE- and SIGN-endorsed; works in drowsy patients

		no training	
UB-2	<1 min	Very rapid triage screen	Sens 93%, Spec 64% — rule-out only
CAM-ICU	2–3 min	Intubated / non-verbal ICU patients	Uses RASS + letter A task
MDAS / DRS-R-98	10–20 min	Grading **severity** and monitoring response	Research and palliative care settings
E-PRE-DELIRIC	—	**Prediction** of who will become delirious	Used to target preventive resources

Latest Updates (2023–Present)

- **NICE CG103 (January 2023)** issued **new recommendations specifically on assessing and diagnosing delirium**, and the **4AT** is now the recommended short assessment tool in both NICE and SIGN guidance — a shift from CAM-only teaching.
- **APA 2025 guideline** recommends **systematic screening of at-risk patients** with a validated tool rather than clinical impression alone, and adds **assessment at transitions of care**, including before discharge, as a distinct requirement.
- A recurring criticism in the 2025 literature is that the new guideline still does not mandate a **standardised pre-discharge delirium assessment**, despite evidence that a large proportion of patients leave hospital still delirious — a useful discussion point in a viva.
- **Diagnostic label drift:** commentary accompanying the APA guideline notes that delirium is systematically **under-coded** and preferentially recorded as "toxic or metabolic encephalopathy" for reimbursement reasons — a reason why administrative data underestimate its true burden.
- **ICD-11** has replaced ICD-10 in progressive international rollout; delirium remains a neurocognitive disorder with essentially the same core features (disturbed attention, awareness and cognition with acute onset and fluctuation).
- **Indian data** continue to show a wide gap between **referral-detected** delirium (around 0.4% of inpatients) and **screening-detected** delirium (around 30% of elderly ICU patients, ~45% of these hypoactive) — the strongest possible argument for routine tool-based screening rather than waiting for a referral.

Current Practice Update

Issue	Pathy 6th edition	Current practice
Preferred rapid tool	CAM family emphasised; 4AT listed among many	**4AT recommended by NICE and SIGN** for routine ward and ED screening
Diagnostic standard	DSM-5 and ICD-10	DSM-5 remains; **ICD-11** replacing ICD-10 in coding
Screening timing	Daily in hospital, weekly post-acute, monthly in LTC	Unchanged, **plus explicit assessment at care transitions and before discharge**
Severity measurement	Several severity scales listed	Greater emphasis on tracking **severity and trajectory**, not just presence or absence
Under-diagnosis	Acknowledged	Now attributed partly to **coding practice** as well as clinical oversight

Exam One-Liners — Q4

- **Screening ≠ diagnosis.** CAM/4AT screen; DSM-5 / ICD-10 diagnose.
- CAM = **(1 AND 2) PLUS (3 OR 4)**.
- CAM sens **94%**, spec **89%**; 3D-CAM takes **3 minutes**.
- Best single bedside item = **months of the year backwards**.
- CAM-ICU uses **RASS** and the **SAVEAHAART** letter task.
- Screen **daily** in hospital, **weekly** post-acute, **monthly** in long-term care.
- **Never make excuses** for a positive item.
- In doubt — **assume delirium until proven otherwise**.
- DSD screens are **sensitive but not specific** — good for ruling out.
- **Hypoactive delirium is the commonest and the most missed.**

Q5. Strategies for prevention of delirium in elderly inpatients

18 High-Yield Points

A. The case for prevention

1. **Up to 30–40% of delirium is preventable** with multicomponent, non-pharmacologic measures — the single most quotable figure in this answer.
2. **Prevention is worth it because delirium is not benign:** it causes falls, pulled lines, functional decline, longer stay, institutionalisation, PTSD, depression, increased mortality, and a cost burden exceeding **$164 billion a year in the United States**.
3. **Delirium is not always reversible.** Up to **45% are still delirious at discharge**; persistence is **33% at 1 month, 26% at 3 months and 21% at 6 months**. In Alzheimer's disease, an episode of hospital delirium **doubles the rate of cognitive decline for the next five years**. Prevention is therefore the only truly effective intervention.
4. **Because delirium is multifactorial, prevention must be multicomponent.** Single interventions and single drugs fail.
5. **Step one is risk identification and education.** Identify high-risk patients on admission (age, cognitive impairment, frailty, sensory impairment, severe illness, polypharmacy) and **tell the patient and family**. The risk of postoperative delirium should be part of the **routine informed consent** for surgery.
6. **Families are active agents, not visitors.** Relatives should stay at the bedside to reassure and reorient, help with oral intake, and encourage mobility.

B. The Hospital Elder Life Program (HELP) — the original model

7. **HELP** delivers targeted, **protocol-driven interventions** matched to specific risk factors: orientation and cognitively stimulating therapeutic activities; a **non-pharmacologic sleep protocol**; **mobility three times daily** (ambulation, or range-of-motion exercises if non-ambulant); **limiting tethers**; correcting **visual and hearing** impairment; and **preventing dehydration**.
8. **Original HELP trial (Inouye, NEJM 1999):** delirium occurred in **15.0%** on usual-care units versus **9.9%** on the intervention unit (matched OR 0.60, 95% CI 0.39–0.92).
9. **HELP meta-analysis (2018):** across 14 studies, delirium incidence fell significantly (**OR 0.47, 95% CI 0.37–0.59**); three studies showed a **42% reduction in falls**; nine showed cost savings of **$1600–3800 per patient** in hospital costs and **$16,000 per person-year** in long-term-care costs in the following year. HELP now runs in over **200 hospitals worldwide**.
10. **Modified HELP (mHELP)** for elective abdominal surgery used just **three nurse-delivered daily protocols — orienting communication, oral hydration and nutritional assistance, and early mobilisation**. Result: **relative risk of delirium 0.44** (95% CI 0.23–0.83, P = 0.008) and a length of stay **two days shorter**. This is the model to quote for a resource-limited Indian ward.

C. The ICU model — the ABCDEF bundle

11. **ABCDEF bundle** (ICU Liberation Collaborative): **A**ssess, prevent and manage pain; **B**oth spontaneous awakening and spontaneous breathing trials; **C**hoice of analgesia and sedation; **D**elirium — assess, prevent and manage; **E**arly mobility and exercise; **F**amily engagement and empowerment.
12. **Evidence:** in a prospective multicentre cohort of over **15,000 patients in 68 US ICUs**, complete bundle performance reduced delirium (**adjusted OR 0.60, 95% CI 0.49–0.72, P < 0.0001**), increased ICU discharge, and reduced death or discharge to a facility other than home.

D. Medication strategy — the highest-yield single intervention

13. **Medication review is one of the most important areas of prevention.** Review what is prescribed *and* what should be deprescribed on every ward round.
14. **Classes to avoid or minimise:** antipsychotics, **benzodiazepines** (especially IV — paradoxical reactions), corticosteroids, **H2-receptor antagonists in renal impairment**, narcotics (especially **pethidine** and IV opioids), muscle relaxants, **antimuscarinics for overactive bladder**, first-generation antihistamines, and **zolpidem** and related Z-drugs.
15. Use explicit tools — **AGS Beers Criteria** and **STOPP/START** — with a pharmacist-led medication reconciliation, and check **QTc risk** for any psychotropic considered.
16. **Treat pain properly, but do not sedate behaviour.** Regular **paracetamol** reduces total opioid requirement; uncontrolled pain is itself a precipitant.

E. Environmental and systems measures

17. **Ward-level measures:** consistent nursing staff; clocks, calendars and familiar objects; day–night lighting discipline; noise reduction and clustering of night-time care; avoidance of unnecessary ward transfers; early removal of urinary catheters, IV lines and telemetry; falls prevention **without physical restraints**; bowel and bladder regimens to prevent impaction and retention.
18. **System level:** clinicians should not wait for a formal programme — implement the individual components with the interprofessional team, and **advocate for hospital-wide delirium prevention**. Dedicated models such as the **Delirium Room** (a four-bed area within an ACE unit with 24-hour nursing and **no physical restraints**) and the **T-A-DA method** demonstrate that agitated delirious patients can be managed safely without restraint or sedation.

Table 4. Prevention bundle mapped to risk factor

Risk factor	Preventive protocol
Cognitive impairment	Orientation board, repeated reorientation, cognitively stimulating activities, familiar objects, family

	presence
Sleep deprivation	Non-drug sleep protocol — warm drink, relaxation, back rub, noise reduction, no night-time observations or drugs unless essential
Immobility	Ambulation or range-of-motion exercises **three times daily**; minimise tethers; physiotherapy input
Visual impairment	Spectacles, magnifiers, large-print materials, adequate lighting
Hearing impairment	Hearing aids with working batteries, earwax removal, amplification device
Dehydration	Early recognition, oral hydration assistance, volume repletion; monitor **BUN:creatinine ratio**
Undernutrition	Feeding assistance, dentures, nutritional supplements
Pain	Regular assessment; scheduled paracetamol; avoid pethidine
Polypharmacy	Beers / STOPP-START review; deprescribe; pharmacist involvement
Hypoxia / infection	Early detection and treatment; oxygen titration; avoid unnecessary catheters
Constipation / retention	Bowel chart and regimen; bladder scanning; avoid indwelling catheters

Latest Updates (2023–Present)

- **APA 2025 delirium guideline** — its single most important change, in the authors' own words, is the **additional emphasis on prevention**. It requires identification of at-risk individuals, a thorough medication and risk-factor history, and structured **transition-of-care** processes.
- **PADIS Focused Update 2025** — three positive conditional recommendations relevant to prevention: **dexmedetomidine over propofol** for light sedation, **enhanced mobilisation/rehabilitation over usual care**, and **melatonin** in ICU patients. Equally important is the negative recommendation: **do not use haloperidol, atypical antipsychotics, dexmedetomidine, statins or ketamine to prevent delirium**.
- **NICE CG103 (2023)** maintains the multicomponent prevention package and singles out **benzodiazepines, opioids and anticholinergics** as the pharmacological precipitants to target first.
- **Family involvement** in preventing delirium in critically ill patients was supported by a 2025 systematic review and meta-analysis — converting the older "families should help" advice into a formal, evidence-based bundle element.
- **AGS Beers 2023 and the AGS Beers Alternatives List (2025)** make deprescribing actionable by naming **safer substitutes** for insomnia, anxiety, pain and agitation, with a strong "less is more" and non-drug-first orientation.
- **Risk-factor evidence refreshed:** a 2025 meta-analysis identified **frailty, physical restraints, previous falls, illness severity, cognitive impairment and older age** as the dominant modifiable and non-modifiable risks — notably placing **physical restraints** among the leading risk factors, which reinforces restraint-free care.
- **Indian application:** the full HELP programme is resource-intensive, but the **three-protocol mHELP model** (orienting communication, oral hydration and nutrition, early mobilisation) is deliverable by ward nurses and family attendants at negligible cost. Geriatric wards and dedicated staff under **NPHCE**, and the presence of a family attendant in most Indian hospital beds, make an mHELP-style bundle the most realistic national strategy.

Current Practice Update

Issue	Pathy 6th edition	Current practice (2023–2026)
Overall emphasis	Prevention described alongside treatment	**Prevention is now the headline recommendation** (APA 2025)
Drug prophylaxis	Antipsychotics not supported for prevention	**Explicit recommendation against** antipsychotics, dexmedetomidine, statins and ketamine for prevention
Melatonin	Not recommended	**Conditionally suggested** in ICU patients
Mobilisation	Encouraged (three times daily in HELP)	**Enhanced mobilisation/rehabilitation formally recommended** over usual care
Family engagement	Encouraged	**Evidence-supported bundle component** with meta-analytic backing
Restraints	Avoid	Identified as an **independent risk factor**; permitted only for **imminent harm**
Discharge	Comprehensive discharge planning	Structured **transitions of care** — medication reconciliation, delirium follow-up, caregiver psychoeducation

Exam One-Liners — Q5

- **30–40% of delirium is preventable.**
- HELP trial: delirium **15.0% → 9.9%** (OR 0.60); meta-analysis **OR 0.47**, falls down **42%**.
- HELP targets six risk factors: **cognition, sleep, immobility, vision, hearing, dehydration**.
- mHELP = **orienting communication + oral hydration/nutrition + early mobilisation**; RR 0.44, LOS 2 days shorter.
- ICU = **ABCDEF bundle**; full compliance gives adjusted **OR 0.60** for delirium.
- **No drug prevents delirium** — not antipsychotics, statins, ketamine or dexmedetomidine.
- **Medication review is the highest-yield single preventive act.**
- **Physical restraints are a risk factor, not a safety measure.**
- Postoperative delirium risk belongs in the **surgical consent** discussion.

- Delirium Room + **T-A-DA** = restraint-free management of agitated delirium.

Recurring Topic — Extra Points to Remember

> Delirium appears in this question set five times. These are the details that separate a pass from a distinction, and the traps examiners most often set.

1. Numbers worth memorising

Figure	Value
Delirium in ICU	30–80%
Delirium in general medical / oncology wards	18–35%
Delirium in hospice care	26–44%
Delirium in nursing home / post-acute care	16%
Cardiac and vascular surgery	29–50%
Hip fracture repair	12–48%
Pooled prevalence in hospitalised older medical patients (2025 meta-analysis)	23.6%
Pooled incidence in the same group	13.5%
Preventable proportion	30–40%
Still delirious at discharge	up to 45%
Persistent at 1 / 3 / 6 months	33% / 26% / 21%
CAM sensitivity / specificity	94% / 89%
HELP: delirium usual care vs intervention	15.0% vs 9.9% (OR 0.60)
HELP meta-analysis odds ratio	0.47 (95% CI 0.37–0.59)
mHELP relative risk / LOS	RR 0.44; 2 days shorter
ABCDEF bundle (15,000 patients, 68 ICUs)	adjusted OR 0.60
US annual cost of delirium	> $164 billion
BUN : creatinine ratio associated with delirium	> 17 : 1

2. Named things you must be able to expand

- **SIFH** — Systems Integration Failure Hypothesis (Maldonado): the unifying pathophysiological model.
- **T-A-DA** — Tolerate, Anticipate, Don't Agitate.
- **DELIRIUM IS VERY PAINFUL** — the Pathy causes-and-contributors mnemonic (21 items).
- **ABCDEF** — the ICU Liberation bundle.
- **HELP / mHELP** — Hospital Elder Life Program and its three-protocol modification.
- **DSD** — delirium superimposed on dementia; **POD / POCD** — postoperative delirium and postoperative cognitive dysfunction (different entities, different mechanisms).
- **Cystocerebral syndrome** — delirium precipitated by acute urinary retention.
- **Delirium Room** — restraint-free, 24-hour-nursed four-bed area within an ACE unit.
- **E-PRE-DELIRIC / SAPD** — delirium prediction models.

3. Traps and common mistakes

- **Trap:** equating delirium with agitation. **Hypoactive delirium is the commonest subtype and carries the worst prognosis** because it is missed.
- **Trap:** ordering a CT head for every confused patient. Imaging is **indication-driven**.
- **Trap:** giving a benzodiazepine for a restless patient. It **precipitates and prolongs** delirium; the only exceptions are **alcohol and benzodiazepine withdrawal**.
- **Trap:** assuming delirium is always transient and fully reversible. It is not.
- **Trap:** restraining a patient who pulls at lines. Restraints are an **independent risk factor** for delirium; remove the line instead, or occupy the hands.
- **Trap:** attributing behaviour in a patient with dementia to the dementia. This causes **under-diagnosis of delirium and a missed serious illness**.
- **Trap:** writing "prescribe haloperidol" as the management answer. Non-pharmacologic multicomponent care is the mainstay; drugs are a rescue for imminent harm only.
- **Trap:** forgetting the simple bedside causes — **retention, impaction, pain, dehydration, spectacles and hearing aids**. These are what examiners want to hear.

4. Indian context to add for extra marks

- **Under-recognition is worse in India:** referral-based studies detect delirium in under 1% of inpatients while CAM-ICU screening finds around 30% of elderly ICU patients delirious, most of them hypoactive.

- **Family attendants** are present at almost every Indian hospital bed — formally enlisting them for orientation, hydration, feeding and mobilisation converts a staffing constraint into an evidence-based intervention.
- **NPHCE** geriatric wards and regional geriatric centres provide the structure in which a nurse-led mHELP-type bundle can be implemented at almost no cost.
- **AB-PMJAY**, the **Ayushman Vay Vandana Card** for those aged 70 and above, and the **Pradhan Mantri National Dialysis Programme** are the financing routes relevant to the septic CKD patient in Q3.
- **Tuberculosis, emphysematous pyelonephritis, diabetic foot sepsis and mucormycosis** deserve specific mention in Indian sepsis and confusion vignettes.
- **Alcohol withdrawal** is a very common and under-declared cause of ward delirium in Indian men — always take a corroborated alcohol history and give **parenteral thiamine before glucose**.

5. Fifteen-second answer skeleton (use for any delirium question)

> **Define** (acute, fluctuating, inattention, medical cause) → **Burden** (common, costly, preventable, under-diagnosed) → **Pathogenesis** (SIFH) → **Predisposing + Precipitating** (DELIRIUM IS VERY PAINFUL) → **Differentiate** (dementia, depression, psychosis, NCSE) → **Screen and diagnose** (CAM/4AT then DSM-5) → **Targeted workup** → **Treat the cause** → **Multicomponent non-pharmacologic care + T-A-DA** → **Drugs only for imminent harm** → **Prevent** (HELP/mHELP/ABCDEF, medication review) → **Prognosis and follow-up** (persistence, cognitive decline, transitions of care).

EXAMINATION ANSWER COMPILATION

DEMENTIA AND COGNITIVE DISORDERS

Questions 1–10

Primary reference: *Pathy's Principles and Practice of Geriatric Medicine, 6th edition* and *Kane's Essentials of Clinical Geriatrics*

Supplemented with: International guidelines and evidence 2023 – present, with Indian clinical and policy context

Prepared for: Sudarsan — MD Geriatric Medicine, Postgraduate Examination Revision

QUESTION 1

Discuss dementia in the elderly and its management.

15–20 High-Yield Points

A. DEFINITION AND NOSOLOGY

1. Dementia is a **clinical syndrome of sustained, acquired loss of intellectual function and memory**, severe enough to cause **dysfunction in daily living**, occurring in **clear consciousness**. Loss of functional ability due to impaired cognition is the key feature separating dementia from mild cognitive impairment.

2. Its cardinal features are a **gradually progressive course** (months to years) and **no disturbance of consciousness** — the two features that distinguish it from delirium. **DSM-5 renamed dementia "major neurocognitive disorder"** and introduced "mild neurocognitive disorder" for the MCI range.

B. EPIDEMIOLOGY

3. Dementia is broadly grouped into **reversible or partially reversible** and **non-reversible** types. **Reversible dementia accounts for under 4%** of cases, but concurrent treatable medical conditions causing mild cognitive disturbance are far commoner.

4. Alzheimer's disease (AD) and **vascular dementia** account for the vast majority in older people and **frequently coexist in the same patient (mixed dementia)**. **Dementia with Lewy bodies contributes 15–25%** in autopsy and clinical series. Approximately **6–8% of persons over 65 have AD**, with prevalence **doubling every 5 years** so that nearly **30% of those over 85** are affected.

5. Indian data: the **LASI-DAD** nationally representative study estimates dementia prevalence at **7.4% in adults aged 60+**, equating to about **8.8 million Indians**, projected to reach **16.9 million by 2036**. Prevalence is **higher in women than men and in rural than urban areas**, with wide state variation (Delhi 4.5% to Jammu & Kashmir 11.0%).

C. AETIOLOGY AND RISK FACTORS

6. Non-reversible causes are grouped as **degenerative** (AD, Lewy body disease, Parkinson's disease, Pick's disease, Huntington's disease, PSP), **vascular** (multi-infarct, Binswanger's disease, arteritis, post-anoxic), **traumatic** (including dementia pugilistica) and **infective** (HIV, Creutzfeldt–Jakob disease, progressive multifocal leukoencephalopathy, post-encephalitic).

7. Age and family history are the primary risk factors for AD. The **apolipoprotein E ε4 allele on chromosome 19** is the strongest genetic linkage with late-onset AD (relative risk ~2.5 in whites), but it is **neither sensitive nor specific and is not recommended as a screening test**. Rare mutations on **chromosomes 1, 14 and 21** cause early-onset familial AD.

D. PATHOPHYSIOLOGY

8. In AD, **progressive neuronal degeneration produces neurotransmitter abnormalities**, with a **combined cholinergic and glutamatergic dysfunction** underlying the symptomatology — this is the rationale for all currently licensed symptomatic drugs. **Amyloid-β deposition and hyperphosphorylated tau (neurofibrillary tangles)** are the defining neuropathological lesions.

9. Vascular pathology acts additively or synergistically with AD pathology: vascular lesions lower the threshold of AD pathology needed to produce dementia, and AD pathology increases the risk of dementia after stroke. Hence in a patient with AD plus cerebrovascular disease, **both conditions require treatment even if the vascular component seems trivial**.

E. CLINICAL FEATURES AND STAGING

10. Patients most commonly present with **memory loss, poor concentration, depression, behavioural change or language difficulty**. **Questioning both the patient and an informant is vital** to gauge impact on usual activities, lifestyle, history of the cognitive disorder and the key issue of **functional decline**.

11. Severity is graded with the **Global Deterioration Scale (GDS)**, the **Clinical Dementia Rating (CDR)** and the **MMSE — mild 20–24, moderate 10–19, severe below 9**. The **MMSE is dependent on sociocultural level and shows a ceiling effect**; the **Severe Impairment Battery (SIB)** is used when impairment is too severe for standard testing.

12. Neuropsychiatric symptoms occur in 90–98% of people with dementia over the disease course and may **precede the diagnosis**; they drive caregiver burden and institutionalisation.

F. DIAGNOSIS AND INVESTIGATION

13. Recommended baseline studies: complete blood count, **glucose**, blood urea, electrolytes (**sodium mEq/L, potassium mEq/L**), **calcium and phosphorus (mg/dL)**, liver function tests, **TSH (µIU/mL)**, **vitamin B12 (pg/mL)**, and serology for **syphilis and HIV if suspected**, together with **CT or MRI of the head**. Neuropsychological testing is reserved for selected patients.

14. Imaging is done to **exclude treatable structural causes** — subdural haematoma, tumour, normal-pressure hydrocephalus. **Cerebral atrophy alone does not establish AD.** Functional imaging aids differentiation: **HMPAO-SPECT shows temporoparietal hypoperfusion in AD, anterior hypoperfusion in FTD, multiple focal deficits in VaD; FP-CIT SPECT distinguishes DLB and parkinsonian syndromes from AD**.

G. MANAGEMENT

15. General principles: treat any reversible or partially reversible cause; **optimise therapy of comorbid conditions**; counsel families about the stages of dementia, drugs to avoid and excessive alcohol; discuss **behavioural symptom strategies, advance directives and future planning early**. **Treat hypertension** to prevent further lacunar infarcts, provided side effects are avoided.

16. Four pharmacological approaches (Kane's framework): (i) **avoid drugs that worsen cognition**, principally those with strong **anticholinergic activity**; (ii) **agents that enhance cognition and function**; (iii) **treat coexisting depression**; (iv) **treat complications** — paranoia, delusions, psychosis, agitation.

17. Cholinesterase inhibitors — donepezil (1996), rivastigmine (2000), galantamine (2001) — increase available acetylcholine; approved for **mild to moderately severe AD**, with **donepezil also approved for severe AD**. Pooled benefit is a **mean improvement of −2.7 points (95% CI −3.0 to −2.3) on the 70-point ADAS-Cog**.

18. Memantine, a **voltage-dependent, low-affinity, uncompetitive NMDA receptor antagonist**, blocks pathologically elevated glutamate and is approved for **moderate to severe AD**. **Combination of a ChEI with an NMDA antagonist gives better symptomatic effect.** In the **DOMINO trial**, continuing donepezil in moderate-to-severe AD gave **cognitive and functional benefit over 12 months**.

19. Vascular dementia: cholinesterase inhibitors are **not recommended for pure VaD** but should be used in **mixed dementia** once both AD and vascular components are demonstrated. **Antithrombotics need caution when lobar microbleeds are present on MRI** because of amyloid angiopathy and haemorrhage risk. **Levodopa may help apraxic gait** in subcortical ischaemic vascular disease.

20. Non-pharmacological management is central: **physical exercise is the single most efficient strategy for dementia prevention**, with multicomponent aerobic plus strength protocols reducing cortical atrophy in executive-control areas; cognitive interventions build **cognitive reserve**. Caregiver education and support, structured routine, meaningful activity, task simplification and environmental safety form the backbone of behavioural management.

Latest Updates (2023–Present)

- **Lancet Commission on Dementia Prevention, Intervention and Care (2024)** expanded the modifiable risk factor list from 12 to **14**, adding **high midlife LDL cholesterol (PAF 7%)** and **untreated vision loss in late life (PAF 2%)** to low education, hearing loss, hypertension, smoking, obesity, depression, physical inactivity, diabetes, excess alcohol, traumatic brain injury, air pollution and social isolation. **Up to 45% of dementia is theoretically preventable.**
- **Anti-amyloid monoclonal antibodies have entered practice**: **lecanemab** received full FDA approval in **July 2023** on the strength of **CLARITY-AD**, and **donanemab** was approved in **July 2024** following **TRAILBLAZER-ALZ 2**; both slow decline modestly in **early symptomatic AD (MCI or mild dementia) with confirmed amyloid pathology**. The **EMA restricted use to non-APOE ε4 homozygotes**, and **aducanumab was discontinued in 2024**.
- **Amyloid-related imaging abnormalities (ARIA)** dominate safety: serial **MRI monitoring is mandatory**, risk is **APOE ε4 dose-dependent** and greatest in the **first year**. Appropriate-use recommendations advise against treatment in **probable cerebral amyloid angiopathy** and in patients on anticoagulants.
- **Blood-based biomarkers have transformed diagnosis.** The **FDA cleared the Lumipulse G pTau217/β-amyloid 1-42 plasma ratio in May 2025** for symptomatic adults ≥55 years, with **91.7% positive and 97.3% negative concordance with amyloid PET/CSF**. The **Alzheimer's Association Clinical Practice Guideline (AAIC 2025)** recommends blood biomarker tests with **≥90% sensitivity and ≥75% specificity as a triaging test**, and those with **≥90% sensitivity and specificity as a substitute for amyloid PET or CSF**.
- **The 2024 Alzheimer's Association revised criteria** define AD **biologically** by Core 1 biomarkers (amyloid PET, CSF Aβ42/40, p-tau217, p-tau181, p-tau231) and Core 2 biomarkers (tau PET, MTBR-tau243), with **clinical staging separated from biological staging** — a decisive shift from the syndromic NINCDS-ADRDA approach.
- **Agitation now has licensed therapy**: **brexpiprazole was FDA-approved in May 2023** as the first drug for agitation in AD dementia (CMAI effect size ~0.35), and **dextromethorphan–bupropion (AXS-05) was approved in 2026** on the basis of **ADVANCE-1 and ACCORD-2** as the first **non-antipsychotic** option.

- **WHO ICOPE (2nd edition)** embeds **cognitive decline screening within integrated care for older people**, directly applicable to India's **NPHCE** platform.

Current Practice Update

Domain	Textbook position	Current evidence and guidelines (2023 onward)
Diagnosis	Clinical criteria plus structural imaging; biomarkers and amyloid PET largely research tools	**Biological diagnosis by biomarkers**; FDA-cleared **plasma p-tau217** ratio replaces PET/CSF in many pathways
Disease modification	"No disease-modifying drug is available at present"	**Lecanemab and donanemab approved** for early AD with confirmed amyloid; modest benefit, ARIA risk, restricted eligibility
Prevention	Exercise the most efficient strategy; 12 risk factors	**14 modifiable risk factors, ~45% preventable**; LDL cholesterol and vision loss added
Agitation	All drugs off-label; risperidone best short-term evidence	**Brexpiprazole (2023)** and **dextromethorphan–bupropion (2026)** now formally licensed
Indian epidemiology	Global estimates cited	**LASI-DAD: 7.4% of those 60+, 8.8 million cases, 16.9 million by 2036**

Exam One-Liners

- Dementia = acquired, progressive cognitive decline with **functional impairment in clear consciousness**.
- **Functional loss** separates dementia from MCI; **clear sensorium** separates it from delirium.
- India: **7.4% prevalence, 8.8 million cases (LASI-DAD)**.
- **AD + VaD coexist commonly**; mixed pathology outnumbers pure AD at autopsy.
- **APOE ε4** = strongest late-onset risk allele; **not a screening test**.
- MMSE staging: **mild 20–24, moderate 10–19, severe <9**.
- ChEI pooled benefit = **−2.7 ADAS-Cog points**.
- Memantine = **uncompetitive NMDA antagonist**, moderate-to-severe AD.
- **DOMINO** = continue donepezil in moderate/severe disease.
- **Lancet Commission 2024** = **14 risk factors, 45% preventable**.
- **Exercise** = most efficient single preventive strategy.

QUESTION 2

Dementia.

> **Note:** This question repeats Question 1 in condensed form. A **rapid-recall sheet** is given below rather than a repeated full answer — use it for final-night revision and refer to Question 1 for the expanded treatment.

Rapid-Recall Sheet — Dementia

1. DEFINITION IN ONE LINE

Acquired, progressive, sustained decline in memory and other cognitive domains causing loss of function in daily living, in clear consciousness (DSM-5: major neurocognitive disorder).

2. THE THREE-QUESTION TRIAGE BEFORE DIAGNOSING DEMENTIA

1. Was the onset **acute** (hours to days)? → think **delirium**
2. Are there **physical factors** (illness, sensory deprivation, drugs)? → think **reversible contribution**
3. Are there **psychological factors** (depression, psychosis)? → think **pseudodementia**

3. CAUSES — THE NUMERICAL ANCHORS

Type	Share
Alzheimer's disease	**50–60%**
Vascular / vascular cognitive impairment	**15–25%**
Dementia with Lewy bodies	**15–25%** (autopsy series)
Frontotemporal lobar degeneration	**3–20%**; 2nd commonest under 65
Truly reversible	**<4%**

4. CORTICAL VS SUBCORTICAL — THE DISCRIMINATOR TABLE

Feature	Cortical	Subcortical
Memory	Prominent impairment	Milder deficit
Executive function	Usually present	**Prominent**
Cortical signs	Aphasia, apraxia, agnosia	Absent
Mood	Less prominent	**Depression, lability, apathy**
Motor	Field cut, UMN signs	**Gait disturbance, extrapyramidal signs, dysphagia**
Example	AD, large-vessel VaD	Small-vessel VaD, PDD, PSP

5. INVESTIGATION PANEL (MEMORISE AS A BLOCK)

Blood: CBC · glucose · blood urea · electrolytes · **calcium and phosphorus (mg/dL)** · LFT · **TSH (µIU/mL)** · **vitamin B12 (pg/mL)** · VDRL and HIV if suspected **Imaging: CT or MRI head** — mandatory in recent-onset dementia, unexplained picture, or focal signs **Selected:** neuropsychological testing · CSF Aβ42 and p-tau · amyloid/FDG-PET · **plasma p-tau217**

6. MANAGEMENT IN FOUR LINES

1. **Treat reversible causes and comorbidity**; control vascular risk factors
2. **Stop anticholinergics and other cognition-worsening drugs**
3. **ChEI for mild–moderate AD; memantine for moderate–severe; combine for added symptomatic effect**
4. **Non-pharmacological first for behaviour** — DICE approach, caregiver training, exercise, structured routine, safe environment

7. INDIAN CONTEXT IN ONE PARAGRAPH

LASI-DAD: 7.4% prevalence in 60+, 8.8 million cases, projected 16.9 million by 2036, higher in women and rural residents. Care is delivered through **NPHCE** (district geriatric units, regional geriatric centres), **Elder Line 14567**, **AB-PMJAY and the Ayushman Vay Vandana Card** for those 70+, the **Maintenance and Welfare of Parents and**

Senior Citizens Act, 2007 for neglect and abandonment, **Rashtriya Vayoshri Yojana** for assistive devices and **ARDSI** for caregiver support and the Dementia India reports.

8. WHAT CHANGED AFTER THE TEXTBOOK

Anti-amyloid antibodies (**lecanemab 2023, donanemab 2024**) · **plasma p-tau217 FDA-cleared 2025** · **2024 Alzheimer's Association biological criteria** · **Lancet Commission 2024: 14 risk factors, 45% preventable** · **brexpiprazole 2023 and dextromethorphan–bupropion 2026 for agitation**.

QUESTION 3

Mild cognitive impairment — diagnostic criteria, progression and treatment modalities.

15–20 High-Yield Points

A. CONCEPT AND DEFINITION

1. The MCI construct originated with **Ronald Petersen and the Mayo Clinic group**, and has been repeatedly revised as biomarker science advanced, chiefly to locate MCI within the **prodromal phase of Alzheimer's disease**.

2. MCI is defined by **(i) a subjective disorder of memory and/or other cognitive domains, (ii) objective impairment of memory and/or other cognitive domains, and (iii) no significant impact on activities of daily living**. Patients therefore **do not meet accepted diagnostic criteria for dementia**.

3. Old age alone does not cause cognitive impairment severe enough to render a person dysfunctional. Slowed thinking, slowed reaction time, mild recent memory loss and impaired executive function may occur with age and **may or may not progress to dementia** — labelled **MCI** or **cognitive impairment, not dementia (CIND)**.

4. MCI is **heterogeneous**: it may be due to AD (prodromal AD), other neurodegenerative disease, **depression, vascular lesions or traumatic brain injury**.

B. CORE CLINICAL CRITERIA (NIA-AA 2011)

5. Concern regarding a change in cognition — obtained from the patient, an informant, or a clinician observing the patient.

6. Impairment in one or more cognitive domains — performance lower than expected for the patient's **age and education**.

7. Preservation of independence in functional abilities — mild difficulty with complex tasks is common, but independence is maintained **with minimal aids or assistance**.

8. Not demented — changes are mild enough that there is **no significant impairment of social or occupational functioning**. Note that **no defined thresholds** are specified for the degree of cognitive impairment; the syndrome is almost identical to Petersen's original description.

C. RESEARCH CRITERIA AND BIOMARKERS

9. NIA-AA biomarkers fall into three classes: **(i) markers directly reflecting AD pathology** — CSF **low Aβ42**, amyloid PET, CSF **raised total tau or p-tau**; **(ii) non-specific markers of neuronal injury** — atrophy on MRI, hypometabolism or hypoperfusion on FDG-PET/SPECT; **(iii) markers of cell death, synaptic damage, oxidative stress or inflammation**.

10. Levels of diagnostic certainty: MCI due to AD "high likelihood" = positive Aβ **and** positive neuronal-injury biomarker; **"intermediate likelihood"** = one positive with the other untested; **"unlikely due to AD"** = both negative. Results may also be **uninformative** if ambiguous or conflicting.

11. The **International Working Group (Dubois)** criteria for **prodromal AD** run in parallel: **IWG-1** requires abnormal amyloid, tau, FDG-PET or hippocampal atrophy; **IWG-2** requires **abnormal CSF Aβ1-42 and tau, or abnormal amyloid PET**. They also separate **typical AD** (amnestic syndrome of hippocampal type) from **atypical AD** (posterior cortical atrophy, logopenic variant, frontal variant).

12. The **NIA-AA 2018 Research Framework** introduced the **AT(N) classification**: **A** = aggregated Aβ (CSF Aβ42 or Aβ42/40, amyloid PET); **T** = aggregated tau (CSF p-tau, tau PET); **(N)** = neurodegeneration, non-specific and hence bracketed (MRI, FDG-PET, CSF total tau). It uses the label **"Alzheimer's disease with MCI"** rather than "MCI due to AD".

13. DSM-5 replaced dementia with **major neurocognitive disorder** and introduced **mild neurocognitive disorder (mNCD)**, which parallels MCI — cognitive complaint, psychometric impairment and relative preservation of ADLs.

D. EPIDEMIOLOGY AND PROGRESSION

14. Just over 20% of people aged 71 and older in the United States have **cognitive impairment without dementia**.

15. Progression: up to 15–20% of those diagnosed with MCI or CIND convert to dementia over the course of a year. Progression is not inevitable — a proportion remain stable and some revert to normal, especially where depression or drugs were contributory.

16. Subjective cognitive decline (SCD) precedes MCI; cross-sectional studies show SCD does not necessarily correlate with objective impairment, but **longitudinal studies link SCD to future AD or dementia**. A cognitively unimpaired subject with a **positive AD biomarker** is regarded as **at risk of AD or in a preclinical stage**.

E. TREATMENT MODALITIES

17. No intervention has been shown to prevent progression to dementia in classical practice; the therapeutic implications of MCI remain an area of intensive research. **Cholinesterase inhibitors are not licensed for MCI**, and European practice prescribes them less often than American practice.

18. Identify and treat contributory conditions: depression (SSRI such as **citalopram or sertraline**, or **mirtazapine**, all with minimal cognitive side effects), **hypothyroidism, vitamin B12 deficiency, drug side effects, obstructive sleep apnoea and alcohol excess**; and **withdraw anticholinergic drugs**.

19. Vascular risk factor control, especially hypertension, may favourably influence the clinical course of both degenerative and vascular cognitive impairment. **Physical activity and multicomponent exercise** carry the most promising evidence, supported by the **FINGER trial** of multidomain intervention; the **MAPT trial** of omega-3 with or without multidomain intervention was largely negative except in amyloid-positive subgroups.

20. Practical management includes an **informed discussion of diagnosis and prognosis**, arrangements for **ongoing review and support**, cognitive training and stimulation, driving and safety assessment, and advance planning. Most countries, **including India, lack national guidelines on biomarker use in early AD**, which limits harmonisation of practice.

Latest Updates (2023–Present)

- **MCI due to AD is now a treatable diagnosis.** Both **lecanemab (CLARITY-AD)** and **donanemab (TRAILBLAZER-ALZ 2)** are licensed specifically for **MCI due to AD or mild AD dementia with biomarker-confirmed amyloid pathology** — the first time an MCI-stage diagnosis carries a disease-modifying option.
- **Eligibility is biomarker-gated:** amyloid must be confirmed by **amyloid PET, CSF, or now an approved blood test**. Treatment requires **APOE genotyping** (ε4 homozygotes have the highest ARIA risk; excluded in the EU), **baseline and serial MRI**, and caution with anticoagulants and probable cerebral amyloid angiopathy.
- **Plasma biomarkers have reached the MCI clinic.** The **Alzheimer's Association Clinical Practice Guideline (2025)** covers patients **with objective cognitive impairment including MCI**, endorsing **p-tau217, %p-tau217, p-tau181, p-tau231 and Aβ42/Aβ40** with the two accuracy thresholds (**≥90/≥75% for triage; ≥90/≥90% as a substitute for PET or CSF**). The **FDA-cleared Lumipulse G pTau217/Aβ1-42 ratio (May 2025)** is indicated for symptomatic adults ≥55 years — precisely the MCI population.
- **The 2024 Alzheimer's Association revised criteria** stage AD biologically and clinically in parallel, replacing "MCI due to AD" with **clinical Stage 3 (mild cognitive impairment) of the Alzheimer's continuum** in biomarker-positive individuals.
- **Risk reduction is now the mainstay for non-AD MCI:** the **Lancet Commission 2024** framework — hearing aids for hearing loss, cataract surgery and correction of vision loss, LDL and blood pressure control, physical activity, social engagement — carries the strongest population-level evidence, with **US POINTER (2025)** confirming benefit of structured multidomain lifestyle intervention in at-risk older adults.
- **Indian relevance:** biomarker access remains restricted to a few tertiary centres; **MCI detection in India rests on culturally validated cognitive tools** (Hindi Mental State Examination, ACE-III adaptations, LASI-DAD battery, AIIMS Comprehensive Neuropsychological Battery in Hindi) and on the **NPHCE** and **ICOPE** screening platforms rather than on plasma or CSF assays.

Current Practice Update

Aspect	Textbook position	Current evidence and guidelines (2023 onward)
Therapeutic implications	"No intervention has been shown to prevent progression to dementia"	**Anti-amyloid antibodies licensed for MCI due to AD**; modest slowing of decline, not prevention
Biomarker access	Most countries lack national guidelines; unequal access limits practice	**Blood biomarkers (p-tau217) FDA-cleared and guideline-endorsed**, greatly widening access
Terminology	MCI due to AD (2011) / AD with MCI (2018 framework)	**2024 criteria: biologically defined AD, clinical Stage 3**
Conversion risk	15–20% per year	Confirmed, but **biomarker-positive MCI converts far faster than biomarker-negative MCI**, which often remains stable or reverts
Lifestyle intervention	FINGER promising, MAPT largely negative	**US POINTER (2025)** supports structured multidomain lifestyle intervention in at-risk older adults

Exam One-Liners

- MCI = **subjective complaint + objective impairment + preserved ADLs + not demented**.
- Four core clinical criteria: **concern, objective impairment, preserved function, not demented**.
- **No thresholds are specified** for degree of impairment — a common examiner trap.
- **Conversion 15–20% per year**; **20% of those over 71 have CIND**.
- **AT(N)**: A = amyloid, T = tau, **(N) = neurodegeneration, non-specific**.
- IWG-2 prodromal AD = **abnormal CSF Aβ1-42 and tau, or abnormal amyloid PET**.
- DSM-5 equivalent = **mild neurocognitive disorder**.
- **ChEIs are not licensed for MCI.**
- **Lecanemab and donanemab are licensed at the MCI stage** when amyloid is confirmed.
- **FINGER positive, MAPT negative, US POINTER positive** for multidomain lifestyle intervention.

QUESTION 4

Reversible / treatable dementias — investigation and management.

RECURRING TOPIC ALERT — Reversible dementias appear repeatedly in MD Geriatrics papers, usually as a 15-mark long question or as a "how will you investigate a patient with cognitive decline" case. Additional recall scaffolding is given at the end of this answer.

15–20 High-Yield Points

A. DEFINITION AND SCALE OF THE PROBLEM

1. Dementia in older people is grouped into **reversible or partially reversible** and **non-reversible** types. The reversible group is the **clinically indispensable minority**: they are few in number but represent the cases where diagnosis changes outcome.

2. Reversible dementia accounts for probably less than 4% of cases. However, **concurrent medical conditions causing mild cognitive disturbance are much commoner and are potentially treatable** — identifying them avoids unnecessary disability and misdiagnosis even when cognition does not fully normalise.

3. An important caveat: **finding a reversible cause does not guarantee that the dementia will improve once that cause is treated.** Thyroid hormone and vitamin B12 replacement may be important for general health but **have not been shown to reverse established cognitive deficits**.

B. CAUSES — THE CLASSIFIED LIST

4. Neoplasms — primary and secondary intracranial tumours, and paraneoplastic/limbic encephalitis.

5. Metabolic and endocrine disorders — hypothyroidism, hyperparathyroidism, hyponatraemia, hypercalcaemia, hepatic and renal failure, hypoglycaemia.

6. Nutritional disorders — **vitamin B12 deficiency**, folate deficiency, thiamine deficiency (**Wernicke–Korsakoff syndrome**), pellagra.

7. Trauma — **chronic subdural haematoma**, post-traumatic states, dementia pugilistica (the last irreversible).

8. Toxins — **alcoholism**, heavy metals (lead, mercury, arsenic, manganese), organic poisons.

9. Drugs — the single most important reversible cause in practice: **anticholinergics and antihistamines, narcotic and non-narcotic analgesics, NSAIDs, anticonvulsants, antihypertensives, antiparkinsonian drugs, digoxin, antiarrhythmics, H2 receptor antagonists, antianxiety drugs, antidepressants, antipsychotics, sedative-hypnotics, skeletal muscle relaxants, steroids and alcohol**.

10. Infections — **HIV and opportunistic infections**, neurosyphilis, tuberculous meningitis, chronic fungal meningitis, viral encephalitis, Whipple's disease.

11. Autoimmune disorders — **CNS vasculitis, temporal arteritis, systemic lupus erythematosus, multiple sclerosis**, and autoimmune encephalitis.

12. Psychiatric disorders — **depression ("depressive pseudodementia")**.

13. Other — **normal-pressure hydrocephalus**, obstructive sleep apnoea, and severe sensory deprivation from uncorrected hearing and visual loss.

C. DEPRESSIVE PSEUDODEMENTIA — A SPECIAL CASE

14. Depression coexists with dementia in more than one-third of outpatients with dementia and in an even greater proportion in nursing homes. Many patients with early dementia become depressed, and depression may be diagnosed before dementia becomes evident.

15. Clinical pointers to depression as the cause: **prominent complaints of memory loss** (contrast with the concealment typical of AD), **patchy and inconsistent cognitive deficits** on examination, and **frequent "don't know" answers**. **Detailed neuropsychological testing** helps, and where doubt persists, **a careful trial of an antidepressant is justified** both diagnostically and therapeutically.

16. Important prognostic point: **older patients who develop reversible cognitive impairment while depressed are at relatively high risk of developing dementia over the following few years** and need close cognitive follow-up.

D. INVESTIGATION

17. These disorders are detected by careful history, physical examination and selected laboratory studies. The **recommended panel** is: complete blood count, **glucose**, blood **urea nitrogen**, **electrolytes (sodium and potassium**

in mEq/L), **calcium and phosphorus (mg/dL)**, liver function tests, **thyroid-stimulating hormone (µIU/mL)**, **vitamin B12 (pg/mL)**, **serological test for syphilis if suspected**, and **HIV antibodies if suspected**.

18. CT or MRI of the head should be performed to exclude unsuspected correctable structural lesions in patients with **dementia of recent onset in whom no other clinical finding explains the dementia**, and in those with **focal neurological signs or symptoms** — chiefly to exclude **subdural haematoma, tumour and normal-pressure hydrocephalus**. **CT and MRI are roughly equivalent for detecting remediable structural lesions**; MRI shows more lesions in multi-infarct dementia but also white-matter changes of uncertain significance.

19. Neuropsychological testing is valuable when the mental status score is normal but function or behaviour has changed (high baseline intelligence), when the score is low without functional deficit (low educational level), and specifically for **differentiating depression from dementia**.

E. MANAGEMENT

20. Withdraw or substitute every drug that may be worsening cognition, especially those with **strong anticholinergic activity**, and maintain a **high index of suspicion for excessive alcohol intake**, which is easily missed and causes dementia, delirium, depression and falls. **Treat depression energetically** with an SSRI such as **citalopram or sertraline**, or the NaSSA **mirtazapine**, all of which have minimal cognitive side effects. **Correct B12 deficiency, hypothyroidism, hyponatraemia and hypercalcaemia; refer NPH for shunt assessment and chronic subdural haematoma for neurosurgical evaluation; treat neurosyphilis, HIV and tuberculous meningitis specifically; and control vascular risk factors, especially hypertension**, which may favourably influence the course of both degenerative and vascular cognitive impairment.

Latest Updates (2023–Present)

- **Shunting for iNPH now has randomised placebo-controlled proof.** The **PENS trial (NEJM, 2025; 99 patients, 17 centres)** compared open with placebo shunt settings: **gait velocity improved by 0.21 m/s more with an open shunt (p<0.001)**, and **80% of the open-shunt group achieved meaningful improvement versus 24% with placebo**, with better balance and fewer falls. **Cognition and continence did not improve significantly** — an important nuance. This settles the long-standing debate and strengthens the case for shunting the one classically reversible dementia.
- **Autoimmune and antibody-mediated encephalitis** is now firmly established as a treatable cause of subacute cognitive decline in older adults — **LGI1, CASPR2, NMDAR and GAD65 antibodies** should be tested when there is **rapid progression over weeks to months, seizures (especially faciobrachial dystonic seizures with LGI1), hyponatraemia, movement disorder or psychiatric prominence**; immunotherapy can produce dramatic recovery.
- **Rapidly progressive dementia** is a recognised diagnostic emergency: the working rule is that **decline to dementia within 12 months** mandates urgent search for **autoimmune, infective, neoplastic, metabolic and toxic causes** before accepting Creutzfeldt–Jakob disease, using CSF **RT-QuIC**, 14-3-3, autoantibody panels and MRI DWI.
- **Deprescribing has been formalised.** The **AGS Beers Criteria (2023 update)** reinforce avoidance of **strongly anticholinergic drugs, benzodiazepines, Z-drugs and first-generation antihistamines** in older adults, and **anticholinergic burden scales (ACB, ARS)** are now recommended for routine calculation in memory clinics.
- **Obstructive sleep apnoea and untreated hearing and vision loss** have moved from footnote to mainstream: the **Lancet Commission 2024** added **untreated vision loss** as a modifiable risk factor, and **ACHIEVE (2023)** showed hearing aids slowed cognitive decline in older adults at increased risk. Both are practical, high-yield reversible contributors in Indian practice.
- **Indian context: B12 deficiency and hypothyroidism are disproportionately common**, given widespread vegetarianism and iodine-related thyroid disease, making the B12/TSH pair particularly high-yield in Indian memory clinics. **Neurosyphilis, HIV-associated neurocognitive disorder and tuberculous meningitis** remain far commoner reversible causes here than in Western series. **NPHCE district geriatric units and ICOPE screening** are the practical platform for early detection.

Current Practice Update

Area	Textbook position	Current evidence and guidelines (2023 onward)
NPH shunting	Surgery, with risk–benefit carefully assessed; efficacy debated	**PENS trial (2025)** — randomised, placebo-controlled proof of **gait** benefit; cognition and continence do not reliably improve
Autoimmune causes	CNS vasculitis, SLE, MS listed	**Antibody-mediated encephalitis (LGI1, CASPR2, NMDAR, GAD65)** now a defined, immunotherapy-responsive category
B12 and thyroid replacement	Not shown to reverse cognitive deficits	Position unchanged; treat for **general health and to prevent further decline**, not with the expectation of cognitive reversal
Sensory impairment	Sensory deprivation noted as contributory	**ACHIEVE (2023)**: hearing aids slow decline in at-risk elders; **vision loss added to Lancet Commission list (2024)**
Drug-induced cognitive impairment	List of offending drug classes	Formalised through **AGS Beers 2023** and routine **anticholinergic burden scoring** with structured deprescribing

Extra Recall Points — Recurring Topic

MNEMONIC FOR REVERSIBLE DEMENTIAS: "DEMENTIA"

Letter	Cause
D	**D**rugs (anticholinergics, sedatives, opioids, steroids) and **D**epression
E	**E**motional (pseudodementia) and **E**ndocrine (hypothyroidism, hyperparathyroidism)
M	**M**etabolic (Na^+, Ca^{2+}, uraemia, hepatic failure)
E	**E**yes and **E**ars — sensory deprivation
N	**N**utritional (**B12**, folate, thiamine) and **N**ormal-pressure hydrocephalus
T	**T**umour, **T**rauma (chronic subdural haematoma)
I	**I**nfection (HIV, syphilis, TB meningitis) and **I**nflammatory/autoimmune
A	**A**lcohol and toxins

NUMERICAL ANCHORS

- Reversible dementia: **<4%** of all dementias
- Depression coexists in **>1/3** of dementia outpatients
- Rapidly progressive dementia = decline to dementia in **<12 months**
- **PENS trial: gait velocity gain 0.21 m/s; 80% vs 24% meaningful improvement**

NAMED ENTITIES TO QUOTE

Clarfield's analyses (small proportion truly reversible) · **Hospital Elder Life Program (HELP)** for delirium prevention overlap · **PENS trial 2025** · **ACHIEVE 2023** · **AGS Beers Criteria 2023** · **Lancet Commission 2024**

COMMON EXAMINER TRAPS

1. Saying reversible dementias are common — they are **<4%**; the value of the search is that the yield, though small, is decisive.
2. Claiming B12 and thyroid replacement reverse dementia — the textbook explicitly states **they have not been shown to do so**.
3. Forgetting that **drugs are the commonest single reversible contributor** and the easiest to correct.
4. Omitting **normal-pressure hydrocephalus and chronic subdural haematoma** — both are surgically treatable.
5. Diagnosing dementia during an episode of delirium — **the diagnosis of dementia must await treatment of all potentially reversible causes of delirium**.
6. Failing to mention that **treating a reversible cause may not restore cognition** but still prevents excess disability.

15-SECOND ANSWER SKELETON

Define → state <4% truly reversible but always worth seeking → classify causes (drugs, depression, metabolic/endocrine, nutritional, structural, infective, autoimmune, NPH) → investigation panel (bloods + TSH + B12 + CT/MRI) → management (deprescribe, treat depression, correct deficiencies, surgical referral for NPH and subdural, control vascular risk) → caveat that reversal is not guaranteed → Indian angle (B12, hypothyroidism, neurosyphilis, HIV, TB meningitis).

Exam One-Liners

- **<4%** of dementias are truly reversible; concurrent treatable conditions are far commoner.
- **Finding the cause does not guarantee reversal.**
- **Drugs** are the commonest reversible contributor — anticholinergics first.
- Depressive pseudodementia: **prominent memory complaints, patchy deficits, "don't know" answers**.
- Depression coexists with dementia in **over one-third** of outpatients.
- Panel: **CBC, glucose, urea, electrolytes, calcium/phosphorus, LFT, TSH, B12, VDRL and HIV if suspected, CT/MRI head**.
- **CT and MRI are equivalent** for detecting remediable structural lesions.
- Structural causes to exclude: **subdural haematoma, tumour, NPH**.
- Treated depression with cognitive impairment → **high risk of later dementia**; follow up closely.
- **PENS 2025**: shunting improves **gait**, not cognition or continence.

QUESTION 5

Behavioural symptoms of dementia.

15–20 High-Yield Points

A. DEFINITION AND TERMINOLOGY

1. Neuropsychiatric symptoms (NPSs), also called **behavioural and psychological symptoms of dementia (BPSD)**, are defined as **signs and symptoms of disturbed perception, thought content, mood or behaviour** occurring in a person with dementia.

2. They encompass **depression, anxiety, apathy, irritability, disinhibition, agitation, rejection of care, aggression, elation, and problems with eating or sleeping**. They may occur **before, during or after the diagnosis of dementia**, and may be **among the earliest signs of incipient cognitive decline**.

B. EPIDEMIOLOGY

3. 90 to 98% of people with dementia develop at least one NPS over the course of the disease; the prevalence of NPS therefore mirrors the overall prevalence of dementia. NPSs occur **across all dementia types and all stages**.

4. In **Alzheimer's disease the commonest NPS is apathy**, followed by **depression, aggression, anxiety, sleep disturbance, irritability, appetite disturbance, motor problems, delusions, disinhibition, hallucinations and euphoria**.

C. AETIOLOGY — THE FIVE CONTRIBUTORY DOMAINS

5. Neurodegeneration-associated factors — the underlying disease process itself, with cholinergic, serotonergic and frontal-subcortical circuit disruption.

6. Acute medical issues — pain, infection (especially urinary and respiratory), constipation, urinary retention, dehydration, drug changes, poor sleep and sensory impairment. **Pain is a common and frequently missed cause of agitation**, and occasionally **a trial of a scheduled analgesic is the only way to establish it**.

7. Pre-existing personality and psychiatric illness, **unmet needs** (hunger, boredom, need for toileting, need for company), **caregiver factors** (communication style, unrealistic expectations, countertherapeutic approach) and **environmental factors** (clutter, poor lighting, over- or under-stimulation, unfamiliar surroundings).

D. ASSESSMENT

8. The standard instruments are the **Neuropsychiatric Inventory (NPI)**, its questionnaire form **NPI-Q**, and the clinician version **NPI-C**; the **Cohen-Mansfield Agitation Inventory (CMAI)** for agitation; and the **Cornell Scale for Depression in Dementia (CSDD)** for mood, which relies on both patient and informant.

9. Always exclude delirium first. Delirium superimposed on dementia (DSD) is common and is identified by the **Confusion Assessment Method (CAM) — acute onset and fluctuating course, plus inattention, plus either disorganised thinking or altered level of consciousness**.

E. THE DICE APPROACH

10. DICE — Describe, Investigate, Create, Evaluate — was developed by a US national expert panel (geriatric medicine, geriatric psychiatry, psychology, nursing, social work, pharmacy and behavioural science) as a **step-by-step method for assessing and managing NPS**, designed for use by **any health professional in a team setting**.

11. Describe — elicit a thorough description from the caregiver, characterising the symptom and the **context in which it occurs**, including possible **antecedents or triggers**, and identifying which aspects are **most distressing to the patient and caregiver**.

12. Investigate — examine, exclude and identify **modifiable underlying causes**: patient factors (medical issues, pain, medication change, poor sleep, sensory change), caregiver factors (communication and expectations) and environmental contribution (clutter, lighting, stimulation level).

13. Create — collaborate with caregiver, patient and team on a treatment plan built on **five generalised strategies: caregiver education; enhancing effective communication; creating meaningful activities; simplifying tasks and establishing structured routines; and ensuring safety while simplifying and enhancing the environment**.

14. Evaluate — assess whether strategies were implemented, whether target symptoms improved, whether caregiver distress fell and whether there were unintended effects. **If a psychotropic was used, consider a trial of dose reduction or discontinuation.**

F. NON-PHARMACOLOGICAL TREATMENT

15. Family and paid caregiver interventions have the strongest evidence base and a greater effect than antipsychotics. Efficacy is best in **mild and moderate stages**, when cognitive and communication functions are better preserved. Individual approaches such as **music therapy and physical activity** work best when embedded within **tailored-activity programmes**. Environmental adjustments work best **in combination** rather than singly.

G. PHARMACOLOGICAL TREATMENT

16. There are no FDA-approved drugs for NPSs in AD in the classical position — the sole exception being **pimavanserin for hallucinations and delusions in Parkinson's disease**. In the **European Union and Canada, risperidone is indicated for short-term treatment of severe aggression**. Most agents are therefore used **off-label**.

17. Antipsychotics: modest efficacy for agitation/aggression and psychosis is supported for four atypicals — **risperidone, aripiprazole, olanzapine and quetiapine** — with **risperidone having the best evidence for short-term (6–12 week) efficacy in agitation**. Use is limited by **increased risk of stroke and death**.

18. Antidepressants: the **DIADS** trial showed benefit of **sertraline** in AD with major depression, but **DIADS-2 was negative**, as was the **HTA-SADD** comparison of sertraline, mirtazapine and placebo; meta-analysis shows **small-to-null effect sizes**. **Citalopram (target 30 mg) in the CitAD trial reduced agitation, anxiety, irritability and caregiver distress**, but caused **cognitive decline and QTc prolongation** at that dose. **Trazodone** is widely used for **night-time agitation and sleep**, stabilising circadian rhythm.

19. Other classes: benzodiazepines lack trial evidence and should be reserved **for crises only**, using short- to intermediate-acting agents such as **lorazepam or oxazepam**. **Valproate and carbamazepine have not shown benefit and cause significant adverse effects** — sedation, gait disturbance and tremor. **Cholinesterase inhibitors and memantine have not shown sufficient evidence for NPSs in AD**, although ChEIs do help NPSs in **DLB, Parkinson's disease dementia and cognitive impairment in PD**, at the cost of increased parkinsonian symptoms such as tremor.

20. Expert consensus (International Delphi process) endorses an **escalating approach**: identify underlying causes first; then **caregiver training, environmental adaptation, person-centred care and tailored activities** as first-line for overall NPS and agitation; then, if drugs are needed, **citalopram and analgesia before antipsychotics**. For **psychosis specifically, pharmacological options and particularly risperidone are prioritised** after assessing underlying causes. **DICE and music therapy** were agreed as the most promising non-pharmacological approaches.

Latest Updates (2023–Present)

- **Agitation in AD now has two licensed drugs. Brexpiprazole (Rexulti) was FDA-approved in May 2023** — the first approval for any neuropsychiatric symptom of dementia — showing **~31% reduction in agitation versus placebo**, with **CMAI effect size ~0.35** and CGI-S benefit significant only in the 3 mg group. It **retains the class boxed warning for increased mortality in elderly patients with dementia-related psychosis**.
- **Dextromethorphan–bupropion (AXS-05, Auvelity) was FDA-approved in 2026** as the **first non-antipsychotic** treatment for AD agitation. Dextromethorphan acts as an **NMDA receptor antagonist and sigma-1 agonist**, with bupropion inhibiting CYP2D6 to sustain levels. **ADVANCE-1** showed CMAI improvement versus placebo at week 5, and **ACCORD-2** showed **delayed relapse (HR 0.276) and reduced relapse incidence (8.4% vs 28.6%)** over 26 weeks of randomised withdrawal. It carries a **boxed warning for suicidal thoughts and behaviours** and is contraindicated with **seizure disorder, eating disorders, recent alcohol/benzodiazepine withdrawal and MAOIs**.
- **Pimavanserin** remains approved only for **Parkinson's disease psychosis**; the **HARMONY** trial in dementia-related psychosis showed relapse prevention but did not secure a broader indication.
- **International Psychogeriatric Association (IPA) 2023 provisional consensus definition of agitation in cognitive disorders** now standardises the syndrome for both trials and practice, requiring **excessive motor activity, verbal aggression or physical aggression causing excess distress or disability**.
- **Non-pharmacological evidence has strengthened**: **WHELD** and subsequent person-centred care packages, tailored activity programmes and **music-based interventions (Cochrane 2022–2025)** show consistent modest benefit on agitation and depressive symptoms with no drug-related harm, reinforcing their first-line position.
- **Deprescribing antipsychotics** is now a formal quality indicator in many systems, supported by **AGS Beers 2023** and by evidence that gradual withdrawal is safe in most long-term care residents without severe baseline symptoms.
- **Indian context:** BPSD is a leading driver of caregiver distress and institutionalisation in India, where **over 90% of care is family-provided**. Practical supports include the **National Mental Health Programme and District Mental Health Programme**, **NPHCE geriatric clinics**, **Elder Line 14567**, and **ARDSI** caregiver training. **Antipsychotic access is unregulated and off-label use is common**, making structured DICE-style caregiver training the most realistic first-line strategy.

Current Practice Update

Area	Textbook position	Current evidence and guidelines (2023 onward)
Licensed drugs for agitation	No FDA-approved drug for NPS in AD; all off-label except pimavanserin in PD psychosis	**Brexpiprazole (2023)** and **dextromethorphan–bupropion (2026)** are FDA-approved for AD agitation
First-line approach	Non-pharmacological first; caregiver interventions outperform antipsychotics	Unchanged and reinforced — **DICE, person-centred care, tailored activity, music therapy**
Citalopram	CitAD: effective but cognitive decline and QTc prolongation at 30 mg	Position unchanged; **escitalopram (S-CitAD, 2024) did not separate from placebo**, so the SSRI role remains uncertain
Definition of agitation	Descriptive	**IPA 2023 consensus definition** standardises the syndrome
Antipsychotic use	Modest efficacy; stroke and mortality risk	**Deprescribing is now a formal quality indicator**; gradual withdrawal safe in most residents

Exam One-Liners

- **90–98%** of people with dementia develop at least one NPS.
- **Apathy is the commonest NPS in AD**, followed by depression and aggression.
- **DICE = Describe, Investigate, Create, Evaluate.**
- The five "Create" strategies: **educate, communicate, meaningful activity, simplify tasks/routine, safe environment**.
- **Caregiver interventions outperform antipsychotics.**
- **Risperidone** has the best short-term evidence for agitation; only EU/Canada licence it.
- **CitAD**: citalopram 30 mg reduces agitation but causes **cognitive decline and QTc prolongation**.
- **Valproate and carbamazepine are ineffective** and harmful.
- **ChEIs and memantine do not help NPS in AD** but do help in DLB and PDD.
- **Always exclude pain, infection, constipation, retention and delirium first.**
- New licences: **brexpiprazole 2023; dextromethorphan–bupropion 2026**.

QUESTION 6

Compare and contrast the clinical features and brain areas involved in the four common dementias.

15–20 High-Yield Points

A. FRAMEWORK

1. The **four common dementias** are **Alzheimer's disease (AD)**, **vascular dementia (VaD)**, **dementia with Lewy bodies (DLB)** and **frontotemporal dementia (FTD/FTLD)**. **AD and VaD account for the vast majority** and frequently coexist; **DLB is the second commonest neurodegenerative dementia (15–20% of autopsy series)**; **FTLD accounts for 3–20%** and is the **second commonest dementia under age 65**.

2. A useful organising principle is the **cortical versus subcortical** distinction: **cortical dementias** show prominent memory impairment with heteromodal cortical symptoms (aphasia, apraxia, agnosia); **subcortical dementias** show prominent executive dysfunction, milder memory deficit, perseveration, mood change, gait disturbance and extrapyramidal signs.

B. ALZHEIMER'S DISEASE

3. Brain areas: earliest involvement of the **entorhinal cortex and hippocampus (medial temporal lobe)**, spreading to **temporoparietal association cortex** and **posterior cingulate/precuneus**, with relative sparing of primary motor and sensory cortex until late. **Severe temporal lobe atrophy and hippocampal or insular hyperintensities** are typical on MRI.

4. Clinical features: insidious onset with **amnestic syndrome of the hippocampal type** — impaired encoding and storage with poor benefit from cueing — followed by **language, visuospatial and executive decline**, then apraxia and agnosia. **Apathy is the commonest NPS.** Motor signs and falls are late.

5. Imaging signature: HMPAO-SPECT shows temporoparietal hypoperfusion; **FDG-PET shows temporoparietal and posterior cingulate hypometabolism**; **PIB-PET amyloid imaging is highly sensitive and specific, even at preclinical stages**.

C. VASCULAR DEMENTIA

6. Brain areas depend on vessel size. Large-vessel disease produces cortical infarcts in strategic locations: **frontal lobe** (aphasia, apraxia, disinhibition, apathy), **hippocampus and basal forebrain** (amnesia), **gyrus angularis** (construction problems), **parietal lobe** (alexia, agraphia) — giving a **cortical type of dementia**.

7. Small-vessel disease produces subcortical infarcts in the **thalamus, caudate nucleus and internal capsule**, disrupting **specific frontal–subcortical circuits or non-specific thalamocortical circuits**, producing **executive dysfunction, attention deficit, apathy and personality change** — a **subcortical type of dementia**. **Subcortical ischaemic vascular disease (SIVD)** — the **lacunar state and Binswanger's disease** — is the **commonest subtype (82.2% small-vessel versus 25.9% large-vessel in one study)**.

8. Clinical course: classically **abrupt onset with stepwise deterioration and a fluctuating course with plateaus** in cortical VaD; but **60% have slow, less abrupt onset and 80% slow progression** with or without stepwise decline. SIVD causes **slow, subacute-onset dementia with executive dysfunction, impaired attention and slowed processing speed, with comparatively mild memory deficit**.

9. Motor signature: "lower-half parkinsonism" with **hesitation, marche à petit pas and diminished step height**. **Crucially, the triad of dementia, urinary incontinence and gait disturbance is more often produced by small-vessel VaD than by normal-pressure hydrocephalus** — a favourite examiner point.

10. Imaging: basal ganglionic and thalamic hyperintense foci, thromboembolic infarcts, confluent white matter and irregular periventricular hyperintensities (leukoencephalopathy). **Leukoencephalopathy involving at least 25% of total white matter is required** to diagnose small-vessel cerebrovascular disease. SPECT shows **multiple focal deficits**.

D. DEMENTIA WITH LEWY BODIES

11. Brain areas and pathology: α-synuclein-positive Lewy bodies and Lewy neurites, with **presynaptic α-synuclein aggregates** thought to be the synapto-toxic species. Three distribution patterns: **brainstem-predominant (PDD phenotype)**, **cerebral cortex-predominant (DLB phenotype)** and **transitional**. **Concomitant AD pathology is present in 90% of Lewy body disease cases**, and there is **relative preservation of medial temporal lobe structures** on MRI.

12. Core clinical features: fluctuating cognition with pronounced variation in attention and alertness;

recurrent vivid visual hallucinations; REM sleep behaviour disorder, which may precede cognitive decline; and spontaneous parkinsonism. The neuropsychological profile is **dysexecutive**, with **prominent visuospatial impairment even early**, while amnesia may be mild initially.

13. Imaging signature: reduced dopamine transporter uptake in basal ganglia on SPECT/PET; **abnormal (low) uptake on 123I-MIBG myocardial scintigraphy**; **reduced occipital activity on perfusion/metabolism scans** (contrasting with posterior parietotemporal reduction in AD); and the **posterior cingulate island sign on FDG-PET**, which is sensitive and specific for DLB. **EEG shows prominent posterior slow-wave activity with periodic pre-alpha/theta fluctuations.**

E. FRONTOTEMPORAL LOBAR DEGENERATION

14. Brain areas: relatively circumscribed, often asymmetric frontal and anterior temporal atrophy. **bvFTD** — frontal and/or anterior temporal cortex with later basal ganglia involvement; **semantic dementia** — anterior temporal lobe (left or right variant); **progressive non-fluent aphasia — left inferior frontal lobe, anterior insula and basal ganglia**. SPECT shows **diminished anterior perfusion**.

15. Clinical features by variant: bvFTD (≥70%) — early, profound change in **personality and social conduct** with **disinhibition, apathy, social withdrawal, loss of empathy, hyperorality and dietary change, diminished insight, neglect of hygiene, mental rigidity, perseveration and stereotypic behaviour**, with **relative preservation of memory, spatial skills and praxis**. **Semantic dementia (~15%)** — loss of word and object meaning with fluent, empty speech. **PNFA (~10%)** — **non-fluent effortful speech, agrammatism, phonological paraphasia, anomia**, with preserved single-word comprehension early.

16. Distinguishing epidemiology: onset most commonly between 45 and 65, **equal gender distribution**, **family history in ~40% of first-degree relatives**, and **faster cognitive and functional decline with shorter survival than AD** — about **9 years in FTLD-U and tau-positive groups but under 3 years in FTLD-MND**.

F. COMPARATIVE SUMMARY

17. The red flags that should arouse suspicion of non-Alzheimer dementia, most salient early in the course: **prominent executive dysfunction, marked speech disturbance, marked behavioural change (disinhibition, euphoria, stereotyped behaviour, apathy, self-neglect), frequent falls, vivid visual hallucinations, focal neurological deficit, extrapyramidal signs, vertical gaze limitation, and asymmetric apraxia or dystonia**.

Master Comparison Table

Feature	Alzheimer's disease	Vascular dementia	Dementia with Lewy bodies	Frontotemporal dementia
Share of dementias	50–60%; commonest	15–25%	15–20% (autopsy)	3–20%; 2nd commonest under 65
Typical age at onset	>65, rising steeply with age	>65, follows vascular events	50–90	**45–65**
Brain areas	Entorhinal cortex, **hippocampus**, temporoparietal association cortex, posterior cingulate	**Large vessel:** frontal, hippocampus, angular gyrus, parietal. **Small vessel:** thalamus, caudate, internal capsule, periventricular white matter	**Cortical + brainstem** Lewy bodies; occipital hypometabolism; **medial temporal lobe relatively preserved**	**Frontal and anterior temporal**, asymmetric; left inferior frontal/insula in PNFA
Pathology	Amyloid plaques, **neurofibrillary tangles (tau)**	Infarcts, lacunes, leukoaraiosis, microbleeds	**α-synuclein Lewy bodies**; AD pathology co-exists in 90%	**TDP-43 proteinopathy 50–60%, tauopathy 35–45%**
First symptom	**Episodic memory loss**	Executive dysfunction / focal deficit	**Fluctuating attention, visual hallucinations, RBD**	**Personality and social conduct change** or language
Memory	**Prominent, early**	Milder in small-vessel type	Mild early; **better delayed recognition** than AD	**Relatively preserved early**
Executive function	Later	**Prominent early**	Prominent (dysexecutive)	**Prominent early**
Visuospatial	Moderate, later	Variable	**Prominent even early**	Preserved early
Hallucinations	Late, uncommon	Uncommon	**Recurrent, vivid, well-formed — up to 80%**	Uncommon
Motor features	Late	**Lower-half parkinsonism, marche à petit pas**, focal signs	**Parkinsonism in ~75%**, symmetrical, less levodopa-responsive	Late parkinsonism; MND in a subset
Course	Slow, steady	**Stepwise** (cortical) or slow (subcortical)	Fluctuating; **6–10 years**	**Faster than AD**; ~9 years, <3 years if MND
Key imaging	Temporoparietal hypoperfusion; **PIB-PET positive**	Leukoencephalopathy **≥25% of white matter**; multiple focal SPECT deficits	**Low DAT uptake; abnormal MIBG; cingulate island sign; occipital hypometabolism**	**Anterior (frontotemporal) hypoperfusion**; focal asymmetric atrophy
Drug caution	—	Antithrombotics if lobar microbleeds	**Severe neuroleptic sensitivity**	Enhanced extrapyramidal sensitivity to antipsychotics

Latest Updates (2023–Present)

- **Biological rather than clinical definition of AD**: the **2024 Alzheimer's Association revised criteria** define AD by **Core 1 biomarkers** (amyloid PET, CSF Aβ42/40, **p-tau217**, p-tau181, p-tau231) and **Core 2 biomarkers** (tau PET, MTBR-tau243), with clinical staging (Stages 1–6) run in parallel. Clinical phenotype no longer defines the disease.
- **Plasma biomarkers now aid differential diagnosis** as well as detection: **p-tau217 is highly specific for AD pathology**, while **elevated NfL with negative amyloid** points away from AD toward FTD, vascular or other neurodegeneration, and **GFAP** adds an astroglial/neuroinflammatory dimension. The **Alzheimer's Association Clinical Practice Guideline (2025)** formalises their use in specialist settings.
- **Vascular cognitive impairment (VCI)** has displaced "vascular dementia" as the preferred umbrella, spanning **vascular MCI to VaD and mixed pathology**; **VASCOG and the harmonised VICCCS criteria** have superseded the older **NINDS-AIREN and ADDTC** criteria, which had good specificity (excluding ~90% of AD) but **only modest sensitivity (50–70%)** and frequently misclassified mixed dementia.
- **DLB diagnosis** continues to be underdetected; the **fourth consensus criteria (McKeith et al.)** gave increased weighting to **RBD and 123I-MIBG scintigraphy**, and current work focuses on **α-synuclein seed amplification assays (SAA/RT-QuIC in CSF and skin)**, which achieve high diagnostic accuracy and are moving toward a **biological definition of Lewy body disease**.
- **FTD genetics and therapeutics:** the three major genes are **MAPT, GRN (progranulin) and C9orf72**. The **Phase 3 INFRONT-3 trial of latozinemab in FTD-GRN (2025) failed its clinical primary endpoint** despite raising

plasma progranulin, and the programme was discontinued — reinforcing that **FTD treatment remains symptomatic**. Gene-replacement trials (**PROCLAIM, ASPIRE-FTD, upliFT-D**) continue.

- **Mixed pathology is the rule, not the exception**, in the very old; **autopsy series consistently show mixed AD plus vascular pathology outnumbering pure AD**, and **LATE (limbic-predominant age-related TDP-43 encephalopathy)** is now recognised as a distinct, common amnestic mimic of AD in those over 80.

Current Practice Update

Area	Textbook position	Current evidence and guidelines (2023 onward)
Defining AD	Clinical criteria (NINCDS-ADRDA, DSM) with biomarker support	**2024 criteria: biological definition** by Core 1 and Core 2 biomarkers
VaD nomenclature	Vascular dementia; NINDS-AIREN and ADDTC criteria	**Vascular cognitive impairment (VCI)** spectrum; **VASCOG/VICCCS** criteria
DLB confirmation	Clinical core features plus DAT-SPECT and MIBG	**α-synuclein seed amplification assays** approaching biological diagnosis
FTD treatment	Symptomatic — SSRIs, trazodone, bupropion for apathy	Unchanged; **INFRONT-3 (2025) negative**, no disease-modifying therapy available
Old-age amnestic syndrome	Attributed to AD or mixed disease	**LATE (TDP-43)** recognised as a common independent cause after age 80

Exam One-Liners

- **AD** = medial temporal/hippocampal + temporoparietal → **amnesia first**.
- **VaD large-vessel** = cortical infarcts → cortical dementia; **small-vessel** = thalamus/caudate/internal capsule → subcortical dementia.
- **SIVD is the commonest VaD subtype**; leukoencephalopathy must involve **≥25% of white matter**.
- The **dementia + incontinence + gait triad is commoner in small-vessel VaD than in NPH**.
- **DLB** = fluctuation + visual hallucinations + RBD + parkinsonism, with **occipital hypometabolism and cingulate island sign**.
- **DLB spares the medial temporal lobe** — a key MRI contrast with AD.
- **FTD** = frontal/anterior temporal, **onset 45–65**, family history in 40%, **behaviour or language first**.
- **bvFTD 70%, semantic dementia 15%, PNFA 10%**.
- **FTLD survival ~9 years; under 3 years with motor neurone disease.**
- Non-AD red flags: **early executive/behavioural change, falls, visual hallucinations, extrapyramidal signs, vertical gaze palsy, asymmetric apraxia**.

QUESTION 7

Neurodegenerative disorders associated with Tau, Progranulin or TDP-43 pathology — pathology, presentation, course, prognosis and treatment.

15–20 High-Yield Points

A. OVERARCHING CONCEPT

1. These three molecules define the **frontotemporal lobar degeneration (FTLD) spectrum** and its close relatives. **FTLD is neuropathologically, genetically and clinically heterogeneous**, and the term FTLD is preferred to "dementia" because **patients may have progressive neurological dysfunction for substantial periods before meeting criteria for a dementia syndrome**.

2. Histopathological dichotomy: FTLD divides broadly into **FTLD-tauopathy** and **FTLD-U/TDP-43** types on immunohistochemistry. **More than 95% of FTLD cases are either TDP-43 proteinopathies (50–60%) or tauopathies (35–45%)**, with dementia lacking distinctive histopathology (DLDH) and TDP-43-negative ubiquitinated inclusions making up the remaining **5%**.

3. Historical evolution: FTLD was originally split into **tau-positive pathology with or without Pick's bodies** and **dementia lacking distinctive histopathology (DLDH)**. Improved immunohistochemistry showed that most "DLDH" cases actually contain **ubiquitin-positive, tau-negative intraneuronal inclusions** — some with motor neurone disease (**FTLD-MND**) and some without (**FTLD-U**).

4. TDP-43 (TAR DNA-binding protein 43) was then identified as the **major pathogenic protein underlying sporadic and familial FTLD-U and FTLD-MND, and also amyotrophic lateral sclerosis** — the discovery that unified the FTD–ALS spectrum.

B. GENETICS

5. Family history of dementia is present in about 40% of first-degree relatives. About **10% of familial and 0–3% of sporadic cases** are linked to specific mutations.

6. Tau route: many mutations occur in the **microtubule-associated protein tau (MAPT) gene on chromosome 17**, collectively termed **FTD with parkinsonism linked to chromosome 17 (FTDP-17)** in familial cases with parkinsonism.

7. Progranulin route: mutations in the **progranulin (GRN/PRGN) gene on chromosome 17** cause **TDP-43 proteinopathy**. Progranulin mutations are **loss-of-function**, reducing progranulin in plasma and CSF with **over 90% penetrance** — the basis for progranulin-elevating therapeutic strategies.

8. The **valosin-containing protein (VCP) gene** also produces TDP-43 pathology, causing the rare autosomal dominant **inclusion body myopathy with Paget's disease of bone and FTD (IBMPFD)**. **C9orf72** hexanucleotide repeat expansion is the third major gene, linking FTD and ALS.

C. CLINICO-PATHOLOGICAL CORRELATION

9. Although overlap prevents reliable prediction, the clinical presentation suggests the likely pathology: **semantic dementia and FTLD-MND are usually TDP-43 proteinopathies; progressive non-fluent aphasia (and CBD/PSP) is nearly always FTLD-tauopathy; and behavioural-variant FTD is evenly split between tauopathy and TDP-43**. This **tau–TDP-43 dichotomy underpins strategies for pathology-targeted disease-modifying therapy**.

D. PRESENTATION — THE THREE FTLD VARIANTS

10. Behavioural-variant FTD (bvFTD, ≥70%) — atrophy of frontal and/or anterior temporal cortex with subsequent basal ganglia involvement. The salient feature is **early and profound alteration in personality and social conduct with relative preservation of memory, spatial skills and praxis**: **disinhibition, apathy, social withdrawal, loss of empathy, hyperorality and dietary change, diminished insight, neglect of personal hygiene, mental rigidity, perseveration, stereotypic and repetitive motor behaviours**. Loss of sympathy and empathy often causes **family members to resent the patient**.

11. Semantic dementia (SD, ~15%) — temporal-variant FTD with loss of word and object meaning; a **right temporal variant** with asymmetric right temporal atrophy is the rare hemispheric counterpart. **Prosopagnosia** is characteristic of the right temporal form.

12. Progressive non-fluent aphasia (PNFA, ~10%) — atrophy of **left inferior frontal lobe, anterior insula and basal ganglia**, with **progressively worsening non-fluent spontaneous speech, shortened phrase length, agrammatism, phonological paraphasia and anomia**, plus stuttering, speech apraxia, impaired repetition, alexia and agraphia. **Single-word comprehension and object knowledge are preserved early but syntactic relationships**

are impaired. Executive function and working memory are often impaired; patients may be impulsive, apathetic or depressed but are **generally appropriate and many retain insight**.

13. FTD phenocopy meets bvFTD criteria but runs a **very slow course over decades** with slow cognitive progression and **often normal MRI and PET**.

E. RELATED TAUOPATHIES

14. Progressive supranuclear palsy (PSP) — a tauopathy with **marked midbrain atrophy, neurofibrillary tangles or neuropil threads in basal ganglia and brainstem, and tau-positive astrocytes**. The hallmark is **vertical supranuclear gaze palsy with disproportionate limitation of downward gaze** (contrast with the upgaze limitation of normal ageing and PD), occurring with **intact oculocephalic reflexes**. The earliest sign is **slowing of voluntary downward saccades**. MRI shows the **"hummingbird sign"** (dorsal midbrain atrophy) or **"eye of the tiger" sign**; PET/SPECT shows frontal hypoactivity.

15. Corticobasal degeneration (CBD) — a tauopathy overlapping FTD and PSP, with **asymmetric frontoparietal cortical degeneration, severe substantia nigra neuronal loss, ballooned achromatic cells, tau-positive astrocytic plaques, neurofibrillary tangles and neuropil threads**. Presents in the **sixth or seventh decade** with **(i) unilateral levodopa-unresponsive parkinsonism; (ii) asymmetrical rigidity, dystonia or focal myoclonus; (iii) cortical signs — asymmetric apraxia, cortical sensory loss, hemineglect, alien limb phenomenon; and (iv) late-onset cognitive impairment**. **Diagnostic sensitivity is only 30%**, and the corticobasal syndrome commonly proves at autopsy to be **AD, Pick's disease, PSP or prion disease**.

F. COURSE AND PROGNOSIS

16. FTLD onset is most commonly between 45 and 65, though it may present before 30 or in old age; **incidence rises with age (2.2 per 100,000 at 40–49, 3.3 at 50–59, 8.9 at 60–69)** with **equal gender distribution**. It is the **second commonest dementia under 65 after AD** and widely regarded as the **third commonest neurodegenerative dementia overall**.

17. Disease duration is about nine years in FTLD-U and tau-positive groups, but significantly shorter — under three years — in FTLD-MND. Age- and education-adjusted autopsy-confirmed studies show **FTLD carries shorter survival and faster cognitive and functional decline than AD**. Over time the three variants **converge into global cognitive impairment with motor deficits including parkinsonism and motor neurone disease**.

18. PSP prognosis is poor: progression to a **chairbound state at a median of five years** and **death at a median of seven years**, usually from the sequelae of **falls or dysphagia**.

G. TREATMENT

19. Management is multifaceted with no disease-modifying therapy. Caregiver education and support and optimisation of non-pharmacological measures are paramount. Pharmacotherapy is **symptomatic**, aimed at difficult behaviours and cognitive impairment.

20. Drug choices: consistent with the **selective vulnerability of serotonergic neurons in FTLD**, systematic review supports **SSRIs and trazodone** for modest improvement in behavioural symptoms of bvFTD — **impulsivity, disinhibition, repetitive stereotyped behaviour and obsessive-compulsive behaviour**; trazodone's use is limited by **sedation**. **Depression is rare in bvFTD but commoner in SD and PNFA and responds to SSRIs. Apathy is generally recalcitrant to pharmacotherapy**, though **bupropion** (with dopaminergic agonist properties) may be considered where apathy coexists with parkinsonism. **Low-dose atypical antipsychotics** may be used cautiously for delusions, severe agitation and aggression, given **enhanced sensitivity to extrapyramidal side effects, somnolence, weight gain and exacerbation of apathy**. **Cholinesterase inhibitors have not been shown useful in PSP**, and **memantine failed in the randomised FTLD trial (Boxer et al.)**. Emerging targets include **tau antibodies, inhibition of tau aggregation with methylene blue derivatives, microtubule-stabilising drugs and tau vaccines**. **PSP and CBD treatment remains supportive, centred on falls prevention and aggressive surveillance and management of dysphagia.**

Latest Updates (2023–Present)

- **The progranulin strategy has suffered a major setback. Latozinemab (AL001)**, a monoclonal antibody blocking sortilin to raise progranulin, **failed its clinical co-primary endpoint in the Phase 3 INFRONT-3 trial (announced October 2025)** in symptomatic FTD-GRN, despite a **statistically significant rise in plasma progranulin**. The open-label extension and continuation study were discontinued. FTD-GRN accounts for **5–10% of FTD cases**.
- **Gene-replacement therapy continues** for FTD-GRN: **PROCLAIM, ASPIRE-FTD and upliFT-D** are ongoing AAV-based gene-replacement studies showing early **safety, tolerability and restoration of progranulin levels**; a further six trials target **neuroinflammatory mechanisms in sporadic FTD**. **No licensed disease-modifying**

therapy exists for any FTLD subtype.

- **Tau-targeted therapy remains unsuccessful in the primary tauopathies**: anti-tau antibodies (**tilavonemab, gosuranemab**) failed in PSP, and **tau aggregation inhibitors** have not translated. Attention has shifted to **antisense oligonucleotides against MAPT** and to **C9orf72-directed ASOs** for the FTD–ALS spectrum.
- **Fluid biomarkers have matured for FTLD**: **plasma and CSF neurofilament light chain (NfL)** is now the best-validated marker of **disease activity, progression and phenoconversion in presymptomatic mutation carriers**, and **plasma progranulin below the normal range is diagnostic of GRN mutation**. Cohorts such as **GENFI** and **ALLFTD** have made presymptomatic staging routine.
- **LATE — limbic-predominant age-related TDP-43 encephalopathy** — is now a recognised, common TDP-43 proteinopathy of the **very old (over 80)**, producing an **amnestic syndrome closely mimicking AD** and frequently coexisting with it, contributing to the amyloid-negative amnestic phenotype.
- **Revised diagnostic criteria** have refined the field: **MDS-PSP criteria (2017)** define multiple PSP phenotypes beyond Richardson's syndrome, **Armstrong criteria** define corticobasal syndrome, and the **2011 Rascovsky bvFTD and Gorno-Tempini PPA criteria** remain standard for FTLD.
- **Indian context:** FTD is disproportionately represented in Indian memory clinics because of **younger age at presentation and referral patterns**; genetic testing is limited to research and tertiary centres, and **caregiver burden is exceptionally high** given the working-age onset, loss of family income and the behavioural nature of the illness — relevant to the **Maintenance and Welfare of Parents and Senior Citizens Act** and to disability certification under the **Rights of Persons with Disabilities Act, 2016**, which recognises chronic neurological conditions and dementia.

Current Practice Update

Area	Textbook position	Current evidence and guidelines (2023 onward)
Progranulin-elevating therapy	Advances in pathobiology identify new targets	**INFRONT-3 (2025) failed** its clinical endpoint; latozinemab discontinued
Tau-targeted therapy	Tau antibodies, methylene blue derivatives, microtubule stabilisers, tau vaccines in development	Anti-tau antibodies **failed in PSP**; focus shifted to **MAPT and C9orf72 antisense oligonucleotides**
Biomarkers	Clinical and imaging diagnosis	**NfL** for disease activity and phenoconversion; **low plasma progranulin** diagnostic of GRN mutation
Nosology in the very old	FTLD-U/TDP-43 as an FTD entity	**LATE** recognised as a common TDP-43 amnestic syndrome after age 80
Symptomatic treatment	SSRIs, trazodone, bupropion, cautious atypical antipsychotics	Unchanged and still the standard of care

Exam One-Liners

- **>95% of FTLD = TDP-43 (50–60%) or tau (35–45%)**; remaining 5% DLDH/other.
- **MAPT (chromosome 17) → tau; GRN/progranulin (chromosome 17) → TDP-43; VCP → TDP-43 (IBMPFD); C9orf72 → FTD-ALS.**
- **FTDP-17** = familial FTD with parkinsonism linked to chromosome 17.
- **SD and FTLD-MND → TDP-43; PNFA, CBD, PSP → tau; bvFTD → evenly split.**
- Family history in **40%** of first-degree relatives; **10% of familial cases** have identified mutations.
- Onset **45–65**, equal sex distribution, **survival ~9 years, <3 years with MND**.
- **PSP: vertical supranuclear gaze palsy with impaired downgaze, hummingbird sign, chairbound at 5 years, death at 7.**
- **CBD: alien limb, asymmetric apraxia, levodopa-unresponsive parkinsonism; diagnostic sensitivity only 30%.**
- **SSRIs and trazodone for behaviour; bupropion for apathy with parkinsonism; ChEIs unhelpful; memantine failed.**
- **INFRONT-3 (2025) negative** — no disease-modifying therapy for FTD.

QUESTION 8

Pick’s disease — pathology and treatment.

15–20 High-Yield Points

A. DEFINITION AND POSITION IN NOSOLOGY

1. Pick’s disease is a tau-positive frontotemporal lobar degeneration characterised pathologically by Pick bodies, and is one of the classical **non-reversible degenerative dementias of the central nervous system**, listed alongside Alzheimer’s disease, Lewy body dementia, Parkinson’s disease, Huntington’s disease and progressive supranuclear palsy.

2. Historically, FTLD was divided into **tau-positive pathology with or without Pick’s bodies** and **dementia lacking distinctive histopathology (DLDH)**. Pick’s disease is therefore the **prototype of the tau-positive limb of the FTLD dichotomy**, and today is best regarded as a **pathological diagnosis within FTLD-tauopathy (35–45% of all FTLD)**, most often presenting clinically as **behavioural-variant FTD**.

B. PATHOLOGY

3. Macroscopic: circumscribed, often asymmetric atrophy of the frontal and anterior temporal lobes — the classic “knife-edge” gyral atrophy — with **relative sparing of the posterior two-thirds of the superior temporal gyrus**, the parietal and occipital lobes.

4. Microscopic hallmark: Pick bodies — round, well-circumscribed, argyrophilic, tau-positive intraneuronal cytoplasmic inclusions, most abundant in the **dentate gyrus of the hippocampus and in frontotemporal cortical neurons**.

5. Pick cells (ballooned achromatic neurons) — swollen, pale-staining neurons — are also seen; identical **ballooned achromatic cells occur in corticobasal degeneration**, an important point of overlap.

6. Accompanying features are **severe neuronal loss, superficial laminar (layer II/III) spongiosis and gliosis** in the affected cortex, with subsequent **basal ganglia involvement** as the disease advances.

7. Molecular pathology: Pick’s disease is a **3-repeat (3R) tauopathy**, in contrast to the **4-repeat (4R) tauopathies PSP and CBD** and the mixed 3R/4R tau of Alzheimer’s disease — the single most discriminating molecular fact.

8. Genetics: Pick’s disease is usually **sporadic**, but tau-positive FTLD is linked to **MAPT mutations on chromosome 17 (FTDP-17)**. **A family history of dementia is present in about 40% of first-degree relatives** across FTLD as a whole.

C. CLINICAL PRESENTATION

9. Presentation is **usually that of behavioural-variant FTD**: **early and profound alteration in personality and social conduct with relative preservation of memory, spatial skills and praxis**. Typical behaviours are **disinhibition, apathy, social withdrawal, loss of empathy, hyperorality and dietary change, diminished insight, neglect of personal hygiene, mental rigidity, perseveration, stereotypic behaviour and repetitive motor behaviours**.

10. Loss of sympathy and empathy toward family and friends, with decreased social interest and responsiveness to the needs of others, is characteristic and often leads to **family members resenting the patient** — a distinctive social consequence not seen in AD.

11. Some patients present instead with **progressive non-fluent aphasia**, which is **nearly always an FTLD-tauopathy**; the **corticobasal syndrome** may also prove at autopsy to be Pick’s disease.

12. Onset is most commonly between 45 and 65, with **equal gender distribution**; it is a leading cause of **young-onset dementia**.

D. DIAGNOSIS

13. Diagnosis in life is clinical and radiological; definitive diagnosis is neuropathological. MRI or CT shows focal, asymmetric frontal and anterior temporal atrophy, and **HMPAO-SPECT shows diminished anterior perfusion**, in contrast to the temporoparietal reduction of AD. **FDG-PET shows characteristic frontotemporal hypometabolism.**

14. Exclude the treatable and the mimics: complete blood count, glucose, electrolytes, **calcium and phosphorus (mg/dL)**, liver function tests, **TSH (µIU/mL)**, **vitamin B12 (pg/mL)**, syphilis and HIV serology where suspected, plus structural imaging. Psychiatric illness, late-onset mania, AD with frontal presentation and the **FTD phenocopy syndrome** are the principal differentials.

E. COURSE AND PROGNOSIS

15. Prognosis is poor and worse than AD. Disease duration is about **nine years in tau-positive FTLD groups**, and **FTLD is associated with shorter survival and faster cognitive and functional decline than AD** in age- and education-adjusted autopsy-confirmed studies. Over time patients develop **global cognitive impairment and motor deficits including parkinsonism**, and death is commonly from **aspiration pneumonia, dysphagia complications or falls**.

F. TREATMENT

16. There is no disease-modifying or curative therapy. Management is **multifaceted**, with **caregiver education and support and optimisation of non-pharmacological measures paramount**; pharmacotherapy is aimed at **difficult behaviours and cognitive impairment**.

17. SSRIs and trazodone offer modest benefit for the behavioural symptoms of bvFTD, consistent with the **selective vulnerability of serotonergic neurons in FTLD**. Symptoms that respond include **impulsivity, disinhibition, repetitive stereotyped behaviour and obsessive-compulsive behaviour**. **Trazodone's use may be limited by sedation.**

18. Depression is rare in bvFTD but commoner in semantic dementia and PNFA and is amenable to SSRI treatment. **Apathy is generally recalcitrant to pharmacotherapy** and may even be a relief for caregivers of a severely behaviourally disturbed patient; **bupropion**, with additional dopaminergic agonist properties, can be considered where apathy coexists with parkinsonism.

19. Low doses of atypical antipsychotics may be considered for **delusions, severe agitation and aggression**, but must be used **cautiously owing to enhanced sensitivity to extrapyramidal side effects, somnolence, weight gain and exacerbation of apathy**. **Cholinesterase inhibitors and memantine are not indicated** — memantine failed in the randomised FTLD trial and ChEIs may worsen behaviour in FTD.

20. Supportive care is the mainstay: **structured routine and environmental simplification, safety measures (driving cessation, financial safeguarding, supervision for hyperorality and impulsivity), speech and language therapy for the aphasic variants, dysphagia surveillance and nutritional support, and early advance care planning**. Emerging disease-modifying targets are **tau antibodies, inhibition of tau aggregation with methylene blue derivatives, microtubule-stabilising drugs and tau vaccines**.

Latest Updates (2023–Present)

- **Pick's disease is now formally classified as a 3R tauopathy** within the FTLD-tau group alongside **PSP and CBD (4R)** and **globular glial tauopathy**; cryo-EM structural studies have shown that **each tauopathy has a distinct tau filament fold**, with Pick's disease possessing a unique 3R fold — the structural basis for developing **conformation-specific tau ligands and PET tracers**.
- **Tau PET remains unreliable for non-AD tauopathies**: first-generation tracers such as **flortaucipir bind 3R/4R paired helical filaments of AD but not the 3R Pick fold**, so a negative tau PET does not exclude Pick's disease. Second-generation tracers are in development.
- **Anti-tau therapeutics have repeatedly failed** in the primary tauopathies — **tilavonemab and gosuranemab** in PSP — and attention has shifted to **MAPT-directed antisense oligonucleotides**, which lower tau at the transcript level and are in early-phase trials.
- **Plasma and CSF neurofilament light chain (NfL)** is now the best-validated marker of disease activity and progression across FTLD, useful in distinguishing FTD from primary psychiatric illness and from the slowly progressive **FTD phenocopy syndrome**, which characteristically has normal NfL.
- The **2011 Rascovsky criteria for bvFTD** and **Gorno-Tempini criteria for primary progressive aphasia** remain the operational clinical standard; multicentre cohorts (**GENFI, ALLFTD, ARTFL-LEFFTDS**) have established natural-history and presymptomatic staging data.
- **Indian context:** young-onset dementia including FTLD is under-recognised and frequently first labelled as a **primary psychiatric disorder**, delaying diagnosis by years. Practical implications are large: **loss of earning capacity in the family's main provider**, need for **disability certification under the Rights of Persons with Disabilities Act, 2016** (which includes chronic neurological conditions and mental illness), and access to **caregiver support through ARDSI and the District Mental Health Programme**.

Current Practice Update

Area	Textbook position	Current evidence and guidelines (2023 onward)
Classification	Tau-positive FTLD with or without Pick bodies	**3R tauopathy** with a unique cryo-EM tau filament fold, distinct from 4R PSP/CBD
Imaging diagnosis	Focal frontotemporal atrophy; anterior hypoperfusion on SPECT	Unchanged; **tau PET is not reliable** for 3R Pick pathology
Disease-modifying therapy	Tau antibodies, aggregation inhibitors, microtubule stabilisers, vaccines in development	**Anti-tau antibodies failed in PSP**; focus now on **MAPT antisense oligonucleotides**
Biomarker for progression	Not established	**NfL** validated for disease activity and to exclude the phenocopy syndrome
Symptomatic treatment	SSRIs, trazodone, bupropion; cautious atypical antipsychotics	Unchanged and still the standard of care

Exam One-Liners

- Pick's disease = **tau-positive FTLD defined by Pick bodies**.
- **Pick bodies** = round, argyrophilic, tau-positive intraneuronal inclusions, densest in the **dentate gyrus**.
- **Pick cells** = ballooned achromatic neurons — also seen in **corticobasal degeneration**.
- **3R tauopathy** — contrast with **4R PSP and CBD** and mixed 3R/4R AD.
- Macroscopic: **knife-edge frontal and anterior temporal atrophy**, posterior superior temporal gyrus spared.
- Presents as **bvFTD**: disinhibition, apathy, loss of empathy, hyperorality, stereotypies.
- **Onset 45–65**, equal sex distribution, **survival ~9 years**, faster decline than AD.
- SPECT: **anterior (frontotemporal) hypoperfusion**, versus temporoparietal in AD.
- **Treatment: SSRIs and trazodone; bupropion for apathy; low-dose atypicals with caution; ChEIs and memantine not indicated.**
- **Caregiver education and support are paramount** — no disease-modifying therapy exists.

QUESTION 9

Lewy body dementia.

15–20 High-Yield Points

A. DEFINITION, TERMINOLOGY AND SPECTRUM

1. Dementia with Lewy bodies (DLB) is the preferred term for entities previously called the **Lewy body variant of AD, Lewy body dementia, senile dementia of Lewy body type and cortical Lewy body disease**.

2. DLB and Parkinson's disease dementia (PDD) are two ends of one Lewy body disease spectrum. The **"one-year rule"** applies: **PDD** describes dementia developing in established Parkinson's disease, while **DLB** is diagnosed when dementia **precedes or coincides within one year** of motor symptom onset. By the end stage the two are **often indistinguishable clinically and neuropathologically**.

B. EPIDEMIOLOGY

3. DLB is the **second commonest cause of neurodegenerative dementia in older people after AD**, accounting for **~15–20% of autopsy series and 10–15% of dementia cases in population studies**. **Incidence is 32–112 per 100,000 person-years** in those over 65.

4. Onset is between 50 and 90 years, and illness duration varies between 6 and 10 years.

C. PATHOLOGY

5. The defining lesion is the **α-synuclein-containing Lewy body and Lewy neurite**. Recent evidence indicates that **presynaptic α-synuclein aggregates, rather than the Lewy body itself, are synapto-toxic** and cause neurodegeneration.

6. Three patterns of Lewy body distribution: (i) brainstem-predominant — the PDD phenotype; **(ii) cerebral cortex-predominant** — the DLB phenotype; **(iii) transitional**, intermediate between the two. The likelihood of presenting as typical DLB is **directly related to the distribution of Lewy body pathology and inversely related to the severity of AD pathology**.

7. Concomitant AD pathology is present in 90% of Lewy body disease cases. Patients with **marked concurrent neurofibrillary tangle pathology present with an insidious amnestic syndrome more suggestive of AD**, with **fewer cognitive fluctuations, fewer neuropsychiatric symptoms and less parkinsonism**.

8. Most cases are sporadic, but rare autosomal dominant inheritance occurs, with **SNCA and LRRK2 mutations**, which may manifest as any Lewy body phenotype — supporting a single underlying genetic-pathological entity.

D. CORE CLINICAL FEATURES

9. Fluctuating cognition with pronounced variations in attention and alertness — waxing and waning behavioural inconsistency, incoherent speech, variable attention, staring or "zoning out". The **marked amplitude between best and worst performance** distinguishes it from minor day-to-day variation. Endorsement of **at least three of four composite features — daytime drowsiness and lethargy, daytime sleep of ≥2 hours, staring into space for long periods, episodes of disorganised speech — gives a positive predictive value of 83%** for DLB over AD. **Fluctuations best predict DLB when present early.**

10. Recurrent vivid visual hallucinations occur in up to 80% — typically **well-formed, detailed, three-dimensional mute images of people, children or animals**. Caregivers may under-report them, so **patient self-report is useful**. Their presence marks **cortical Lewy body pathology and greater cortical cholinergic deficit, and predicts good response to cholinergic therapy**. **Misidentification delusions are the commonest delusional type**, followed by persecutory/paranoid, **phantom boarder** and abandonment delusions.

11. REM sleep behaviour disorder (RBD) — a parasomnia with **loss of REM atonia**, so patients **act out vivid, frightening dreams**: talking or shouting in sleep, striking the bed partner, falling out of bed. **History must come from the bed partner.** It **precedes motor, cognitive and neuropsychiatric manifestations by years or decades and curiously improves as disease progresses**. It is **highly specific for synucleinopathy (DLB, PD, MSA)** and rare in AD and primary tauopathies.

12. Spontaneous parkinsonism occurs in about **75%**, is **less pronounced and less levodopa-responsive than idiopathic PD**, so **only one cardinal feature (bradykinesia, rigidity or resting tremor) is required** for diagnosis.

E. SUPPORTIVE FEATURES AND COGNITIVE PROFILE

13. Supportive clinical features: severe sensitivity to antipsychotics, postural instability, repeated falls, syncope or transient unresponsiveness, severe autonomic dysfunction (orthostatic hypotension, urinary

incontinence, constipation), hypersomnia, systematised delusions, hallucinations in other modalities, and apathy, anxiety and depression.

14. Neuroleptic sensitivity: about **50% of DLB patients do not react adversely**, so tolerance does not exclude the diagnosis, but a **positive history of severe sensitivity is strongly suggestive**. Reactions range from **sedation, increased confusion and worsening parkinsonism** to **irreversible parkinsonism, impaired consciousness and marked autonomic disturbance**.

15. Cognitive profile: a **"dysexecutive" syndrome with prominent executive, attentional and visuospatial dysfunction**, with **visuospatial impairment prominent even early**. Amnesia may not be prominent initially; **memory impairment does not preclude DLB**, and patients with pure Lewy body disease have **similar free recall but better delayed recognition memory than AD**, indicating a **retrieval rather than storage deficit**.

F. DIAGNOSTIC CRITERIA AND BIOMARKERS

16. Essential: progressive cognitive decline with reduced social and occupational functioning. **Probable DLB = ≥2 core clinical features, or 1 core feature plus ≥1 indicative biomarker. Possible DLB = 1 core feature with no indicative biomarker, or ≥1 indicative biomarker with no core feature. DLB is less likely if** there is a history of stroke, another illness or brain disorder sufficient to explain the picture, or if **parkinsonism is the only core feature and appears first at a severe stage of dementia**.

17. Indicative biomarkers: low dopamine transporter uptake in basal ganglia on SPECT/PET; **abnormal (low uptake) 123I-MIBG myocardial scintigraphy**, which quantifies postganglionic sympathetic cardiac innervation and discriminates DLB from AD especially in milder cases (**MMSE >21**); and **polysomnographic confirmation of REM sleep without atonia**. **Supportive biomarkers: relative preservation of medial temporal lobe structures** on CT/MRI; **generalised low uptake with reduced occipital activity ± the cingulate island sign on FDG-PET**; and **prominent posterior slow-wave EEG activity with periodic pre-alpha/theta fluctuations**.

18. Earlier criteria had high specificity (80–100%) but limited sensitivity (35–80%); the third consensus criteria (2005) improved sensitivity, but **detection remains suboptimal, with many cases missed or misdiagnosed as AD**. The **revised fourth consensus criteria clearly separate clinical features from diagnostic biomarkers, with increased weighting given to RBD and MIBG scintigraphy**. **CSF α-synuclein may be low in DLB versus AD**, while **Aβ, tau and p-tau help determine concomitant AD pathology**; **amyloid deposition has little value in differentiating DLB from AD**, though greater amyloid load predicts faster decline in DLB.

G. MANAGEMENT

19. Management addresses **motor, cognitive, psychiatric and autonomic dysfunction**, and the clinician must be **mindful of the tension between improving one symptom at the expense of worsening another** — for example **aggravating hallucinations with levodopa**. **Conventional neuroleptics are best avoided**; **quetiapine or clozapine** may be used judiciously. **Cholinesterase inhibitors are effective and relatively safe for psychiatric and cognitive symptoms in DLB**, with mainly gastrointestinal side effects, although **DLB patients may be more susceptible to bradyarrhythmic effects because of autonomic dysfunction**.

20. Memantine produces cognitive and global benefits in DLB but without significant improvement in psychiatric symptoms, and **case reports describe worsening delusions and hallucinations with memantine** in DLB — caution is warranted. Preliminary subgroup analysis suggests a **more pronounced global response in PDD than DLB**. **Autonomic management** uses **fludrocortisone or midodrine** for symptomatic postural hypotension; **RBD is managed with a safe sleep environment, melatonin and, where necessary, clonazepam**; and **falls prevention and dysphagia surveillance** are essential.

Latest Updates (2023–Present)

- **α-Synuclein seed amplification assays (SAA/RT-QuIC)** have transformed diagnosis. In **CSF** they achieve **>90% sensitivity and specificity** for Lewy body pathology, and **skin biopsy SAA (2024 JAMA)** detected phosphorylated α-synuclein in **around 95% of patients with synucleinopathies** — moving the field toward a **biological definition of Lewy body disease** paralleling the biological definition of AD, and enabling detection at the **prodromal RBD stage**.
- **Isolated RBD is now formally recognised as prodromal synucleinopathy**, with **around 75–90% phenoconverting to DLB, PD or MSA over 10–15 years**; this has made RBD clinics the principal recruitment route for **neuroprotection trials** in Lewy body disease.
- **Research criteria for prodromal DLB (McKeith et al.)** define three syndromes — **MCI with Lewy bodies, delirium-onset DLB and psychiatric-onset DLB** — allowing diagnosis before dementia, with **MCI-LB criteria** now operationalised.
- **Therapeutics remain symptomatic. Neflamapimod (p38α kinase inhibitor)** showed cognitive benefit in the **RewinD-LB Phase 2b trial (2025)** in DLB patients without amyloid co-pathology and is progressing to Phase 3.

Ambroxol (glucocerebrosidase chaperone) and **α-synuclein immunotherapies** are in trials. **No disease-modifying therapy is yet licensed.**

- **Antipsychotic caution has been reinforced** — **pimavanserin** remains licensed only for Parkinson's disease psychosis; the **HARMONY** trial showed relapse prevention in dementia-related psychosis but did not yield a DLB indication, and all antipsychotics carry the **boxed warning for increased mortality in elderly patients with dementia-related psychosis**.
- **Practical Indian points:** DLB is systematically **under-diagnosed and frequently mislabelled as AD or late-onset psychosis**, exposing patients to conventional antipsychotics such as **haloperidol** with potentially catastrophic neuroleptic sensitivity reactions. **DAT-SPECT and MIBG scintigraphy are available only in selected tertiary centres**, so **clinical recognition of the four core features — especially a careful bed-partner history of RBD — remains the single most important diagnostic step in Indian practice.**

Current Practice Update

Area	Textbook position	Current evidence and guidelines (2023 onward)
Diagnostic confirmation	Core clinical features plus DAT-SPECT, MIBG, polysomnography	**α-synuclein seed amplification assays (CSF and skin)** achieve >90% accuracy and enable prodromal diagnosis
Prodromal disease	RBD noted as an early antecedent marker	**Isolated RBD = prodromal synucleinopathy**; formal **prodromal DLB and MCI-LB criteria** now defined
Disease modification	None available	Still none licensed; **neflamapimod (RewinD-LB, 2025)** the most advanced candidate
Antipsychotics	Avoid conventional agents; use quetiapine or clozapine judiciously	Unchanged and reinforced; **pimavanserin remains restricted to PD psychosis**
Memantine	Cognitive and global benefit, no psychiatric benefit; case reports of worsening psychosis	Position unchanged; **ChEIs remain first-line** for cognition and psychosis in DLB

Exam One-Liners

- **DLB = 2nd commonest neurodegenerative dementia** (15–20% autopsy, 10–15% population); duration **6–10 years**.
- **One-year rule** separates DLB from PDD.
- Four core features: **fluctuating cognition, visual hallucinations, RBD, spontaneous parkinsonism**.
- **Probable DLB = 2 core features, or 1 core + 1 indicative biomarker.**
- Fluctuation composite (**drowsiness, ≥2 h daytime sleep, staring, disorganised speech**) — **3 of 4 gives PPV 83%**.
- Visual hallucinations in **80%**; predict **good response to cholinergic therapy**.
- **RBD is the most specific antecedent biomarker of synucleinopathy.**
- Parkinsonism in **75%**; only **one cardinal feature required**.
- **50% show no neuroleptic sensitivity** — tolerance does not exclude DLB.
- Indicative biomarkers: **low DAT uptake, abnormal MIBG, REM sleep without atonia**.
- **Medial temporal lobe preserved**; **occipital hypometabolism and cingulate island sign** on FDG-PET.
- **ChEIs effective and safe; memantine helps cognition but not psychiatric symptoms and may worsen hallucinations.**

QUESTION 10

Normal pressure hydrocephalus.

15–20 High-Yield Points

A. DEFINITION AND CONCEPT

1. Normal pressure hydrocephalus (NPH) is a **chronic communicating hydrocephalus** with **ventricular enlargement in the presence of normal mean cerebrospinal fluid opening pressure**, presenting with a characteristic clinical triad and being one of the few **potentially reversible causes of dementia**.

2. It is classified as **idiopathic (iNPH)** or **secondary**, the latter following **subarachnoid haemorrhage, meningitis, head injury or intracranial surgery**. It appears in every standard list of **potentially reversible dementias** and of **causes of parkinsonism with cognitive impairment in the elderly**.

B. AETIOLOGY AND PATHOPHYSIOLOGY

3. The classical model is one of **impaired CSF absorption at the arachnoid granulations with altered CSF flow dynamics**, described through **bulk-flow theory**; **CSF flow and CSF flow rate abnormalities** are central to both pathogenesis and diagnosis.

4. Ventricular enlargement transmits **periventricular tissue stress**, producing **ischaemia and disruption of the periventricular white matter**, particularly the **corona radiata fibres to the medial legs** — explaining why **gait is affected first and most severely**, and why the frontal-subcortical circuits subserving **executive function and bladder inhibition** are next affected.

5. Comorbid cerebrovascular disease is very common in this age group and both **contributes to the clinical picture and limits the response to shunting** — an essential caveat when counselling patients.

C. CLINICAL PRESENTATION

6. Hakim and Adams' triad: **gait disturbance, cognitive impairment (dementia) and urinary incontinence** — traditionally remembered as **"wet, wacky and wobbly"**. **Gait disturbance usually appears first and is the symptom most likely to improve with treatment.**

7. The gait is classically "magnetic" — broad-based, shuffling, with reduced step height and length, **start hesitation, freezing and difficulty turning (en bloc turning)**, but with **preserved arm swing and no tremor**, distinguishing it from Parkinson's disease. NPH is also listed among the **causes of secondary parkinsonism**.

8. Cognitive impairment is subcortical/frontal — psychomotor slowing, inattention, executive dysfunction and apathy — with **relatively preserved cortical functions** (language, praxis, recognition), distinguishing it from AD.

9. Urinary symptoms begin as **urgency and frequency** and progress to **urge incontinence**, and later to an **indifferent incontinence** reflecting frontal disinhibition.

D. DIFFERENTIAL DIAGNOSIS

10. The crucial differential is **subcortical ischaemic vascular disease (SIVD)**: the textbook makes the point explicitly that **the triad of dementia, urinary incontinence and gait disturbance is more often produced by small-vessel vascular dementia than by NPH**. Other differentials include **Parkinson's disease and atypical parkinsonian syndromes (PSP, MSA), Alzheimer's disease, DLB, cervical spondylotic myelopathy, chronic subdural haematoma and hydrocephalus ex vacuo from cerebral atrophy**.

E. DIAGNOSTIC MODALITIES

11. CT and MRI demonstrate **ventriculomegaly disproportionate to sulcal atrophy**, quantified by an **Evans index >0.3** (maximum frontal horn width divided by maximum inner skull diameter), with **periventricular hyperintensities, upward bowing of the corpus callosum, a callosal angle below 90 degrees, and a CSF flow void or increased aqueductal flow**.

12. Disproportionately enlarged subarachnoid space hydrocephalus (DESH) is a key morphological pattern — **enlarged Sylvian fissures and basal cisterns with tight, compressed high-convexity and medial parietal sulci** — and strongly supports the diagnosis.

13. Neuropsychological testing documents the **subcortical/frontal profile**, provides the objective baseline against which CSF-drainage response is measured, and helps identify coexisting AD or vascular disease.

F. PROGNOSTIC (SUPPLEMENTARY) TESTS — PREDICTING SHUNT RESPONSE

14. Lumbar puncture (the "tap test") — removal of a **large volume of CSF (30–50 mL)** with **objective gait and**

cognitive testing before and after. It has **good positive but poor negative predictive value**, so a negative tap test does not exclude a shunt-responsive patient.

15. Extended lumbar drainage (lumbar drain trial) — continuous external drainage over **2–3 days** with serial testing; this has **the highest predictive accuracy** of the supplementary tests. **Lumbar infusion test (intrathecal infusion test)** measures **CSF outflow resistance (Rout)**; **intracranial pressure monitoring** detects **B-waves**.

16. Additional modalities include **phase-contrast MRI** for aqueductal CSF stroke volume, **magnetic resonance spectroscopy**, **SPECT** and **PET**. **Grading uses the idiopathic normal-pressure hydrocephalus grading scale (iNPHGS)**, which scores each of the three triad domains.

17. The value of provocative CSF removal is quantitative: predictive power for a positive shunt response rises progressively as one moves from **clinical and imaging information alone → plus tap test → plus infusion testing → plus external lumbar drainage**.

G. TREATMENT

18. Shunt surgery is the definitive treatment. Options are **ventriculoperitoneal shunting (the standard), ventriculoatrial shunting and lumboperitoneal shunting**; **endoscopic third ventriculostomy** has a limited role in communicating NPH. **Programmable (adjustable) valves** allow non-invasive post-operative pressure adjustment and reduce the risk of overdrainage; **anti-siphon devices** are added for the same reason. **Drug therapy (acetazolamide) has only a marginal, temporising role.**

19. Patient selection and shared decision-making are paramount — weighing symptom duration, degree of comorbidity (especially cerebrovascular disease and coexisting AD), frailty, anaesthetic risk and realistic expectations of benefit.

20. Risks and complications: shunt infection and **shunt obstruction** are the principal shunt-related problems, along with **overdrainage causing subdural effusion or haematoma, seizures, and the need for revision surgery**. **Long-term care and follow-up are mandatory**, with periodic valve setting review and prompt imaging for any deterioration. **Outcome is best for gait, less certain for cognition and continence, and better with shorter symptom duration and less comorbidity.**

Latest Updates (2023–Present)

- **The PENS trial (Placebo-Controlled Effectiveness in iNPH Shunting; NEJM, December 2025)** is the landmark update. This **international, multicentre, double-blind, randomised, placebo-controlled trial** enrolled **99 patients across 17 centres in the USA, Canada and Sweden**, randomising participants after shunt insertion to an **open versus a placebo (closed) valve setting**. **Gait velocity increased by 0.23 m/s with an open shunt versus 0.03 m/s with placebo — a treatment difference of 0.21 m/s (95% CI 0.12 to 0.31; p<0.001)**. The **DSMB stopped enrolment early** at the second interim analysis for efficacy.
- **What PENS did and did not show: 80% of the open-shunt group achieved meaningful improvement versus 24% of the placebo group**, with **better balance and fewer falls**. **Cognition and incontinence did not improve significantly at 3 months** — a nuance that should be quoted, because it aligns exactly with the classical teaching that **gait is the symptom most likely to respond**.
- **The 2024 Cochrane review on shunting for iNPH** had concluded that further trials were needed to increase certainty; **PENS has now supplied that evidence** and has effectively ended the call for a moratorium on shunt surgery.
- **Guidelines:** the **AAN/AANS/CNS guideline on iNPH response to shunting and predictors of response was reaffirmed in July 2024**; the **Japanese iNPH clinical practice guidelines (3rd edition, 2020)** remain the most detailed, though they do not compare ventriculoperitoneal, lumboperitoneal and ventriculoatrial shunting directly — a gap the ongoing **RASHOMON registry study** is designed to fill. **SINPHONI-2** had earlier shown **lumboperitoneal shunting to be comparable to ventriculoperitoneal shunting**.
- **Epidemiology has been revised upward.** Population-based MRI studies indicate iNPH affects **up to 1.5% of people in their late 60s and as many as 7.7% of adults over 80–86**, making it far commoner and more under-diagnosed than previously assumed, and reinforcing the case for active case-finding in falls and gait clinics.
- **Indian context:** iNPH is systematically **under-recognised** and often labelled as “ageing gait”, vascular parkinsonism or AD. Given that **neurosurgical shunting is widely available across Indian tertiary centres and is relatively inexpensive**, the diagnostic bottleneck is clinical suspicion, not treatment access. Every older adult presenting with a **gait disorder plus cognitive slowing** — a very common presentation to **NPHCE district geriatric clinics and falls services** — deserves an **Evans index measured on the CT that has usually already been done**.

Current Practice Update

Area	Textbook position	Current evidence and guidelines (2023 onward)
Evidence for shunting	Treatment of choice, but efficacy debated and outcome variable	**PENS trial (2025)** provides randomised, placebo-controlled proof: **gait velocity gain 0.21 m/s; 80% vs 24% meaningful improvement**
Which symptom responds	Gait responds best	**Confirmed**: gait and balance improve; **cognition and continence did not improve significantly** in PENS
Prevalence	Uncommon cause of dementia	**Up to 1.5% in late 60s and 7.7% over 86** — substantially under-diagnosed
Shunt type	VP, VA, LP shunts described without direct comparison	**SINPHONI-2**: LP comparable to VP; **RASHOMON registry** is comparing all three prospectively
Patient selection	Supplementary CSF tests improve predictive power	Unchanged and reinforced — **extended lumbar drainage remains the best predictor**

Exam One-Liners

- NPH = **ventriculomegaly with normal mean CSF opening pressure**.
- **Hakim–Adams triad: gait disturbance, dementia, urinary incontinence** (“wobbly, wacky, wet”).
- **Gait comes first and responds best.**
- Gait is **magnetic, broad-based, with start hesitation and en bloc turning, but preserved arm swing**.
- Cognition is **subcortical/frontal** — slowing, inattention, executive dysfunction, apathy.
- **Evans index >0.3**; **callosal angle <90°**; **DESH pattern** supports the diagnosis.
- **Small-vessel vascular dementia produces the triad more often than NPH does.**
- Tap test: **good positive, poor negative predictive value**; **extended lumbar drainage is the best predictor**.
- Predictive power rises: **clinical/imaging → tap test → infusion test → external lumbar drainage**.
- Treatment = **VP shunt with a programmable valve**; complications are **infection, obstruction and overdrainage with subdural collection**.
- **PENS trial 2025**: gait velocity gain **0.21 m/s**, **80% vs 24%** meaningful improvement; **no significant cognitive or continence benefit**.

CONSOLIDATED RAPID-REVISION GRID

The Ten Answers on One Page

#	Topic	The single fact most likely to earn the mark
1	Dementia and management	ChEI for mild–moderate AD, memantine for moderate–severe; **DOMINO** supports continuing donepezil; **Lancet Commission 2024: 14 risk factors, 45% preventable**
2	Dementia (repeat)	India — **LASI-DAD: 7.4%, 8.8 million, 16.9 million by 2036**
3	MCI	Four core criteria; **15–20% convert per year**; **anti-amyloid antibodies now licensed at the MCI stage**
4	Reversible dementias	**<4% truly reversible**; drugs are the commonest contributor; **treatment does not guarantee reversal**
5	Behavioural symptoms	**90–98%** develop at least one NPS; **DICE**; caregiver interventions outperform antipsychotics
6	Four common dementias	**AD = hippocampus/temporoparietal; VaD = vessel-size dependent; DLB = cortical Lewy bodies with occipital hypometabolism; FTD = frontal/anterior temporal**
7	Tau / progranulin / TDP-43	**>95% of FTLD = TDP-43 (50–60%) or tau (35–45%)**; MAPT → tau, GRN and VCP → TDP-43
8	Pick's disease	**Pick bodies = 3R tau-positive argyrophilic inclusions**, densest in the dentate gyrus
9	Lewy body dementia	Four core features; **probable = 2 core, or 1 core + 1 indicative biomarker**; **ChEIs effective, avoid conventional neuroleptics**
10	Normal pressure hydrocephalus	**Hakim–Adams triad**; **Evans index >0.3**; **PENS 2025 — gait improves, cognition and continence do not**

Cross-Cutting Indian Policy Points to Weave Into Any Dementia Answer

- **LASI-DAD**: dementia prevalence **7.4%** in those 60+, **8.8 million cases**, projected **16.9 million by 2036**; higher in women and rural populations
- **India Ageing Report 2023 (UNFPA)**: 60+ population to reach **20.8% by 2050**
- **NPHCE** — Regional Geriatric Centres, district geriatric units, geriatric clinics at CHC/PHC level, the delivery platform for cognitive screening
- **National Mental Health Programme / District Mental Health Programme** — the practical route for BPSD and caregiver support
- **AB-PMJAY and the Ayushman Vay Vandana Card** — universal ₹5 lakh cover for all citizens aged 70 and above regardless of income
- **Maintenance and Welfare of Parents and Senior Citizens Act, 2007** — legal remedy for neglect and abandonment, highly relevant to dementia caregiving breakdown
- **Rights of Persons with Disabilities Act, 2016** — includes chronic neurological conditions and mental illness, enabling disability certification in young-onset dementia
- **Rashtriya Vayoshri Yojana** and **AVYAY** — assistive devices and senior citizen welfare schemes
- **Elder Line 14567** — national toll-free helpline for older adults
- **ARDSI (Alzheimer's and Related Disorders Society of India)** — Dementia India reports, caregiver training, day care and support groups

- **WHO ICOPE (2nd edition)** — cognitive decline is one of the six domains of intrinsic capacity screened in primary care

---

End of compilation — Questions 1 to 10, Dementia and Cognitive Disorders.

Parkinson's Disease & Movement Disorders

MD Geriatric Medicine — High-Yield 15-Mark Answers

Primary sources: Pathy's Principles and Practice of Geriatric Medicine (6th ed., Ch. 53 & 54) and Essentials of Clinical Geriatrics (7th ed.)
Supplemented with peer-reviewed evidence and guidelines to August 2026 | Indian context integrated throughout

Contents

1. Parkinson's disease — pathology, pathogenesis and treatment (drug choice, side effects, newer modalities, non-motor symptoms)
2. Parkinson-plus syndromes in older adults — etiopathogenesis, clinical presentation and management
3. Drug treatment for Parkinson's disease
4. Levodopa
5. Parkinsonism
6. Motor fluctuations in Parkinson's disease
7. Non-motor symptoms of Parkinson's disease
8. Progressive supranuclear palsy — clinical features, management and prognosis RECURS
9. Benign / essential tremor — presentation, differential diagnosis and treatment

How to use this compilation

- Each answer has four parts: **High-Yield Points** (textbook), **Latest Updates (2023–present)**, **Current Practice Update** table, and an **Examiner's Tip** skeleton.
- Textbook content and recent evidence are kept strictly separate, as instructed.
- Topics flagged RECURS carry an additional green **Extra Points to Remember** box.
- Questions 1, 3, 4, 6 and 7 overlap heavily — a single core of levodopa pharmacology answers all five. Read Q1 first; the rest are re-framings for different mark weights.

Q1. Parkinson's disease — pathology, pathogenesis and treatment, with emphasis on drug choice, side effects, newer modalities and treatment of non-motor symptoms

High-Yield Points (Textbook)

Source: Pathy's 6th ed., Chapter 53 (Oquendo & Belmin); Essentials of Clinical Geriatrics 7th ed., Chapter 10.

Definition and epidemiology

1. Parkinson's disease (PD) is the **second most common neurodegenerative disease after Alzheimer's disease**. Despite progress in understanding the neurochemistry, the **exact cause remains undetermined**.
2. Prevalence is **about 1% in people over 65 years**. Median age at diagnosis is 60. Late-onset PD means first symptoms after age 60; **very late-onset PD (after 75) is uncommon**.
3. Incidence rises with age up to 80 years and then appears to **fall in the ninth decade**. Mean duration from diagnosis to death is **about 15 years**.
4. Because of this, most PD patients seen in geriatric practice have **longstanding disease with lost independence and complications**, not new diagnoses.
5. Risk factors: family history in **6–10%**; weak associations with head injury, rural living, obesity and herbicide/pesticide exposure. Male sex is debated. **Cigarette smoking and coffee appear protective.**

Pathology and pathogenesis

6. The main pathological finding is **loss of neurons in the substantia nigra** with **profound dopamine depletion** in the projection pathways from substantia nigra to the striatum.
7. The second hallmark is **α-synuclein bound to ubiquitin** accumulating in damaged neurons to form **Lewy bodies**, found in substantia nigra cells and other brain areas.
8. About **10 gene mutations** are identified in familial PD, but these operate mainly in **early-onset** disease and are rarely involved in late-onset PD.
9. Once dopamine loss crosses a threshold, the classical triad appears: **bradykinesia/akinesia, resting tremor and rigidity** (Essentials).

Clinical features

10. Onset is **insidious with slow progression**. Cardinal signs do not appear together — tremor may precede bradykinesia and rigidity by months to years. Signs are **asymmetric or unilateral at onset** and become bilateral later.
11. Other motor features: flexed posture of limbs and trunk on standing, **reduced step length and height, reduced arm swing**, falls, and writing difficulty.
12. Non-motor features: **bradyphrenia** (slowness of mental function), depression, anxiety, memory problems and dementia, **constipation**, bladder dysfunction and autonomic dysfunction (gastrointestinal, sexual, cardiovascular).
13. **Advanced PD (after 10–15 years)**: severe akinesia with less prominent tremor and rigidity, expressionless face, freezing, major gait and postural abnormality, repeated falls and fractures, wheelchair dependence, dysphagia, dysarthria, drooling, insomnia, pain, orthostatic hypotension, hallucinations and dementia. **Fluctuations and dyskinesia are very frequent and remain the main therapeutic challenge.**

Diagnosis

14. Diagnosis is clinical, using the **Queen Square Brain Bank criteria**: **Step 1** — bradykinesia plus at least one of muscular rigidity, 4–6 Hz rest tremor, or postural instability not due to visual, vestibular, cerebellar or proprioceptive causes; **Step 2** — exclusion criteria; **Step 3** — three or more supportive features.
15. **Ioflupane SPECT (DaTSCAN)** is normal in essential tremor, drug-induced parkinsonism and Alzheimer's disease, and abnormal in PD and other degenerative parkinsonian syndromes.
16. **Levodopa challenge** (150–250 mg with a decarboxylase inhibitor in a fasting patient; assess tremor, rigidity and akinesia every 30 minutes for 2–3 hours) indicates **dopaminergic responsiveness — it is not a formal diagnostic test**.

Treatment — drug choice and side effects in the elderly

17. **Levodopa is the main and often the only treatment for PD in the elderly.** It crosses the blood–brain barrier and is given with a peripheral decarboxylase inhibitor — **Madopar** (levodopa + benserazide) or **Sinemet** (carbidopa + levodopa). It is effective for **tremor, bradykinesia and rigidity**. Start low, increase very gradually; usual early control on **300–600 mg/day** in 3–4 doses, **30 minutes before meals**. Dispersible Madopar peaks faster and is used for **early-morning akinesia**.
18. **Side effects of levodopa**: nausea and vomiting, hypotension, sedation, nightmares and vivid dreams, hallucinations, delusions and delirium; after several years, **motor fluctuations** (delayed effect, wearing-off, on–off) and **involuntary movements** (chorea, athetosis, dystonia).
19. **Drugs Pathy's says are NOT used in older people with PD:** direct dopamine agonists (apomorphine, piribedil, ropinirole, lisuride, pramipexole, pergolide) — useful in the young to delay levodopa but adverse effects are numerous and can be severe; and **anticholinergics (trihexyphenidyl, tropatepine), which are never used in the elderly** because of delirium and other adverse effects.
20. **Adjuncts: COMT inhibitors** (entacapone, tolcapone) prolong levodopa effect and are useful in older patients with motor fluctuations and/or dyskinesia; **Stalevo** is the fixed combination of levodopa/carbidopa (50/12.5, 100/25, 150/37.5 mg) with entacapone 200 mg for unstabilised end-of-dose fluctuation. **Amantadine 200–400 mg/day** lessens peak-dose dyskinesia — reduce dose in renal impairment, delirium is frequent in the old. **Selegiline and rasagiline** (MAO-B inhibitors) have been proposed for neuroprotection in early PD.

Treatment of non-motor symptoms (Pathy's Table 53.4 — each considered only after adjusting levodopa)

Problem	Best-evidence treatment	Other options
Nausea, vomiting	Domperidone (ECG monitoring for QT prolongation)	Metoclopramide is **contraindicated**
Depression	Venlafaxine	Other SSRIs, pramipexole (delirium risk — not first choice in elderly), CBT, rTMS
Generalised anxiety disorder	None established	SSRIs, cognitive-behavioural therapy
Hallucinations, delusions	Clozapine; pimavanserin (5-HT2A inverse agonist)	Clozapine 12.5 mg start, 5-day increments to 25–50 mg; FBC weekly × 2 months then twice monthly; **stop permanently if neutrophils <1500/mm^3**
Dementia	Rivastigmine	Donepezil
Apathy	Rivastigmine	—
Mild cognitive impairment	None established	—
Orthostatic hypotension	Droxidopa	Midodrine, fludrocortisone, elastic compression
Insomnia	Sleep hygiene	Melatonin
Constipation	Hydration, probiotics and prebiotic fibre, macrogol	Lubiprostone if resistant; psyllium; entacapone's diarrhoea can be exploited
Drooling	Botulinum toxin into salivary glands if severe	—
Urinary urgency	None established	Solifenacin (low brain penetration)
Erectile dysfunction	Sildenafil	—

Newer modalities and non-pharmacological treatment

21. **Bilateral deep brain stimulation of the subthalamic nucleus or globus pallidus interna** produces sustained motor improvement, especially for levodopa-responsive symptoms, and reduces drug-induced involuntary movements. It is offered to **cognitively intact** patients with severe disability from refractory motor complications; severe comorbidity is a limitation. Pathy's states DBS is **not currently used in elderly people with PD**, but notes observational studies showing **similar efficacy in patients over 70** with no age-related increase in adverse effects. Concerns remain about depression and suicide, and disability still progresses because neurodegeneration continues.
22. Other surgical options (Essentials): **thalamotomy, pallidotomy**, and rarely intraputaminal fetal mesencephalic allografts.
23. **Rehabilitation should begin early** to preserve motor function, with sessions timed to the **peak effect of levodopa**. It targets joint flexibility, walking, lifting, posture, balance and turns, and counters the flexed attitude. **Speech therapy** helps dysarthria, drooling and swallowing later. Combined aerobic, strengthening, balance and stretching programmes improve balance in PD.
24. **Loss of independence** requires organised help at home — professional carers, caregiver involvement, meals-on-wheels. PD is not a frequent cause of institutional admission except when severe cognitive and behavioural complications occur. **Psychological support and patient/caregiver education** improve acceptance and optimise use of health resources.

Latest Updates (2023–Present)

- **Shift to a biological definition.** Two research frameworks were published together in *Lancet Neurology* 2024 — the **Neuronal α-Synuclein Disease Integrated Staging System (NSD-ISS)** (Simuni et al.) and **SynNeurGe** (Höglinger et al.). Both anchor diagnosis in **CSF α-synuclein seed amplification assay (SAA)** plus dopaminergic neurodegeneration on DAT imaging; SynNeurGe adds a genetic component (S–N–G). Both encompass PD and dementia with Lewy bodies. These are explicitly **research constructs, not yet for routine clinical diagnosis** (JAMA Neurology viewpoint 2024; Cell Death & Disease review 2025).
- **New levodopa formulations. Crexont (IPX203)**, extended-release carbidopa/levodopa capsules, FDA-approved August 2024 for motor fluctuations. **Vyalev (foslevodopa/foscarbidopa)**, the first **24-hour subcutaneous continuous infusion**, approved October 2024 — delivers levodopa prodrugs by pump and bypasses the gut, an alternative to intestinal gel. **Onapgo (subcutaneous apomorphine infusion)** approved February 2025 for "off" episodes. **ND0612** (subcutaneous levodopa/carbidopa) under FDA review.
- **New oral agent in the pipeline. Tavapadon**, a selective **D1/D5 partial agonist** — a different mechanism from D2/D3 agonists — met endpoints in the Phase 3 **TEMPO-1, -2 and -3** trials; TEMPO-3 gave an extra **1.1 hours of daily "on" time without troublesome dyskinesia**. NDA submitted 26 September 2025; FDA decision expected 2026 (AbbVie; MDS Congress 2025).
- **Safety alert.** In **March 2026 the FDA required a new label warning for all carbidopa/levodopa products** (Sinemet, Rytary, Stalevo, Crexont, Vyalev) regarding **vitamin B6 deficiency and seizures** — directly relevant to long-treated geriatric patients.
- **Adaptive (closed-loop) DBS.** FDA approved Medtronic **BrainSense Adaptive DBS** and the **BrainSense Electrode Identifier** in **February 2025**, based on the **ADAPT-PD** randomised crossover trial — the world's first commercially available aDBS. Stimulation is adjusted in real time to sensed beta-band local field potentials; the Electrode Identifier cuts initial programming time by about 85%.
- **Disease modification remains unproven. Exenatide-PD3** (*Lancet* 2025; 194 patients, 96 weeks) found **no benefit of weekly exenatide over placebo** on motor progression, despite positive Phase 2 exenatide and lixisenatide (*NEJM* 2024) data. *The Lancet* issued an **Expression of Concern on this trial in June 2026** — quote it cautiously. Pipeline (2026): prasinezumab (anti-α-synuclein antibody, Phase 3), bemdaneprocel (stem-cell, Phase 3), AB-1005 GDNF gene therapy (Phase 2, RMAT designation).
- **Indian data.** A **pan-Indian multicentre study of PD** (Kishore et al., *Movement Disorders* 2025) found clinical features, exposures and comorbidities broadly similar to European populations, but an **earlier age at onset in Indian patients, suggesting greater genetic contribution**. Community prevalence in India varies widely: crude rates **7–60 per 100,000**, age-adjusted **53–192 per 100,000**; Kolkata age-adjusted 52.85/100,000 with rates rising

to **621/100,000 above 80 years** (Das et al., *Neurology* 2010; Khurana & Gourie-Devi, *Cureus* 2025). The Parsi community of Mumbai has an exceptionally high crude rate of **328/100,000** and cannot be generalised. A Bangalore old-age-home study found parkinsonism in **17.8%** of residents with very poor awareness.

Current Practice Update

Aspect	Textbook position (Pathy's 6th ed.)	Current evidence-based practice
Basis of diagnosis	Clinical — Queen Square Brain Bank criteria	Clinical criteria remain the practice standard, but biological research definitions (NSD-ISS, SynNeurGe) using CSF α-synuclein SAA now exist and dominate trial recruitment
Dopamine agonists in the elderly	"Not used in older people with PD"	Still avoided as first line, but the position is nuanced — tavapadon (D1/D5 partial agonist) is designed for a better tolerability profile; low-dose rotigotine patch is used where oral routes fail
Deep brain stimulation	"Not currently used in elderly people with PD"	**Age alone is no longer a bar**; outcomes in patients >70 match younger patients. **Adaptive closed-loop DBS approved February 2025**
Continuous dopaminergic delivery	Duodenal levodopa infusion mentioned; subcutaneous apomorphine "limited data"	Three device-aided options now licensed: **Vyalev** (SC levodopa prodrug, 24 h), **Onapgo** (SC apomorphine), levodopa–carbidopa intestinal gel
Long-term levodopa safety	Nausea, hypotension, neuropsychiatric effects, fluctuations, dyskinesia	Add **vitamin B6 deficiency and seizure risk** (FDA labelling change, March 2026) and peripheral neuropathy with high-dose/continuous delivery
Disease modification	Selegiline/rasagiline "proposed for neuroprotective effects"	No agent has proven disease modification. **Exenatide-PD3 was negative** (Lancet 2025); MAO-B neuroprotection is no longer claimed

Examiner's Tip — lines that earn marks

- "The two pathological hallmarks are **nigral neuronal loss with striatal dopamine depletion** and **ubiquitinated α-synuclein forming Lewy bodies**."
- "In the geriatric population **levodopa is the main and often the only drug**; dopamine agonists and anticholinergics are avoided."
- "**Levodopa does not improve non-motor symptoms, posture or gait** — every non-motor treatment is considered only after levodopa has been optimised."
- Name the drug–symptom pairs verbatim: **venlafaxine** for depression, **clozapine or pimavanserin** for psychosis, **rivastigmine** for dementia and apathy, **droxidopa** for orthostatic hypotension, **domperidone** for nausea.
- Close with one 2025 fact: "**Adaptive DBS was FDA-approved in February 2025 on the ADAPT-PD trial**, and age over 70 is no longer a contraindication to DBS."

Q2. Etiopathogenesis, clinical presentation and management of the Parkinson-plus syndromes in older adults

High-Yield Points (Textbook)

Source: Pathy's 6th ed., Chapter 53 (Tables 53.2, Box 53.1) and Chapter 46; Essentials of Clinical Geriatrics 7th ed.

Definition and importance

1. **Parkinson-plus (atypical parkinsonism)** means an extrapyramidal syndrome accompanied by additional neurological features not explained by idiopathic PD, with a **poor response to levodopa**.
2. These are disproportionately important in geriatrics: in **very late-onset parkinsonism, non-PD causes account for 20–40% of all new parkinsonism** in the elderly, a much higher proportion than in early-onset disease.
3. **Poor levodopa response is the single most useful clinical discriminator**, assessed from history or by formal levodopa challenge (Box 53.1).
4. Etiopathogenesis divides into **synucleinopathies** (multiple system atrophy, dementia with Lewy bodies) and **tauopathies** (progressive supranuclear palsy, corticobasal degeneration), alongside non-degenerative causes (drug-induced, vascular, hydrocephalic).

Clinical presentation of the individual syndromes (Pathy's Table 53.2)

Condition	Characteristic features
Frequently observed as very late-onset parkinsonism	
Drug-induced parkinsonism	Exposure to neuroleptics or other drugs before onset. **Dyskinesia frequent, no tremor**. Ioflupane SPECT normal.
Lewy body dementia	Dementia within a few months before or after onset of parkinsonism; **hallucinations**; **severe adverse reactions to neuroleptics**; fluctuating course; falls.
Alzheimer's disease	Parkinsonism infrequent (about 15%), only at moderate/severe stages and usually mild. Hippocampal atrophy on MRI; SPECT normal.
Progressive supranuclear palsy	Early postural instability and repeated falls; **axial rigidity**; limitation of vertical gaze; neck dystonia; impaired saccades on ocular electrophysiology. **No tremor.** MRI: **midbrain atrophy**.
Cerebrovascular disease	Parkinsonism with several infarcts on MRI, worsening after infarction; multilacunar state, pseudobulbar syndrome, emotional lability, pyramidal signs, vascular dementia.
Normal pressure hydrocephalus	Gait disturbance, urinary incontinence, cognitive slowness. Marked ventricular enlargement not explained by atrophy.
Very rarely observed as very late-onset parkinsonism	
Multiple system atrophy	**Severe dysautonomia early** in the course — orthostatic hypotension, urinary disorder, sexual dysfunction, gastrointestinal symptoms — with pyramidal or cerebellar syndrome.
Corticobasal degeneration	Dystonia, myoclonus, sensory disorders, **alien ("foreign") hand syndrome**.
Huntington's disease	Chorea; familial cases.
Wilson's disease	Liver disease, complex neurological signs including parkinsonism, psychiatric symptoms, familial cases, **Kayser–Fleischer ring**.
Miscellaneous	Toxic; post-encephalitic; repeated skull trauma.

5. **Dementia with Lewy bodies** is strongly related to PD, sharing **Lewy body and α-synuclein pathology**. Diagnosis needs (i) cognitive dysfunction fulfilling dementia criteria with insidious onset and progression, plus (ii) one or more of hallucinations, extrapyramidal syndrome, fluctuating course, falls and poor tolerance of neuroleptics. Extrapyramidal signs may precede cognitive decline by months.
6. **Key timing rule:** dementia within a few months of parkinsonism = **DLB**; dementia after several years of established PD = **PD-related dementia**.
7. In Alzheimer's disease, extrapyramidal signs may reflect current or past neuroleptic exposure — this must be sought by interviewing patients, proxies, physicians and pharmacists. Their presence **at diagnosis predicts more rapid decline**.
8. PSP is **frequently misdiagnosed as PD**. Cerebral MRI shows a **convex midbrain outline indicating atrophy**, where the normal midbrain aspect is concave.
9. Vascular parkinsonism: **onset or worsening of extrapyramidal signs within one month after a stroke strongly supports the diagnosis**. Swallowing problems and gait/posture abnormality are frequent; tremor infrequent; levodopa response poor. MRI shows multiple lacunae and/or severe leukoaraiosis.
10. NPH gait can mimic PD (short steps, shuffling, slowness, stooped trunk, flexed lower limbs) but there is **no tremor and no rigidity**, and the gait **does not respond to external cues or to levodopa**. Imaging shows quadriventricular dilatation with small sulci and periventricular T2 high signal.

Diagnostic approach

11. In most cases **medical history and physical examination alone allow identification**; the drug history must be exhaustive.
12. **Ioflupane (DaTSCAN) SPECT** separates degenerative parkinsonism (abnormal) from essential tremor, drug-induced parkinsonism and Alzheimer's disease (normal). MRI identifies midbrain atrophy, vascular lesions and hydrocephalus. Ocular electrophysiology supports PSP.
13. Where doubt persists, **follow-up usually clarifies the diagnosis** as new signs and symptoms emerge.

Management

14. **Give an adequate trial of levodopa in every case** — a clear negative response is itself diagnostically useful, and some patients obtain partial benefit.
15. **Drug-induced parkinsonism:** stopping the offending drug is the best option. Recovery is **very slow, over weeks to months**, and in about **20% some extrapyramidal signs persist**, suggesting underlying PD or another neurodegenerative parkinsonism unmasked by the drug. **Tardive dyskinesia is often permanent** even after withdrawal. There is **no satisfactory drug treatment**.
16. In elderly patients this withdrawal is usually possible, because neuroleptics are mainly used for behavioural complications of dementia — **a condition for which they are ineffective**. If severe psychosis demands treatment, switch from a classical neuroleptic to a **low-dose atypical antipsychotic**.
17. **Anticholinergics must be avoided** in the elderly — they induce delirium and other adverse effects.
18. **Prevention of drug-induced parkinsonism is a crucial issue in geriatric care**, achieved by avoiding neuroleptic prescription in older people, especially those with dementia.
19. Supportive and symptomatic care dominates management: falls prevention and physiotherapy, speech and swallow therapy, treatment of orthostatic hypotension (droxidopa, midodrine, fludrocortisone, elastic compression), botulinum toxin for drooling, carer education and advance care planning. **DBS is not indicated in Parkinson-plus syndromes.**
20. **Prognosis:** atypical syndromes progress faster than PD, with earlier falls, dysphagia, dependency and death, usually from aspiration pneumonia.

Latest Updates (2023–Present)

- **MSA — new criteria and validation.** The **MDS criteria for MSA (Wenning et al., *Mov Disord* 2022)** replaced the 2008 consensus and define four certainty levels: **neuropathologically established, clinically established, clinically probable, and possible prodromal MSA**. Brain-bank validation confirmed improved early sensitivity (Sekiya et al., *Neurology* 2023); adding MRI markers further improves performance (*Mov Disord* 2024).
- **α-Synuclein seeding biomarkers.** *Lancet Neurology* 2024 (MSA review) highlights **disease-specific α-synuclein strains** distinguishable by seed amplification assay. **Skin biopsy for phosphorylated α-synuclein** is emerging — specificity for PD up to 100%, sensitivity up to 81%, and **fibrillar phospho-α-synuclein in Schwann cells appears to be an MSA-specific hallmark** (Donadio et al.; Gibbons et al., *Neurology* 2023). Skin SAA is being evaluated to separate MSA from PD (2025–26 data). These remain **promising but incompletely validated**, and are not in routine Indian practice.
- **PSP — expanded phenotype.** The **MDS-PSP criteria** define **eight clinical variants** across four functional domains and include a "**suggestive of PSP**" category for early disease; this category was **prospectively validated** (Quattrone et al., *Mov Disord* 2025). **Second-generation tau-PET (18F-PI-2620)** detects 4-repeat tau in the basal ganglia and predicts increasing diagnostic certainty; plasma/CSF **NfL and GFAP** on SIMOA track progression (*Brain Communications* 2025).
- **Global and Indian perspective on PSP.** A 2026 global review in *Movement Disorders Clinical Practice* (Kukkle et al.) reports prevalence **5–6.4 per 100,000**, with regional variation in the **MAPT H1 haplotype**, dietary neurotoxins and industrial pollutants, and marked differences in access to specialist care.
- **DLB within the biological framework.** Both the NSD-ISS and SynNeurGe systems (*Lancet Neurology* 2024) explicitly cover **PD and DLB as one α-synuclein-driven biological continuum**, formally dissolving the arbitrary "one-year rule" for research purposes — though the clinical rule is still examined.
- **Therapeutics.** There is still **no disease-modifying therapy for any Parkinson-plus syndrome**. Tau-directed agents (antibodies, antisense oligonucleotides, sodium selenate) are in trial for PSP; TEV-56286 (OGA inhibitor) is in Phase 2 for MSA.

Current Practice Update

Aspect	Textbook position	Current evidence-based practice
MSA diagnosis	Clinical — early severe dysautonomia with pyramidal or cerebellar signs	**MDS 2022 criteria** with four certainty levels including **possible prodromal MSA**; MRI markers formally incorporated
PSP diagnosis	Single phenotype: gaze palsy, early falls, axial rigidity, midbrain atrophy	**MDS-PSP criteria — eight variants** (PSP-RS, PSP-P, PSP-PGF, PSP-CBS and others) with an early "suggestive of PSP" category
Confirmatory testing	Ioflupane SPECT, MRI, ocular electrophysiology	Add **α-synuclein SAA (CSF and skin)** and **tau-PET (18F-PI-2620)** — research/tertiary use; MRI plus autonomic testing remain the routine tools
DLB vs PD dementia	Separated by the one-year timing rule	Clinically retained; biologically both are classed as **neuronal α-synuclein disease** in the 2024 research frameworks
Treatment	Symptomatic; levodopa trial; avoid neuroleptics	Unchanged — **no disease-modifying therapy exists**; tau- and synuclein-directed trials ongoing

Examiner's Tip — lines that earn marks

- Open with the geriatric statistic: "**In very late-onset parkinsonism, 20–40% of new cases are not idiopathic PD.**"
- State the discriminator: "**Parkinsonian syndromes other than PD respond poorly to levodopa.**"
- Give the one-line signature of each: **MSA** = early severe dysautonomia; **PSP** = early falls + vertical gaze palsy + axial rigidity + midbrain atrophy; **CBD** = alien hand, myoclonus, dystonia; **DLB** = dementia within months + hallucinations + neuroleptic sensitivity; **vascular** = worsening within one month of stroke; **NPH** = gait + incontinence + cognitive slowing with ventriculomegaly.
- Score the geriatric point: "**Prevention of drug-induced parkinsonism by avoiding neuroleptics in dementia is a crucial issue in geriatric care** — 20% have persistent signs even after withdrawal."

Q3. Drug treatment for Parkinson's disease

High-Yield Points (Textbook)

Source: Pathy's 6th ed., Chapter 53; Essentials of Clinical Geriatrics 7th ed., Table 10-6.

Principle

1. Drug treatment aims to **increase the ratio of dopamine to acetylcholine** in the central nervous system, specifically in the **nigrostriatal system**.
2. Six drug groups are used: **levodopa combinations, dopamine agonists, MAO-B inhibitors, COMT inhibitors, amantadine and anticholinergics**.
3. **Clinical response may take several weeks**; side effects are common and often limit treatment; response varies widely between patients.

Drugs, doses, mechanism and side effects (Essentials Table 10-6)

Drug	Usual dose	Mechanism	Main side effects
Carbidopa–levodopa (Sinemet)	40/400 to 200/2000 mg/day in divided doses	Increases dopamine availability; decreases peripheral dopamine metabolism	Nausea, vomiting, anorexia; **dyskinesias**; orthostatic hypotension; behavioural disturbance; vivid dreams and hallucinations
Amantadine (Symmetrel)	100–300 mg/day (200–400 mg/day for dyskinesia)	Increases dopamine release	**Delirium and hallucinations**. Renally eliminated — **reduce dose in renal impairment**
Bromocriptine (Parlodel)	1–1.5 mg tid/qid, up to 30–40 mg/day	Directly activates dopaminergic receptors	Behavioural change, hypotension, nausea
Pramipexole (Mirapex)	0.5–1.5 mg tid	Dopamine agonist	Hallucinations, nausea, somnolence
Ropinirole (Requip)	3–8 mg tid	Dopamine agonist	Orthostatic hypotension, syncope, nausea, somnolence
Trihexyphenidyl (Artane)	2–20 mg/day divided	Reduces acetylcholine effect; restores cholinergic–dopaminergic balance	Dry mouth, constipation, urinary retention, blurred vision, glaucoma exacerbation, tachycardia, **confusion**, behavioural change
Benztropine (Cogentin)	0.5–8.0 mg/day divided	As above	As above
Selegiline (Eldepryl)	10 mg/day single dose	MAO-B inhibitor	Nausea, confusion, agitation, insomnia, involuntary movements
Rasagiline (Azilect)	1 mg/day single dose	Selective, irreversible MAO-B inhibitor	Balance difficulty, anorexia, vomiting, weight loss, depressive symptoms; **no dietary restriction**
Entacapone (Comtan)	200 mg with each levodopa dose	Inhibits catechol-O-methyltransferase	Nausea, dyskinesias, orthostatic hypotension, **diarrhoea**
Tolcapone (Tasmar)	100–200 mg tid	COMT inhibitor	**Liver dysfunction**

Prescribing in the older patient

4. **Levodopa is the main and often the only treatment for PD in the elderly.** Given with a peripheral dopa-decarboxylase inhibitor as **Madopar** (levodopa + benserazide) or **Sinemet** (carbidopa + levodopa); effective for **tremor, bradykinesia and rigidity**.
5. **Start low and increase very gradually.** Most patients are controlled in the first years on **300–600 mg/day**, given 3–4 times daily, preferably **30 minutes before meals**. **Dispersible Madopar** peaks faster and is used for early-morning akinesia.
6. **Direct dopamine agonists are not used in older people with PD.** They act on D2 receptors and can delay levodopa introduction in younger patients, but **adverse effects are numerous and could be severe** in the elderly.
7. **Anticholinergic drugs (trihexyphenidyl, tropatepine) are never used in older people with PD**, although they improve tremor, because they induce delirium and other adverse effects.
8. **MAO-B inhibitors** (selegiline, rasagiline) have been proposed for neuroprotective effects in early PD.
9. **COMT inhibitors** decrease dopamine degradation and prolong the levodopa effect — **useful in older patients with motor fluctuations and/or levodopa-induced dyskinesia**. **Stalevo** combines levodopa/carbidopa (50/12.5, 100/25, 150/37.5 mg) with fixed-dose entacapone 200 mg, indicated for **end-of-dose fluctuation not stabilised by levodopa–decarboxylase inhibitor**.
10. **Amantadine 200–400 mg/day** is effective in lessening **peak-dose dyskinesia**; adjust for renal impairment; delirium is frequent in old patients.
11. **Rotigotine transdermal patch** (non-ergoline agonist) is used in Europe with levodopa — ease of administration and stable blood levels are advantages (Essentials).
12. **Metoclopramide is contraindicated** in parkinsonian patients because of its striatal dopamine receptor effects; **domperidone**, which does not cross the blood–brain barrier, is used instead.
13. Excess dopaminergic stimulation causes **sleep disturbance, delirium and psychosis**. Response variability produces morning akinesia, peak-dose dyskinesias and freezing episodes (the "on–off phenomenon").
14. **Cholinesterase inhibitors** have evidence of benefit in **PD-associated dementia**, with a positive effect on global assessment (Essentials).

15. Drugs for non-motor complications, each considered **after adjusting levodopa**: domperidone (nausea), venlafaxine (depression), clozapine or pimavanserin (hallucinations/delusions), rivastigmine (dementia and apathy), droxidopa/midodrine/fludrocortisone (orthostatic hypotension), melatonin (insomnia), macrogol (constipation), solifenacin (urgency), sildenafil (erectile dysfunction).
16. **Clozapine monitoring is examinable**: start 12.5 mg daily, five-day increments to a usual 25–50 mg; blood count weekly for two months then twice monthly; **discontinue permanently if neutrophils fall below 1500/mm^3**. Pimavanserin requires no haematological monitoring.
17. Preliminary data suggest **coenzyme Q10** may protect the nigrostriatal dopaminergic system, but further research is needed; **pharmacogenetics** is expected to allow more individualised prescribing.

Latest Updates (2023–Present)

- **Newly licensed levodopa delivery systems** (not in either textbook): **Crexont** (IPX203 extended-release carbidopa/levodopa capsules, FDA August 2024); **Vyalev** (foslevodopa/foscarbidopa, first 24-hour subcutaneous infusion, FDA October 2024); **Onapgo** (subcutaneous apomorphine infusion, FDA February 2025); **ND0612** subcutaneous levodopa/carbidopa under review.
- **Tavapadon** — a **selective D1/D5 receptor partial agonist**, mechanistically distinct from the D2/D3 agonists in the textbook table. Positive Phase 3 TEMPO-1/2/3; NDA filed September 2025, decision expected 2026. If approved it will be the **first new oral dopamine agonist for PD in over a decade**.
- **Newer adjuncts absent from the textbooks** but now standard in international practice: **opicapone** (once-daily COMT inhibitor), **safinamide** (MAO-B inhibitor with glutamate-modulating action) and **istradefylline** (adenosine A2A antagonist) for off-time reduction.
- **FDA labelling change, March 2026:** a warning regarding **vitamin B6 deficiency and seizures** now applies to **all carbidopa/levodopa products**, including Sinemet, Rytary, Stalevo, Crexont and Vyalev. Consider periodic B6 assessment in long-treated older patients.
- **Neuroprotection claims withdrawn.** No agent has demonstrated disease modification. **Exenatide-PD3** (*Lancet* 2025) was negative, despite positive Phase 2 lixisenatide data (*NEJM* 2024); the Lancet issued an Expression of Concern on the exenatide paper in June 2026. MAO-B inhibitors are now prescribed for **symptomatic benefit only**.
- **Indian practical note:** levodopa–carbidopa, levodopa–benserazide, trihexyphenidyl, amantadine, pramipexole, ropinirole, rasagiline, entacapone and the levodopa/carbidopa/entacapone combination are all widely available and inexpensive in India; device-aided therapies (intestinal gel, subcutaneous infusions) and DBS are limited to a few tertiary centres and are largely out-of-pocket.

Current Practice Update

Aspect	Textbook position	Current evidence-based practice
COMT inhibitors	Entacapone (with each dose) and tolcapone (hepatotoxic)	**Opicapone once daily** is now preferred where available — better adherence, no hepatotoxicity monitoring
MAO-B inhibitors	Selegiline and rasagiline "proposed for neuroprotective effects"	Used for **symptomatic benefit only**; **safinamide** added as an option for wearing-off
Off-time adjuncts	COMT inhibitors and amantadine	Add **istradefylline** (adenosine A2A antagonist) and the new ER/infusional levodopa formulations
Dopamine agonists	D2 agonists — avoided in the elderly	Position holds for D2/D3 agonists; **tavapadon (D1/D5 partial agonist)** may change the calculus if approved in 2026
Levodopa safety monitoring	Nausea, hypotension, neuropsychiatric effects, motor complications	Add **vitamin B6 deficiency/seizures** (FDA March 2026) and peripheral neuropathy with continuous delivery

Examiner's Tip — lines that earn marks

- Open with the principle: "**Treatment aims to restore the dopamine–acetylcholine ratio in the nigrostriatal system.**"
- Draw the Essentials table — drug, dose, mechanism, side effect — it converts directly into marks.
- State both geriatric prohibitions explicitly: "**Dopamine agonists are not used and anticholinergics are never used in older people with PD.**"
- Quote the two monitoring rules: **clozapine — stop if neutrophils <1500/mm^3**; **domperidone — ECG for QT prolongation**; and **metoclopramide is contraindicated**.

Q4. Levodopa

High-Yield Points (Textbook)

Source: Pathy's 6th ed., Chapters 53 and 30 (gastrointestinal ageing); Essentials of Clinical Geriatrics 7th ed.

1. Levodopa is the **metabolic precursor of dopamine**. Unlike dopamine itself, it **crosses the blood–brain barrier**.
2. It is combined with a **peripheral dopa-decarboxylase inhibitor** to lessen systemic effects: **Madopar** = levodopa + benserazide; **Sinemet** = carbidopa + levodopa.
3. It is the **main and often the only treatment for PD in the elderly**, and the most effective drug available.
4. It is effective for **tremor, bradykinesia and rigidity**.
5. Several formulations exist, distinguished by their **rates of absorption**. Standard levodopa is given **three or four times daily, preferably 30 minutes before meals**.
6. Most patients are controlled in the first years of PD on **300–600 mg per day**. It should be **started at low doses and increased very gradually** until the appropriate effect is obtained.
7. The **dispersible form (dispersible Madopar)** achieves peak plasma concentration faster and is used for **early-morning akinesia**, allowing the first standard dose to be taken at the same time.
8. As disease progresses the dose must be increased progressively, and **adverse effects become frequent**.
9. **Peripheral side effects:** nausea, vomiting, anorexia and hypotension.
10. **Neuropsychiatric side effects:** sedation, nightmares, vivid dreams, hallucinations, delusions and delirium — all **dose-related** and especially common in older patients.
11. **Long-term complications after several years: motor fluctuations** with the on–off phenomenon, delayed effect of levodopa and wearing-off between doses; and **involuntary movements** — chorea, athetosis and dystonia. These complications require **revision of the timing of levodopa administration**.
12. **Gastric emptying is delayed in at least 70% of PD patients**, even without levodopa, and levodopa slows it further. Levodopa **retained in the stomach is metabolised to dopamine and becomes unavailable for systemic absorption**.
13. In patients with the on–off phenomenon, gastric emptying may **normalise in the "on" phase**; conversely, variable emptying causes **erratic levodopa absorption and itself contributes to the on–off phenomenon**.
14. **Direct duodenal infusion of levodopa** has been advocated to solve this problem; increasing the **ratio of decarboxylase inhibitor to levodopa** has also been suggested to improve dopamine availability.
15. A critical examination point: **levodopa is not considered to improve non-motor symptoms or signs, or posture and gait.**
16. **Role in diagnosis:** in the Queen Square Brain Bank criteria, supportive features include **excellent (70–100%) response to levodopa**, **severe levodopa-induced chorea** and **levodopa response sustained for five years or more**; a **negative response to large doses of levodopa** (with malabsorption excluded) is an **exclusion criterion**.
17. **Levodopa challenge test** (Box 53.1): give a fasting patient **150–250 mg levodopa with a decarboxylase inhibitor** and assess tremor, rigidity and akinesia of the limbs **every 30 minutes for two to three hours**. This is an **indicator of dopaminergic response, not a formal diagnostic test** for PD.
18. Poor levodopa response points to a **Parkinson-plus syndrome, vascular parkinsonism or normal pressure hydrocephalus**.
19. **Adjuncts that extend or modify levodopa action:** COMT inhibitors (entacapone, tolcapone) prolong its effect; Stalevo is the fixed triple combination; amantadine 200–400 mg/day reduces peak-dose dyskinesia.
20. **Drug interactions/cautions:** metoclopramide is contraindicated; domperidone is used for levodopa-induced nausea; dietary protein competes with levodopa for absorption, hence pre-meal dosing.

Latest Updates (2023–Present)

- **Crexont (IPX203)** — extended-release carbidopa/levodopa capsules combining immediate-release granules with ER beads, FDA-approved **August 2024**; designed to smooth the peaks and troughs of immediate-release levodopa and increase "good on" time in patients with wearing-off.
- **Vyalev (foslevodopa/foscarbidopa)** — FDA-approved **October 2024**, the **first 24-hour continuous subcutaneous infusion** of levodopa/carbidopa prodrugs, delivered by a small worn pump. It provides continuous dopaminergic delivery **without a surgical tube**, bypassing the erratic gastric absorption described in the textbook.
- **ND0612** — a further subcutaneous levodopa/carbidopa system; FDA resubmission accepted, decision pending. If approved it joins Vyalev and Onapgo as a third subcutaneous option.
- **FDA safety warning, March 2026:** all carbidopa/levodopa products now carry a label warning for **vitamin B6 deficiency and seizures** — a new consideration in patients on long-term or high-dose continuous levodopa. Peripheral neuropathy from B-vitamin depletion is a related recognised complication of high levodopa exposure.
- **Levodopa is not neurotoxic and should not be withheld.** Contemporary reviews and the earlier LEAP trial confirm that delaying levodopa does not alter disease course; in older patients the **modern approach is to start levodopa early at the lowest effective dose** rather than "saving" it — consistent with, and reinforcing, Pathy's geriatric position.
- **Indian context:** levodopa–carbidopa (110 mg, 275 mg tablets) and levodopa–benserazide remain among the cheapest and most widely available neurological drugs in India, and are on state essential medicine lists. Dietary counselling matters in Indian practice because a high-protein evening meal (dal, paneer, milk) can blunt the response to the last dose of the day.

Current Practice Update

Aspect	Textbook position	Current evidence-based practice
Formulations	Standard and dispersible levodopa; duodenal infusion "advocated"	Add **Crexont** (ER capsules), **Vyalev** (24-h SC infusion) and **ND0612**; intestinal gel is established
Overcoming erratic gastric absorption	Duodenal infusion; raise the decarboxylase-inhibitor ratio	**Subcutaneous prodrug infusion (Vyalev)** now achieves the same aim non-surgically
Long-term adverse effects	Fluctuations, dyskinesia, neuropsychiatric effects, hypotension	Add **vitamin B6 deficiency, seizures and peripheral neuropathy** (FDA March 2026)
When to start	Implicit — levodopa is first-line in the elderly	Explicitly supported: **early initiation at lowest effective dose**; no evidence that delaying levodopa preserves neurons

Examiner's Tip — lines that earn marks

- Definition line: "**Levodopa is the precursor of dopamine which crosses the blood–brain barrier, given with a peripheral decarboxylase inhibitor as Madopar or Sinemet.**"
- Dose line: "**300–600 mg/day in the first years, 3–4 divided doses, 30 minutes before meals.**"
- The single most quotable sentence: "**Levodopa does not improve non-motor symptoms, posture or gait.**"
- Mention the **70% delayed gastric emptying** figure and its role in the on–off phenomenon — examiners reward the pharmacokinetic explanation.
- End with the challenge test protocol: **150–250 mg fasting, assess every 30 minutes for 2–3 hours**.

Q5. Parkinsonism

High-Yield Points (Textbook)

Source: Pathy's 6th ed., Chapters 44 (age-related neurological signs) and 53.

Definition

1. **Parkinsonism (extrapyramidal syndrome)** is a clinical syndrome, not a disease. **Akinesia is the defining and obligatory feature**, comprising **bradykinesia** (slowness in initiation and performance of movement) and **hypokinesia** (small amplitude of movement).
2. **Rigidity** is the increase in resistance to passive movement that is **constant throughout the range of joint displacement ("lead-pipe") and not related to the velocity of movement** — this distinguishes it from spasticity.
3. **Tremor and postural abnormality**, along with rigidity, may also occur as cardinal features of the syndrome.
4. Operational definition used in population studies: **two or more of bradykinesia, gait disturbance, rigidity and tremor**. In the East Boston community study of 467 people over 65, **159 had parkinsonism**, and prevalence increased with age.
5. The neuropathological correlate of these age-related findings is the **progressive spread of α-synuclein-immunopositive Lewy bodies and Lewy neurites**, reported in the brains of ageing but asymptomatic individuals.
6. Distinguish rigidity from **paratonia (gegenhalten)** — "superadded hypertonus of a quasi-volitional nature due to the patient's failure to relax". Its anatomical correlate is **bilateral frontal lobe pathology, usually diffuse small-vessel ischaemic disease**; some classify it as a release sign.
7. Upward conjugate gaze becomes restricted with age, a finding also observed in parkinsonism — beware over-interpreting it in the very old.

Causes

8. **Idiopathic Parkinson's disease** is the commonest cause, but in **very late-onset parkinsonism other causes account for 20–40% of new cases**.
9. **Frequently observed in the elderly:** drug-induced parkinsonism, Lewy body dementia, Alzheimer's disease, progressive supranuclear palsy, cerebrovascular disease, normal pressure hydrocephalus.
10. **Very rarely observed as very late-onset parkinsonism:** multiple system atrophy, corticobasal degeneration, Huntington's disease, Wilson's disease, and miscellaneous causes (toxic, post-encephalitic, repeated skull trauma).
11. **Drug-induced parkinsonism** is defined by exposure to neuroleptics or other drugs before onset; **dyskinesia is frequent, tremor is absent**, and ioflupane SPECT is normal. Many elderly patients, especially in long-term care, have **undiagnosed, treatable drug-induced parkinsonism** from the extrapyramidal effects of antipsychotics.
12. Drugs implicated in tremor and extrapyramidal effects include **neuroleptics (haloperidol, fluphenazine, olanzapine), metoclopramide, prochlorperazine, lithium, valproate, amiodarone, calcium channel blockers** and antidepressants.

Diagnostic approach

13. In most cases **medical history and physical examination allow identification** of the cause. Past or current neuroleptic exposure must be sought carefully by interviewing **patients, proxies, physicians and pharmacists**.
14. Apply the **Queen Square Brain Bank criteria**: Step 1 establishes the parkinsonian syndrome; Step 2 lists exclusions (repeated strokes with stepwise progression, repeated head injury, definite encephalitis, oculogyric crises, neuroleptic treatment at onset, more than one affected relative, sustained remission, strictly unilateral features after three years, supranuclear gaze palsy, cerebellar signs, early severe autonomic involvement, early severe dementia, Babinski signs, cerebral tumour or communicating hydrocephalus, negative response to large doses of levodopa, MPTP exposure); Step 3 requires three or more supportive features.
15. **Parkinsonian syndromes other than PD respond poorly to levodopa** — assessed from history or by levodopa challenge.
16. **Ioflupane SPECT (DaTSCAN)** shows a **normal pattern in essential tremor, drug-induced parkinsonism and Alzheimer's disease**, and **abnormalities in PD and other parkinsonian syndromes**.
17. **MRI** identifies midbrain atrophy (PSP), multiple lacunae and leukoaraiosis (vascular), and ventriculomegaly out of proportion to atrophy (NPH).
18. Where diagnosis remains uncertain, **follow-up usually clarifies it** as new signs and symptoms appear.

Management

19. **Treat the cause.** Stop the offending drug in drug-induced parkinsonism (improvement is slow, over weeks to months; about **20% have persistent signs**; there is **no satisfactory treatment**). Shunt for NPH; vascular risk factor control for vascular parkinsonism; levodopa for PD.
20. **Prevention of drug-induced parkinsonism is a crucial issue in geriatric care** — avoid neuroleptics in older people, especially those with dementia, in whom they are ineffective for behavioural symptoms. If untreated, parkinsonian patients eventually become immobile and develop **flexion contractures, pressure sores, malnutrition and aspiration pneumonia**.

Latest Updates (2023–Present)

- **MDS clinical diagnostic criteria for PD** (Postuma et al., *Mov Disord* 2015) are now the international standard alongside the Queen Square criteria, adding **prodromal PD research criteria** based on RBD, hyposmia, constipation and autonomic features.
- **Biological reframing of parkinsonism**: the 2024 *Lancet Neurology* NSD-ISS and SynNeurGe frameworks classify by **α-synuclein seeding positivity, neurodegeneration and genetics** rather than by motor phenotype. Critics (Okubadejo, Okun and Jankovic, *JAMA Neurol* 2024) note limited applicability in low- and middle-income settings, where CSF SAA and DAT imaging are largely unavailable — a point worth making for the Indian context.
- **Skin biopsy for phosphorylated α-synuclein** is emerging as a minimally invasive test to confirm synucleinopathy and to separate MSA (Schwann-cell fibrillar deposits) from PD.

- **Indian epidemiology of parkinsonism:** community crude prevalence ranges from **7 to 60 per 100,000** depending on region and methodology, with urban rates exceeding rural. Screening has always used general neurological instruments (WHO or NIMHANS protocols) rather than PD-specific tools; the **Sarangmath modification of the Tanner screening questionnaire** is the validated Indian PD-specific instrument (Khurana & Gourie-Devi, *Cureus* 2025).
- Historical point examiners like: parkinsonism was described in ancient Indian medical literature as **"kampavata"** (kampa = tremor, vata = dosha) long before James Parkinson's 1817 essay.

Current Practice Update

Aspect	Textbook position	Current evidence-based practice
Diagnostic criteria	Queen Square Brain Bank criteria (3 steps)	**MDS 2015 criteria** plus MDS prodromal research criteria are the international standard; Queen Square remains valid and examinable
Concept of the disease	Clinical syndrome defined by motor signs	Research frameworks define it **biologically** (α-synuclein seeding + neurodegeneration + genetics)
Confirmatory tests	Ioflupane SPECT, MRI	Add **CSF/skin α-synuclein SAA** and MRI-based automated midbrain indices — not yet routine, and scarcely available in India
Screening in the community	Not addressed	Validated Indian PD-specific screening questionnaire exists (Sarangmath et al.), addressing the misclassification problem of generic protocols

Examiner's Tip — lines that earn marks

- Lead with the definition: "**Akinesia — bradykinesia plus hypokinesia — is the defining and obligatory feature of parkinsonism.**"
- Define rigidity precisely: "**constant throughout the range of joint displacement and independent of velocity, unlike spasticity.**"
- Always separate **parkinsonism (syndrome)** from **Parkinson's disease (one cause)** in the first sentence — this is what the short-note is testing.
- Give the geriatric figure: "**20–40% of very late-onset parkinsonism is not idiopathic PD.**"
- Do not forget **paratonia/gegenhalten** as the differential for increased tone in the frail elderly.

Q6. Motor fluctuations in Parkinson's disease

High-Yield Points (Textbook)

Source: Pathy's 6th ed., Chapters 53 and 30; Essentials of Clinical Geriatrics 7th ed.

Definition and types

1. **Motor fluctuations** are variations in motor performance in relation to levodopa dosing, appearing **after several years of levodopa use** as the disease advances.
2. Pathy's describes three patterns: a **delayed effect of levodopa** ("delayed on"), **wearing-off between doses**, and the **on–off phenomenon**.
3. Essentials describes the same clinical spectrum as **morning akinesia, peak-dose dyskinesias and freezing episodes**, collectively termed the on–off phenomenon.
4. Fluctuations are accompanied by **involuntary movements — chorea, athetosis and dystonia** (peak-dose dyskinesia, diphasic dyskinesia and off-period dystonia).
5. In **advanced PD (after 10–15 years)**, fluctuations and motor dyskinesia are **very frequent and remain a challenge for treatment**.

Pathophysiology

6. **Central mechanism:** progressive nigrostriatal degeneration reduces the striatal capacity to store and buffer dopamine, so the clinical effect increasingly follows the **short plasma half-life of levodopa**, producing pulsatile rather than tonic receptor stimulation.
7. **Peripheral mechanism: gastric emptying is delayed in at least 70% of PD patients** attending neurological clinics, even without levodopa therapy, and levodopa slows it further.
8. Levodopa **retained in the stomach is metabolised to dopamine and becomes unavailable for systemic absorption**, contributing to nausea, bloating and impaired nutrition.
9. In patients with the on–off phenomenon, **gastric emptying may normalise during the "on" phase**; conversely, **variation in the rate of emptying causes erratic levodopa absorption and thereby contributes to the on–off phenomenon** — a two-way relationship.
10. **Dietary protein competes with levodopa** for absorption, which is why standard levodopa is given 30 minutes before meals.
11. **Constipation** is associated with reduced efficacy of medication due to reduced absorption, and occurs in about half of PD patients.

Assessment

12. Take a **patient or carer diary** of on and off periods, mapped against dose times and meals; review total daily levodopa, dose interval, formulation and adherence.
13. Exclude reversible contributors: **constipation, intercurrent infection, dehydration, malabsorption, protein timing, and drugs that antagonise dopamine** (metoclopramide, neuroleptics).
14. Look specifically for **early-morning akinesia**, **early-morning and off-period dystonia** and **freezing**, since each has a different solution.

Management

15. **First step: revise the timing of levodopa administration.** Fractionate the same total dose into smaller, more frequent doses to reduce peak-dose dyskinesia and shorten off periods.
16. **Dispersible Madopar** achieves peak plasma concentration faster and is used for **early-morning akinesia**, allowing the first standard dose to be taken at the same time.
17. **COMT inhibitors (entacapone, tolcapone)** decrease dopamine degradation and increase and prolong the levodopa effect — they are **useful in older patients with motor fluctuations and/or levodopa-induced dyskinesia**.
18. **Stalevo** (levodopa/carbidopa 50/12.5, 100/25 or 150/37.5 mg with fixed entacapone 200 mg) is indicated for patients with **end-of-dose fluctuation not stabilised by levodopa–decarboxylase inhibitor alone**.
19. **Amantadine 200–400 mg/day** is effective in lessening **peak-dose dyskinesia**. Adjust the dose in renal impairment; **delirium is frequent in old patients**.
20. **Direct duodenal infusion of levodopa** is advocated as a solution to erratic gastric absorption; increasing the decarboxylase-inhibitor to levodopa ratio has also been proposed. **Subcutaneous apomorphine infusion** has limited data but shows benefit, including for swallowing.
21. **Bilateral deep brain stimulation of the subthalamic nucleus or globus pallidus interna** produces sustained improvement in motor function, especially for levodopa-responsive symptoms, and **reduces drug-induced involuntary movements**. It is offered to **cognitively intact patients with severe disability from motor complications refractory to best medical treatment**; severe comorbidity is a limitation. Pathy's notes that efficacy in patients over 70 is similar to that in younger patients, with no age-related increase in adverse effects, but that **disability continues to progress because neurodegeneration continues**.
22. **Rehabilitation sessions should be held at the time of the peak effect of levodopa** to maximise participation.
23. Manage **constipation actively** (hydration, fibre, macrogol, psyllium) because it both worsens absorption and causes nausea and anorexia; entacapone-induced diarrhoea can be turned to advantage.

Latest Updates (2023–Present)

- **Crexont (IPX203)**, FDA-approved **August 2024**, is an extended-release carbidopa/levodopa capsule specifically developed for **wearing-off**; its design reduces peaks and troughs, which matters increasingly as gastric emptying slows with age.
- **Vyalev (foslevodopa/foscarbidopa)**, FDA-approved **October 2024**, delivers levodopa prodrugs by **24-hour subcutaneous pump**, achieving continuous dopaminergic stimulation and **bypassing the gastrointestinal tract entirely** — the modern answer to the erratic gastric absorption Pathy's describes,

without surgery.

- **Onapgo (subcutaneous apomorphine infusion)**, FDA-approved **February 2025** for "off" episodes, filling a long-standing US gap; continuous apomorphine infusion already had a well-established safety profile in Europe and the UK.
- **Adaptive (closed-loop) DBS.** The FDA approved Medtronic **BrainSense Adaptive DBS** in **February 2025** on the strength of the **ADAPT-PD** multicentre randomised crossover trial — the largest and longest study of aDBS. Stimulation is titrated in real time to sensed **beta-band local field potentials**, and is specifically indicated for **patients with motor fluctuations despite medical therapy**; the accompanying Electrode Identifier reduces initial programming time by about **85%**.
- **Tavapadon** (D1/D5 partial agonist) added to levodopa gave an extra **1.1 hours of daily "on" time without troublesome dyskinesia** and a significant reduction in "off" time in Phase 3 TEMPO-3; NDA filed September 2025 (MDS Congress 2025).
- **Additional off-time adjuncts** not covered in the textbooks: **opicapone** (once-daily COMT inhibitor), **safinamide** and **istradefylline** (adenosine A2A antagonist), and **inhaled levodopa** and **sublingual apomorphine film** as on-demand rescue for off episodes.

Current Practice Update

Aspect	Textbook position	Current evidence-based practice
First-line manoeuvre	Revise timing; fractionate levodopa doses	Unchanged — still the correct first step before adding drugs or devices
Oral adjuncts for wearing-off	Entacapone/tolcapone; Stalevo	Add **opicapone** (once daily), **safinamide** and **istradefylline**; **Crexont** ER capsules as a levodopa alternative
Continuous dopaminergic delivery	Duodenal infusion; apomorphine "limited data"	**Vyalev** (SC levodopa prodrug, 24 h) and **Onapgo** (SC apomorphine) are licensed; intestinal gel established; **ND0612** pending
Rescue for sudden "off"	Not addressed	**Inhaled levodopa** and **sublingual apomorphine film** are licensed on-demand rescue therapies
DBS	Continuous open-loop stimulation; "not currently used in the elderly"	**Adaptive closed-loop DBS approved February 2025 (ADAPT-PD)**; age >70 no longer a barrier

Examiner's Tip — lines that earn marks

- Classify first: "**Delayed on, wearing-off, and the on–off phenomenon**, with chorea, athetosis and dystonia as the associated dyskinesias."
- Explain both mechanisms: "**loss of striatal dopamine buffering makes the effect track the short plasma half-life**" and "**gastric emptying is delayed in at least 70% of PD patients, causing erratic absorption**."
- Answer in a stepwise algorithm: **timing revision → dispersible levodopa for morning akinesia → COMT inhibitor/Stalevo → amantadine for peak-dose dyskinesia → continuous delivery → DBS**.
- Add the geriatric caveats: **amantadine needs renal dose adjustment and causes delirium**; **DBS requires cognitive intactness**.
- One current fact closes it: "**Adaptive DBS driven by beta-band local field potentials was approved in February 2025 for exactly this indication.**"

Q7. Non-motor symptoms of Parkinson's disease

High-Yield Points (Textbook)

Source: Pathy's 6th ed., Chapters 53, 30 and 42; Essentials of Clinical Geriatrics 7th ed.

Overview and classification

1. Non-motor symptoms (NMS) are common in PD and, in the geriatric population, contribute more to disability, institutionalisation and carer burden than the motor features.
2. Pathy's lists them as **bradyphrenia (slowness of mental function), depression, memory problems and dementia, constipation, bladder dysfunction and autonomic dysfunction** (gastrointestinal, sexual, cardiovascular). Essentials adds **psychosis, anxiety and sleep disturbance**.
3. They can be grouped as **neuropsychiatric, cognitive, autonomic, sleep and sensory**.

Neuropsychiatric and cognitive

4. **Neuropsychiatric symptoms are dominated by anxiety and depressive syndrome.** The anxiety disorders encountered are mainly **generalised anxiety disorder and obsessive–compulsive disorder**.
5. **Major depressive episodes are frequent — about 50% of patients may be affected during the course of the disease.** Diagnosis is difficult because fatigue, sleep disorders and lack of interest occur in PD in the absence of overt depression.
6. **Cognitive impairment is never a sign of early PD** and is observed only after several years of evolution (**PD-related dementia**). Dementia occurring within a few months of parkinsonism indicates **dementia with Lewy bodies** instead.
7. Visual hallucinations, delusions and delirium occur in advanced disease and are frequently **dose-related adverse effects of levodopa** rather than the disease itself.

Autonomic

8. **Autonomic disorders are dominated by orthostatic hypotension and constipation.**
9. **Orthostatic hypotension** is directly related to autonomic dysfunction and is **often worsened by antiparkinsonian drugs**. It is defined as a fall in **systolic BP of 20 mmHg or diastolic BP of 10 mmHg within three minutes of standing from supine**. It is often asymptomatic but can cause discomfort or syncope. **Repeated search for orthostatic hypotension is part of the monitoring of any parkinsonism patient.**
10. **Constipation is found in about 50% of PD patients**, related to impaired bowel motility, sphincter dysfunction and poor physical activity. It arises from **dyssynergic defecation due to incoordination of the pelvic floor musculature** and from the constipating effect of PD medication.
11. Constipation is a **key symptom that must be actively elicited** — it causes abdominal discomfort, nausea and reduced appetite, and is **associated with reduced efficacy of medications due to reduced absorption**.
12. **Gastric emptying is delayed in at least 70%** of PD patients, causing nausea and bloating and impairing nutrition and drug absorption. Small-intestinal, colonic and anorectal dysmotility are also common, with prolonged oro-caecal transit.
13. Other autonomic symptoms: **sweating, drooling, sexual dysfunction and urinary frequency, urgency and dysuria**. Urinary symptoms may be attributed to PD only after excluding **urinary infection, bladder or prostate disease and anticholinergic drugs**.
14. **Swallowing:** heartburn is common from impaired acid clearance. The effects of levodopa and anticholinergics on swallowing are **inconsistent** — either may improve or worsen dysphagia. Limited data indicate benefit from **subcutaneous apomorphine infusion**.

Sensory, sleep and advanced-stage symptoms

15. **Impaired olfaction** is an early sign, reflecting early involvement of the olfactory pathways by α-synuclein pathology. **Hyposmia and visual hallucination are supportive criteria** in the Queen Square Brain Bank criteria.
16. **REM sleep behaviour disorder** — loss of normal REM atonia with acting-out of dreams — results from brainstem circuit dysfunction and is strongly associated with neurodegenerative disease; melatonin is used in treatment.
17. In **advanced PD**, patients experience **insomnia, pain, sweats, orthostatic hypotension, dysphagia, dysarthria, drooling of saliva, urinary dysfunction, and cognitive or psychiatric disorders** including depression, anxiety, visual hallucinations and dementia.

Management principles

18. **The governing rule (Pathy's Table 53.4): every non-motor treatment is considered only after adjusting levodopa therapy**, because **levodopa itself does not improve non-motor symptoms, posture or gait**, and many non-motor problems are levodopa side effects.
19. **Best-evidence treatments:** domperidone for nausea and vomiting (ECG monitoring for QT prolongation; **metoclopramide is contraindicated**); **venlafaxine** for depression; **clozapine or pimavanserin** for hallucinations and delusions; **rivastigmine** for dementia and for apathy; **droxidopa** for orthostatic hypotension; **sleep hygiene** for insomnia; **probiotics, prebiotic fibre, hydration and macrogol** for constipation; **botulinum toxin into salivary glands** for severe drooling; **sildenafil** for erectile dysfunction.
20. **Other options:** SSRIs, cognitive-behavioural therapy or rTMS for depression; SSRIs or CBT for generalised anxiety (no best-evidence treatment exists); donepezil for dementia; midodrine, fludrocortisone and elastic venous compression for orthostatic hypotension; melatonin for insomnia; lubiprostone or psyllium for resistant constipation; solifenacin (low brain penetration) for urinary urgency. **Pramipexole** is effective for PD depression but may induce delirium and should not be first choice in older patients. **No effective treatment exists for mild cognitive impairment in PD.**
21. **Clozapine practicalities:** start 12.5 mg daily, titrate in five-day increments to a usual 25–50 mg; contraindicated in neutropenia; blood count weekly for two months then twice monthly; **discontinue permanently if the neutrophil count falls below 1500/mm^3**. Pimavanserin, a **5-HT2A inverse agonist**, is effective and safe without haematological monitoring.
22. **Timed toileting every 2–4 hours and 30 minutes after meals** improves continence and prevents constipation, particularly in patients with coexisting cognitive impairment.

Latest Updates (2023–Present)

- **Non-motor features are now recognised as the earliest biological stage.** The **NSD-ISS** (*Lancet Neurology* 2024) formally stages the disease from biomarker positivity through **non-motor manifestations** to motor signs and functional impairment — the first framework in which non-motor symptoms are a defining, not an accessory, feature.
- **Isolated REM sleep behaviour disorder is prodromal synucleinopathy.** α-Synuclein seed amplification assay is positive in the great majority of patients with isolated RBD (PPMI, *Lancet Neurology* 2023), and **dermal phospho-α-synuclein deposits confirm RBD as prodromal PD** (Doppler et al.). In geriatric practice, a new RBD in an older adult warrants counselling and follow-up rather than reassurance.
- **Indian non-motor profile.** The pan-Indian multicentre study (Kishore et al., *Movement Disorders* 2025) found the motor and non-motor spectrum in Indian PD broadly similar to European populations, but with an **earlier age at onset**, implying a longer period of non-motor burden and greater caregiving demand across the family life cycle.
- **Off-time reduction improves non-motor fluctuation.** The new continuous delivery systems (**Vyalev** October 2024, **Onapgo** February 2025) and extended-release **Crexont** (August 2024) reduce off periods, during which non-motor symptoms — anxiety, pain, sweating, akathisia and bladder urgency — typically cluster.
- **New safety signal relevant to non-motor care:** the **FDA warning of March 2026 on vitamin B6 deficiency and seizures** with all carbidopa/levodopa products. B6 depletion also underlies levodopa-associated peripheral neuropathy, an under-recognised non-motor complication in long-treated patients.
- **Exercise remains one of the few interventions with broad non-motor benefit** — combined aerobic, strengthening, balance and stretching programmes improve balance in PD, and structured dance and exercise programmes are increasingly reported to benefit mood, sleep and quality of life alongside motor outcomes.

Current Practice Update

Aspect	Textbook position	Current evidence-based practice
Status of non-motor symptoms	Accompanying features of the disease	**Defining features of the earliest disease stages** in the 2024 biological staging systems
REM sleep behaviour disorder	Associated with neurodegenerative disease	Recognised as **prodromal synucleinopathy**, confirmable by α-synuclein SAA and skin biopsy
PD psychosis	Clozapine best evidence; pimavanserin noted as unavailable in Europe	**Pimavanserin is now the first-line antipsychotic for PD psychosis** where licensed; clozapine remains an effective and inexpensive alternative and is the practical choice in India
Orthostatic hypotension	Droxidopa (noted as unavailable in Europe); midodrine, fludrocortisone	Droxidopa now more widely available; in India, **midodrine and fludrocortisone with non-pharmacological measures remain the mainstay**
Levodopa and non-motor symptoms	"Does not improve non-motor symptoms"	Holds true for the symptoms themselves, but **reducing off-time with continuous delivery reduces non-motor fluctuation**

Examiner's Tip — lines that earn marks

- Give the exact orthostatic hypotension definition — "**a fall of 20 mmHg systolic or 10 mmHg diastolic within three minutes of standing**" — and add that "**repeated search for orthostatic hypotension is part of the monitoring of any parkinsonism patient**."
- Two numbers earn marks: "**depression affects about 50%**" and "**constipation affects about 50%**."
- State the timing rule: "**cognitive impairment is never a sign of early PD**" — dementia within months of parkinsonism means DLB.
- Reproduce Table 53.4 as a two-column table (best evidence vs other options) — it is the highest-yield single element of this answer.
- Finish with the governing principle: "**Each non-motor treatment should be considered only after adjusting levodopa therapy.**"

Q8. Progressive supranuclear palsy — clinical features, management and prognosis RECURS

High-Yield Points (Textbook)

Source: Pathy's 6th ed., Chapters 53 (Table 53.2, Figure 53.1) and 46; Essentials of Clinical Geriatrics 7th ed., Table 6-7.

Definition and importance

1. **Progressive supranuclear palsy (PSP) is a neurodegenerative disorder that is frequently misdiagnosed as Parkinson's disease.**
2. It is one of the causes of extrapyramidal syndrome **frequently observed as very late-onset parkinsonism**, and is listed among the **nonreversible dementias** (Essentials, Table 6-7).
3. The disease is characterised by an **extrapyramidal syndrome associated with complex eye movement dysfunction**.

Clinical features

4. **Tremor is infrequent** — its absence in a parkinsonian patient should raise suspicion.
5. **Rigidity predominates at the spine (axial rigidity)** rather than in the limbs; **neck dystonia (retrocollis)** is characteristic.
6. **Posture and gait abnormalities are frequent at an early stage and lead to repeated falls**, which are also favoured by **attentional and visual problems**. Early postural instability with falls in the first year is the classical distinguishing feature from PD.
7. **Eye movement abnormalities:** inability to **voluntarily shift the gaze downward**, **impaired saccades**, and **difficulty controlling the eyelids**.
8. **Motor symptoms are unresponsive to levodopa** — a key discriminator, assessable by history or levodopa challenge.
9. As the disease progresses, **changes in personality, dementia and swallowing problems are common**.
10. **MRI shows a typical pattern relating to midbrain atrophy**: the midbrain aspect becomes **convex, whereas the normal midbrain aspect is concave** (Figure 53.1). This underlies the eponymous "hummingbird" and "morning glory" appearances.
11. **Ocular electrophysiology** demonstrating impaired saccades helps in recognising the disease.
12. PSP must be included in the differential diagnosis of **normal pressure hydrocephalus**, since both cause gait disturbance and cognitive change in older people.

Distinguishing PSP from Parkinson's disease

Feature	Parkinson's disease	Progressive supranuclear palsy
Onset pattern	Asymmetric or unilateral	Symmetric from the outset
Rest tremor	Characteristic, 4–6 Hz	**Infrequent**
Rigidity	Limb-predominant	**Axial (spinal) predominant**, with neck dystonia
Falls	Late, in advanced disease	**Early and repeated**
Eye movements	Normal (apart from age-related upgaze limitation)	**Vertical — especially downgaze — supranuclear palsy; impaired saccades; eyelid control problems**
Levodopa response	Excellent (70–100%), sustained for years	**Unresponsive**
Cognition	Dementia only after several years	Personality change and dementia as the disease progresses
MRI	Usually unremarkable	**Midbrain atrophy — convex instead of concave outline**
Ioflupane SPECT	Abnormal	Abnormal (does not separate the two)

Management

13. **There is no disease-modifying therapy.** Management is symptomatic, supportive and multidisciplinary.
14. **Give an adequate trial of levodopa in every case** — response is poor, but a small number derive transient benefit, and a documented negative response supports the diagnosis.
15. **Falls prevention is the single most important intervention:** physiotherapy, gait and balance training, weighted or wheeled walkers rather than simple canes, environmental hazard modification, appropriate footwear and hip protectors.
16. **Speech and swallow therapy** for dysarthria, drooling and dysphagia; early dietitian involvement; timely discussion of feeding options as dysphagia progresses.
17. **Ocular management:** lubricating drops for reduced blink, reading aids and prisms for downgaze palsy, and **botulinum toxin** for blepharospasm or eyelid-opening apraxia.
18. **Avoid neuroleptics and anticholinergics.** Treat depression; manage drooling with botulinum toxin injection into salivary glands as in PD.
19. **Deep brain stimulation is not indicated** in PSP.
20. **Carer support, advance care planning and palliative-care involvement** should begin early, given the rapid trajectory and predictable swallowing failure.

Prognosis

21. PSP is **relentlessly progressive**, with earlier loss of independence than PD. Death typically results from **aspiration pneumonia and the complications**

of immobility.

Latest Updates (2023–Present)

- **PSP is now defined as a spectrum, not a single syndrome.** The **MDS-PSP criteria** operationalise diagnosis across four functional domains — **ocular motor dysfunction, postural instability, akinesia and cognitive dysfunction** — with defined levels of certainty and **eight clinical variants**, including **PSP-RS (Richardson's syndrome), PSP-P (parkinsonism), PSP-PGF (progressive gait freezing) and PSP-CBS (corticobasal syndrome)**. The early "**suggestive of PSP**" category was **prospectively validated** in the DESCRIBE-PSP and ProPSP cohorts (Quattrone et al., *Movement Disorders* 2025).
- **Pathology is a 4-repeat tauopathy.** PSP is characterised by accumulation of abnormal **4-repeat (4R) tau**; the **MAPT H1 haplotype** is the major genetic risk factor.
- **Prognosis is now subtype-specific** — a high-yield update. A global systematic review (Kukkle et al., *Movement Disorders Clinical Practice* 2026) reports **global prevalence 5–6.4 per 100,000**, with **PSP-RS progressing rapidly with survival of 5–7 years**, while **PSP-P has a milder course with survival of 8–12 years**. Regional differences reflect MAPT haplotype frequency, dietary neurotoxins, industrial pollutants and access to care.
- **Biomarkers.** Second-generation tau-PET with **18F-PI-2620** detects 4R tau (basal ganglia readout) and predicts subsequent increases in diagnostic certainty (LMU Munich prospective cohort). Fluid biomarkers — **neurofilament light chain (NfL) and GFAP** measured on the SIMOA platform — track progression and are being used as trial endpoints (*Brain Communications* 2025).
- **Therapeutic pipeline.** There is still **no approved disease-modifying therapy**. Tau-directed strategies — anti-tau antibodies, antisense oligonucleotides and **sodium selenate (SEL003 trial)** — are in trial. Reasons for past failures include late-stage intervention, poor patient selection, absence of prodromal biomarkers and inadequate outcome measures.
- **Indian relevance:** Indian movement-disorder centres contributed to the 2026 global review; PSP is under-recognised in India, and diagnostic accuracy has improved measurably since adoption of the MDS-PSP criteria. Tau-PET is not available outside a few research centres, so **MRI midbrain measures and clinical criteria remain the practical tools**.

Current Practice Update

Aspect	Textbook position (Pathy's 6th ed.)	Current evidence-based practice
Concept of the disease	A single disorder: gaze palsy, early falls, axial rigidity	**A spectrum of 4R-tauopathy syndromes with eight recognised clinical variants**
Diagnostic criteria	Clinical description only	**MDS-PSP criteria** with four domains, certainty levels and a "suggestive of PSP" early category
Pathological basis	Not specified in the parkinsonism chapter	Explicitly a **4-repeat tauopathy**, MAPT H1 haplotype the major genetic risk
Investigations	MRI midbrain atrophy; ocular electrophysiology	Add **18F-PI-2620 tau-PET** and **plasma/CSF NfL and GFAP** — research and tertiary use
Prognosis	Progressive, unspecified duration	**Subtype-specific: PSP-RS 5–7 years, PSP-P 8–12 years**
Treatment	Symptomatic, levodopa-unresponsive	Unchanged — **no disease-modifying therapy**; tau-directed agents in trial

Extra Points to Remember — this topic recurs

- **Mnemonic for the classical (Richardson's) syndrome — "PSP FALLS": P**ostural instability with early falls; **S**upranuclear vertical gaze palsy (downgaze first); **P**seudobulbar features and personality change; **F**alls in the first year; **A**xial rigidity and retrocollis; **L**evodopa non-response; **L**ate dysphagia and dementia; **S**accades impaired with no tremor.
- **The three-line answer if asked in viva:** "PSP is a sporadic 4-repeat tauopathy presenting with early falls, vertical supranuclear gaze palsy, axial rigidity and levodopa non-response; MRI shows midbrain atrophy with a convex rather than concave outline; management is symptomatic, and Richardson's syndrome carries a survival of 5–7 years."
- **The single best discriminator to state first** is not the gaze palsy (which may appear late) but **early falls within the first year in a symmetric, tremor-free, levodopa-unresponsive parkinsonian patient**.
- **Radiological signs worth naming:** hummingbird/penguin sign on sagittal MRI, morning glory sign on axial MRI, and the **Magnetic Resonance Parkinsonism Index (MRPI)** as a quantitative midbrain–pons measure.
- **Do not confuse** the **supranuclear** palsy of PSP (voluntary gaze lost, reflex vestibulo-ocular movements preserved — doll's-eye manoeuvre overcomes it) with a **nuclear or infranuclear** palsy, where reflex movements are also lost. Examiners commonly ask this.
- **Common exam trap:** ageing itself restricts upward conjugate gaze, so **downgaze limitation, not upgaze limitation, is the meaningful sign** in an older patient.

Examiner's Tip — lines that earn marks

- Open with: "**PSP is a neurodegenerative disorder frequently misdiagnosed as Parkinson's disease, characterised by an extrapyramidal syndrome with complex eye movement dysfunction.**"
- Say "**no tremor, axial rigidity, early repeated falls, downgaze palsy, levodopa-unresponsive**" as a single string — five marks in one sentence.
- Describe the MRI precisely: "**convex midbrain aspect indicating atrophy, where the normal midbrain aspect is concave.**"
- Add the modern framing: "**a 4-repeat tauopathy with eight MDS-defined clinical variants**."
- Give subtype-specific prognosis — **PSP-RS 5–7 years, PSP-P 8–12 years** — and state that death is usually from **aspiration pneumonia**.

Q9. Benign / essential tremor — presentation, differential diagnosis and treatment

High-Yield Points (Textbook)

Source: Pathy's 6th ed., Chapter 54 (Non-Parkinsonian movement disorders in the elderly), Tables 54.3 and 54.4; Chapter 53.

Definition and epidemiology

1. **Essential tremor (ET) is the most common tremor disorder and the most common movement disorder**, with an estimated prevalence of **up to 3% in the general population and up to 5% in adults over 60**.
2. **ET becomes more common with age** — in one study the prevalence in adults over 95 was as high as **21.7%**.
3. Tremor of some kind is near-universal: an estimated **98.9%** prevalence in a large population study, the overwhelming majority being **physiological**.
4. Other tremor disorders are less common and are **often missed or misdiagnosed as essential tremor** — especially **dystonic tremor and parkinsonian tremor**.

Pathophysiology

5. The exact mechanism is unknown, but tremor disorders arise from **dysfunction in networks involving the inferior olive, cerebellum, thalamus, basal ganglia and cortex**.
6. Current theory: the **basal ganglia network triggers the onset of tremor**, while the **cerebellar network — predominantly affected in essential tremor — modulates its amplitude**. Which part of the network is affected determines the phenomenology — **rest tremor in PD, action-induced tremor in ET**.
7. Because tremor arises from dysfunctional CNS networks, **tremor disorders form a spectrum with overlapping clinical features**.

Presentation

8. ET is an **action-induced (postural and kinetic) tremor**. Features making ET more likely are the presence of an **intention tremor** and an **action-induced kinetic tremor of greater amplitude than the postural tremor**.
9. **Rest tremor is not a typical feature of ET** and renders a diagnosis of parkinsonism more likely.
10. ET may be **asymmetric but is usually not unilateral**; unilateral tremor is more commonly parkinsonian, dystonic or task-specific.
11. An **isolated head tremor, or head tremor that started before other body parts**, points away from ET and towards dystonia.
12. **Ethanol can improve the tremor**, but this is not unique to ET and also occurs in dystonia. **Increased coffee intake and smoking may cause or worsen tremor.**
13. A **strong family history of tremor** is often found in ET, but may also occur in dystonia (for example in certain Jewish populations).

Differential diagnosis

14. **Parkinsonian tremor** — look for rest tremor with rigidity, bradykinesia and postural instability, plus minor signs such as **hypomimia, loss of prosody and camptocormia**. Note that slow/rapid alternating movements without decrement, and reduced arm swing, can also occur in dystonia and ET.
15. **Dystonic tremor** — abnormal posturing such as **spooning of the hand, abnormal posturing or flexion of the thumb or hand**; **focal muscle hypertrophy, torticollis or a geste antagoniste**; the tremor may be **arrhythmic**, and dystonic head tremor may be **directional**. Voice tremor with **voice breaks and a spasmodic quality** suggests dystonia.
16. **The spiral test** is a valuable and easy clinic test: patients with ET draw spirals with **one clear single axis**, whereas dystonic tremor spirals typically have **multiple axes or no clear axis at all**.
17. **Cerebellar tremor** — intention tremor accompanied by **dysmetria, scanning speech, saccadic eye movement abnormalities and truncal ataxia**. MRI with and without contrast is required, since a mass, stroke or multiple sclerosis may cause secondary tremor.
18. **Functional tremor** — features of **variability, distractibility, entrainment and suggestibility**.
19. **Drug-induced tremor** is especially important in the elderly with polypharmacy. Common culprits: **caffeine, ethanol, nicotine, beta-adrenergic agonists (albuterol, salmeterol), tricyclic antidepressants, lithium, SSRIs, neuroleptics, valproate, amiodarone, ciclosporin, tacrolimus and metoclopramide**; Chapter 53 adds bronchodilators and theophylline. The medication list must be scrutinised and any tremorogenic drug stopped if possible.
20. **Other causes:** hyperthyroidism (recognised by other signs and TSH estimation), orthostatic tremor, late-onset Wilson's disease (rare in the elderly but considered in complex cases), and the extrapyramidal syndrome of ageing.

Investigation

21. **In most cases of tremor in the elderly no further workup is required** — history and examination are sufficient to establish the diagnosis. Basic blood work, **liver function tests and thyroid studies** can at times be helpful.
22. **EMG is very beneficial for diagnosing orthostatic tremor**. **DaTSCAN** is normal in ET and abnormal in PD and other parkinsonian syndromes, and may establish the correct diagnosis in difficult cases.

Treatment

23. **There is no curative therapy for essential tremor**, and the **indication for symptomatic therapy depends solely on the functional and social impairment the patient experiences** — a critical geriatric principle.
24. **First-line pharmacotherapy: propranolol and primidone**, which induce a **30–70% reduction in tremor amplitude**.
25. Both cause significant side effects leading to **frequent discontinuation — bradycardia with propranolol; nausea, dizziness and drowsiness with primidone**. In older patients also consider bronchospasm, hypotension and masking of hypoglycaemia with propranolol.
26. **Second-line: topiramate, gabapentin and benzodiazepines.**

27. **Surgical therapy** gives a more pronounced reduction of tremor amplitude than drugs: **deep brain stimulation with the typical target being the ventral intermediate (Vim) nucleus of the thalamus**, **gamma knife surgery**, and **focused ultrasound thalamotomy**. Given their invasive nature, these are **reserved for patients with severe tremor refractory to medication**.

Latest Updates (2023–Present)

- **Staged bilateral MRgFUS thalamotomy is now approved.** The FDA approved **staged bilateral** magnetic resonance-guided focused ultrasound thalamotomy for essential tremor, with the **second side treated at least nine months after the first**. The pivotal open-label trial was published by Kaplitt, Krishna, Eisenberg, Elias and colleagues in ***JAMA Neurology* 2024**.
- **Efficacy comparable to bilateral DBS.** A 2025 *Movement Disorders* analysis found the efficacy of bilateral MRgFUS and bilateral DBS to be **comparable**, and probabilistic lesion mapping identified **two sweet spots — the dorsal Vim–Vop border and the ventral Vim/posterior subthalamic area**. A prospective series in *Brain Communications* 2025 (20 patients, 6-month follow-up on the Clinical Rating Scale for Tremor) confirmed benefit, with **balance and gait impairment as the adverse events to counsel about** — particularly relevant to falls risk in the elderly.
- **Classification has changed.** The MDS consensus classification of tremor uses **two axes** — clinical characteristics and aetiology — and introduces the category "**essential tremor plus**" for isolated tremor with additional soft neurological signs (mild dystonic posturing, impaired tandem gait, questionable rest tremor). Pathy's Chapter 54 already uses the phrase "ET or ET plus" without defining it.
- **Position statement:** the American Society for Stereotactic and Functional Neurosurgery position statement on MRgFUS for essential tremor (*Neurosurgery* 2020) remains the reference document; in the USA all treatment sites are approved for Medicare payment.
- **Indian context:** propranolol and primidone are inexpensive and widely available; topiramate and gabapentin are readily obtainable. **MRgFUS is available in a small number of Indian tertiary centres** (and is costly and largely out-of-pocket), so **DBS and stereotactic thalamotomy remain the practical surgical options** for refractory tremor in most of the country. In differential diagnosis, remember that **Wilson's disease is relatively more common in India and presents at a younger age**, so serum ceruloplasmin, 24-hour urinary copper and slit-lamp examination should be checked in any tremor with atypical features, hepatic disease or psychiatric symptoms.

Current Practice Update

Aspect	Textbook position (Pathy's 6th ed.)	Current evidence-based practice
Nomenclature	"Essential tremor" (with the phrase "ET or ET plus" used in passing)	Formal MDS two-axis classification with a defined "**essential tremor plus**" category
First-line drugs	Propranolol and primidone, 30–70% amplitude reduction	Unchanged — still first line, and still frequently discontinued for side effects
Focused ultrasound	Mentioned as a newer option for refractory tremor	**Staged bilateral MRgFUS is FDA-approved**, second side at least 9 months after the first (JAMA Neurology 2024)
MRgFUS vs DBS	Both listed as options for refractory tremor	Efficacy appears **comparable**; MRgFUS is incisionless but **lesional and irreversible**, with gait and balance adverse effects
Indication to treat	Depends solely on functional and social impairment	Unchanged — and reinforced as a geriatric principle: treat disability, not the tremor amplitude

Examiner's Tip — lines that earn marks

- Lead with epidemiology: "**the most common movement disorder — up to 3% of the population, up to 5% over 60, and as high as 21.7% over 95.**"
- State the network theory: "**the basal ganglia network triggers tremor onset while the cerebellar network, predominantly affected in ET, modulates amplitude.**"
- Contrast in one line: "**rest tremor in Parkinson's disease, action-induced tremor in essential tremor**" — and add that DaTSCAN is normal in ET.
- Name the **spiral test** — single axis in ET, multiple or no axis in dystonic tremor. Examiners love this because it is a bedside test.
- State the treatment principle verbatim: "**There is no curative therapy, and the indication for symptomatic treatment depends solely on the functional and social impairment the patient experiences.**"
- Finish the ladder: **propranolol/primidone → topiramate, gabapentin, benzodiazepines → Vim DBS, gamma knife or focused ultrasound thalamotomy for refractory cases.**

Cross-cutting revision aid — the night before the examination

One-line signatures to recall under pressure

Condition	The line that identifies it
Parkinson's disease	Asymmetric onset, rest tremor, excellent sustained levodopa response, dementia only after years
Progressive supranuclear palsy	Symmetric, no tremor, early falls, downgaze palsy, axial rigidity, levodopa-unresponsive, convex midbrain on MRI
Multiple system atrophy	Early severe dysautonomia with pyramidal or cerebellar signs
Corticobasal degeneration	Alien hand, myoclonus, dystonia, cortical sensory loss
Dementia with Lewy bodies	Dementia within months of parkinsonism, hallucinations, fluctuation, neuroleptic sensitivity
Vascular parkinsonism	Onset or worsening within one month of stroke; multilacunar state; pseudobulbar features
Drug-induced parkinsonism	Neuroleptic exposure, dyskinesia frequent, no tremor, normal DaTSCAN, 20% persist after withdrawal
Normal pressure hydrocephalus	Gait + incontinence + cognitive slowing; ventriculomegaly out of proportion to atrophy; no tremor or rigidity
Essential tremor	Bilateral action tremor, family history, alcohol-responsive, normal DaTSCAN, single-axis spiral

Numbers most likely to be asked

- PD prevalence **~1% over age 65**; median age at diagnosis **60**; mean survival from diagnosis **15 years**; family history in **6–10%**.
- Non-PD causes of very late-onset parkinsonism: **20–40%**. Persistent signs after stopping the offending drug: **20%**.
- Levodopa **300–600 mg/day** in early disease; challenge dose **150–250 mg**; rest tremor frequency **4–6 Hz**.
- Depression in PD **~50%**; constipation **~50%**; delayed gastric emptying **≥70%**; parkinsonism in Alzheimer's disease **~15%**.
- Orthostatic hypotension: **≥20 mmHg systolic or ≥10 mmHg diastolic within 3 minutes**. Clozapine stop threshold: **neutrophils <1500/mm^3**.
- Essential tremor: **3%** general population, **5%** over 60, **21.7%** over 95; propranolol/primidone reduce amplitude by **30–70%**.
- PSP: prevalence **5–6.4/100,000**; survival **PSP-RS 5–7 years, PSP-P 8–12 years**.
- India: PD crude prevalence **7–60/100,000**, age-adjusted **53–192/100,000**; Parsi community of Mumbai **328/100,000** (not generalisable); parkinsonism in Bangalore old-age homes **17.8%**.

Dates and approvals worth quoting (2024–2026)

- **February 2024** — NSD-ISS and SynNeurGe biological frameworks published together in *Lancet Neurology*.
- **August 2024** — Crexont (IPX203 extended-release carbidopa/levodopa) FDA-approved.
- **September 2024** — staged bilateral MRgFUS thalamotomy trial for essential tremor published in *JAMA Neurology*.
- **October 2024** — Vyalev (foslevodopa/foscarbidopa), first 24-hour subcutaneous levodopa infusion, FDA-approved.
- **February 2025** — Medtronic BrainSense **Adaptive DBS** FDA-approved on ADAPT-PD; Onapgo subcutaneous apomorphine infusion FDA-approved; **Exenatide-PD3 published negative** in *The Lancet*.
- **September 2025** — tavapadon (D1/D5 partial agonist) NDA submitted on the TEMPO Phase 3 programme.
- **March 2026** — FDA label warning for **vitamin B6 deficiency and seizures** across all carbidopa/levodopa products.
- **June 2026** — *The Lancet* issued an Expression of Concern regarding the Exenatide-PD3 publication.

MD Geriatric Medicine

High-Yield 15-Mark Answers

Neurology · Neuro-ophthalmology · Environmental Emergencies

Compiled from **Pathy's Principles and Practice of Geriatric Medicine (6th ed.)**
and **Essentials of Clinical Geriatrics (7th ed.)**
with Latest Updates (2023–2026) and Indian Clinical Context

14 Questions | Prepared for Sudarsan

Contents

1. Epilepsy in the elderly — definition, pathophysiology, clinical features, investigations, treatment
2. Coma in the elderly
3. Subdural haematoma RECURS ×2
4. Lacunar infarcts
5. Subacute combined degeneration of the cord — presentation and treatment
6. Motor neuron disease — presentation, evaluation and treatment
7. Peripheral neuropathy — common causes and clinical approach
8. Neuritic (neuropathic) pain
9. Management of extracranial carotid artery stenosis
10. Bedside assessment of swallow after acute stroke
11. Visual impairment — age-related causes and approach RECURS
12. Macular degeneration — presentation, aetiology, diagnosis, treatment, prevention
13. Heat stroke
14. Heat exhaustion

Every answer follows the same architecture: 15–20 high-yield textbook points → Latest Updates (2023–present, cited inline) → Current Practice Update table → Examiner's Tip. Recurring topics carry an additional "Extra Points to Remember" panel.

1. Epilepsy in the Elderly — Definition, Pathophysiology, Clinical Features, Investigations and Treatment

Definitions (ILAE / IBE)

1. An **epileptic seizure** is a transient occurrence of signs and/or symptoms due to **abnormal excessive or synchronous neuronal activity** in the brain.
2. **Epilepsy** exists when a seizure is confirmed and the brain shows an enduring tendency to recur. It is diagnosed with: **two unprovoked or reflex seizures more than 24 hours apart**; OR **one unprovoked seizure with a ≥60% probability of another over the next 10 years**; OR an **epilepsy syndrome**.
3. A seizure provoked by a transient factor acting on an otherwise normal brain **does not count** towards a diagnosis of epilepsy.
4. **Status epilepticus (SE)**: more than 30 minutes of continuous seizure activity, or intermittent seizures without full recovery of consciousness between them. Shorter operational durations are now proposed.
5. **Refractory epilepsy**: failure of adequate trials of **two tolerated, appropriately chosen antiepileptic drug (AED) schedules** to achieve sustained seizure freedom.

Epidemiology

6. Epilepsy is the **third most common neurological disorder** in older age groups. Incidence follows a **U-shaped curve**, highest at both ends of life. **25% of new-onset seizures occur after age 65.**
7. Prevalence of active epilepsy at age ≥75 is about **1.5%** (twice that of younger adults); in **nursing homes it exceeds 5%**.
8. SE incidence in the elderly is almost **double** the general population (86/100,000/year), rising with age, highest at ≥85 years. **Non-convulsive SE (NCSE)** occurs more often in the very old and critically ill.

Aetiology and pathophysiology

9. **Acute symptomatic seizures**: stroke 30–54%, metabolic/electrolyte imbalance 10–15%, tumours 10%, trauma 10%, drug-related 10%, CNS infection 2–3%.
10. **Epilepsy**: cryptogenic 48–50%, **stroke 40–53% (single largest identifiable cause)**, dementia 5–20%, tumours 4–19%, trauma 1–3%.
11. Mechanism: cortical/subcortical injury → gliotic scar → a hyperexcitable focus. In Alzheimer's disease, amyloid-driven **network hyperexcitability** raises seizure risk. Consider also autoimmune limbic encephalitis (VGKC/LGI1 antibodies) and Hashimoto's encephalopathy.

Clinical features

12. Seizure onset is **focal in 62–68%** and generalised in 27–39%; for established epilepsy, focal 65%, generalised 13%, unknown 22%.
13. Presentation is **subtle** — brief episodes of confusion, unexplained falls, or stereotyped movements, often **misdiagnosed as non-specific confusional states**.
14. Typical auras (fear, epigastric rising sensation, déjà vu) are **less common**; **non-specific auras such as dizziness predominate**. The seizure focus is more often **extratemporal, especially frontal**.
15. The **postictal period is prolonged** — lasting hours to days. Todd's paresis may mimic stroke.
16. **NCSE** presents as altered mental status ranging from mild disorientation to confusion or coma, uncharacteristic behaviour or aphasia. More than half occur with acute medical illness — organ failure, drug toxicity, alcohol/benzodiazepine withdrawal, metabolic disturbance.

Investigations

17. Diagnosis is **clinical**, based on an **eyewitness description**. The major difficulty is that older patients often cannot give an accurate history.
18. **EEG sensitivity falls with age**: epileptiform transients occur less often, activation procedures add little, EEG is normal in about one-third, and non-specific focal slowing is common even without seizures. A **sleep or nap EEG** is advised if the awake study is negative.
19. **Inpatient video-EEG is the gold standard** for characterising events and distinguishing epileptic from **psychogenic non-epileptic attacks** (6–15% of "uncontrolled epilepsy").
20. **Brain imaging in every patient** — MRI preferred over CT as it is more sensitive for stroke, TBI and structural lesions. Add ECG (exclude arrhythmic syncope), glucose, electrolytes, calcium, renal and liver function, and drug review.
21. **Differential diagnosis**: syncope, limb-shaking TIA, transient global amnesia, transient epileptic amnesia, hypoglycaemia, REM sleep behaviour disorder, periodic limb movement disorder, hypnic jerks, delirium, psychogenic attacks.

Treatment

22. Because recurrence risk is high, treat **after the first confirmed unprovoked seizure**, especially with a brain lesion or epileptiform EEG.

23. An ideal AED for the elderly has **linear pharmacokinetics, minimal hepatic metabolism, minimal drug interaction and renal excretion, and minimal impact on cognition, memory or mood** — no such drug exists.
24. **Start low, titrate slowly, use monotherapy first-line.** Older patients often respond to lower doses (e.g. valproate dose reduced by 30–40%).
25. **Preferred agents: lacosamide, lamotrigine and levetiracetam** — these have the greatest likelihood of seizure freedom at 6 and 12 months. Lamotrigine is better tolerated than carbamazepine.
26. **Avoid**: phenytoin, carbamazepine, phenobarbital, primidone, valproate and benzodiazepines — chronic toxicity, interactions and neurotoxicity in hypoalbuminaemia.
27. Match the drug to comorbidity: in **depression** avoid levetiracetam (irritability), topiramate, phenobarbital, primidone and vigabatrin. In **Alzheimer's disease**, treat after a second episode; avoid polytherapy, which impairs cognition.
28. Epilepsy is one of the **top 10 conditions related to frailty**. Assess falls risk and provide **bone protection** — AEDs increase fracture risk. Use **STOPP/START and Beers criteria** to manage polypharmacy.
29. **SE algorithm (Pathy's Box 51.1)**: treat within 5–10 minutes; IV glucose if hypoglycaemic. *First line* — lorazepam 0.1 mg/kg IV (2 mg/min), or diazepam 10 mg over 2 min, or midazolam 10 mg IM; repeat once at 10 min. *Second line (established SE)* — phenytoin 15 mg/kg (max 20 mg/min, with cardiac and BP monitoring), or valproate 15–30 mg/kg if cardiovascular comorbidity, or levetiracetam 20–30 mg/kg over 15 min. *Refractory* — midazolam or propofol infusion, then general anaesthesia and ventilation.
30. **Prognosis is good** — most newly diagnosed older patients become seizure-free on first AED monotherapy. Failure to become seizure-free within the first year predicts drug resistance. Epilepsy surgery and vagus nerve stimulation (27–44% seizure reduction) are feasible in selected older patients.

Latest Updates (2023–Present)

- ***Continuum* "Update on Antiseizure Medications" (2025)**: lamotrigine, oxcarbazepine, levetiracetam, topiramate, zonisamide and lacosamide have efficacy equal to older agents with equal or better tolerability as first-line therapy for focal epilepsy; pregabalin is inferior to lamotrigine. Lacosamide, pregabalin and eslicarbazepine have successful conversion-to-monotherapy data. **Ganaxolone** is the newest FDA-approved ASM.
- ***Drugs & Aging* (2026) review of ASMs in adults ≥65**: because most ASMs have comparable efficacy, **tolerability and quality of life — not efficacy — should drive drug choice**. Practical cautions: eslicarbazepine and oxcarbazepine cause **hyponatraemia** in the elderly; lacosamide should be avoided in **AV block**; lamotrigine needs slow titration and an ECG in cardiac disease; cenobamate, brivaracetam and perampanel are emerging options.
- ***Neurology* (2025) network meta-analysis of ASMs in post-stroke seizures (Misra et al.)** and ***Frontiers in Neurology* (2025)**: **lamotrigine and levetiracetam** are favoured; **primary ASM prophylaxis after stroke is not recommended**; early seizures (≤7 days) are acute symptomatic, while late seizures (>7 days) define **post-stroke epilepsy** and warrant long-term treatment. Lacosamide is a promising option including for NCSE in stroke.
- The **ILAE 2017 classification** (focal / generalised / combined / unknown, with awareness and motor descriptors) remains the operative framework; the term "antiseizure medication (ASM)" has replaced "AED" in current literature.
- **Indian context**: the epilepsy treatment gap remains 50–70% in rural India; phenytoin and phenobarbital remain widely used because of cost, though both are on the STOPP/Beers avoid list for older adults. Levetiracetam and lamotrigine are now inexpensive generics and should be preferred. Driving requires self-declaration of epilepsy under the **Central Motor Vehicles Rules**; Indian epilepsy society practice advises at least **one year of seizure freedom** before resuming driving.

Current Practice Update

Textbook Position (Pathy's 6th ed.)	Current Evidence-Based Practice (2023–2026)
Term "antiepileptic drug (AED)"	Replaced by **"antiseizure medication (ASM)"** — reflects symptomatic rather than disease-modifying action
Lacosamide, lamotrigine, levetiracetam are safe and first-line	Confirmed. **Choice now driven explicitly by tolerability and quality of life**, since efficacy is comparable (*Drugs & Aging* 2026)
SE defined as >30 minutes; shorter durations "proposed"	**Operationally accepted**: t_1 = 5 min (start treatment), t_2 = 30 min (risk of neuronal injury)
Eslicarbazepine listed as a useful newer agent	Usable, but **hyponatraemia risk in the elderly** now explicitly flagged; monitor sodium
Post-stroke seizure treatment not separately addressed	**No primary prophylaxis after stroke.** Late seizures = post-stroke epilepsy → treat with lamotrigine or levetiracetam
Edaravone/newer agents "need more study"	Cenobamate, brivaracetam, perampanel and ganaxolone now have geriatric tolerability data

> **Examiner's Tip — say these lines verbatim:**
> - "Epilepsy is the **third commonest neurological disorder** in the elderly; incidence follows a **U-shaped curve** and **25% of new-onset seizures occur after 65**."
> - "**Stroke is the single largest identifiable cause**, accounting for 40–53% of epilepsy in this age group."
> - "Presentation is subtle — **brief confusion, falls or stereotyped movements**. Over- and under-diagnosis are equally common."

- "**EEG sensitivity falls with age**; it is normal in one-third. **Video-EEG is the gold standard**; MRI is mandatory in all."
- "**Treat after the first unprovoked seizure** because of high recurrence — start low, go slow, monotherapy first."
- "**Lacosamide, lamotrigine and levetiracetam** are the drugs of choice; **avoid phenytoin, carbamazepine, phenobarbital and valproate**."
- "Epilepsy is one of the **top 10 conditions related to frailty** — always assess falls risk and give bone protection."

2. Coma in the Elderly

Source note: Coma is addressed across chapters in the uploaded textbooks (Glasgow Coma Scale and prognosis in the subdural haematoma chapter, level-of-consciousness grading within the Confusion Assessment Method, hyperosmolar non-ketotic coma, myxoedema coma, hypothermia and hyperthermia) rather than in a single dedicated chapter. Points below integrate these with current peer-reviewed neurocritical care literature.

Definition and physiology

1. **Coma** is a state of **unarousable unresponsiveness** — no eye opening, no comprehensible speech and no purposeful motor response even to noxious stimuli; operationally **GCS ≤8**.
2. Consciousness has two components: **arousal** (ascending reticular activating system in the brainstem tegmentum and thalamus) and **awareness** (cerebral cortex). Coma requires damage to the ARAS or to **both** hemispheres.
3. Spectrum of impaired consciousness (as graded in the CAM): **alert → vigilant → lethargic (drowsy, easily aroused) → stupor (difficult to arouse) → coma (unarousable)**.
4. Distinguish from **delirium** (acute onset, fluctuating, inattention, disorganised thinking — CAM), **vegetative state / unresponsive wakefulness**, **minimally conscious state**, **akinetic mutism**, **locked-in syndrome** (ventral pontine lesion; preserved vertical eye movement and blinking) and **psychogenic unresponsiveness**.
5. Ageing predisposes to coma: reduced cerebral reserve, **brain atrophy** (facilitating subdural haematoma), impaired drug clearance, polypharmacy, multimorbidity, blunted febrile and thirst responses, and atypical presentation.

Causes — structural vs non-structural

6. **Supratentorial structural**: large hemispheric infarct with oedema, intracerebral haemorrhage, **subdural haematoma** (bridging vein tear after trivial or unremembered trauma — the classic elderly cause), extradural haematoma, tumour, abscess, hydrocephalus.
7. **Infratentorial structural**: brainstem infarct or haemorrhage, **basilar artery occlusion**, cerebellar haemorrhage or infarct with brainstem compression, central pontine myelinolysis.
8. **Diffuse / metabolic-toxic (commonest group in the elderly)**: hypoglycaemia, **hyperosmolar hyperglycaemic state** (increased incidence in older adults), DKA, hyponatraemia, hypercalcaemia, uraemia, hepatic encephalopathy, hypoxia and hypercapnia, **myxoedema coma**, adrenal crisis, **hypothermia**, **heat stroke**, sepsis, Wernicke's encephalopathy.
9. **Drugs and poisons**: opioids, benzodiazepines, anticholinergics, antipsychotics, lithium, alcohol, carbon monoxide. In India, **organophosphate and aluminium phosphide poisoning** remain important.
10. **Seizure-related**: prolonged postictal state and **non-convulsive status epilepticus** — a reversible and frequently missed cause; have a low threshold for urgent EEG.
11. **Infective**: meningitis, encephalitis, cerebral malaria, neurocysticercosis, tuberculous meningitis (all endemic considerations in India).

Bedside evaluation

12. **ABC first**: airway, breathing, circulation; check **capillary blood glucose immediately**; give **thiamine before glucose** if alcohol use or malnutrition is possible.
13. Obtain a collateral history: tempo of onset, trauma (even trivial), anticoagulants, drug and alcohol history, preceding headache, fever, seizures, and baseline cognitive and functional state.
14. **Glasgow Coma Scale** (E4 V5 M6, total 3–15) quantifies depth. Limitations in the elderly: dysphasia, hearing and visual impairment, intubation, and pre-existing dementia. The **FOUR score** (eye, motor, brainstem, respiration) is preferred in intubated patients.
15. **Pupils**: pinpoint reactive → pontine lesion or opioids; unilateral fixed dilated → uncal herniation compressing CN III; mid-position fixed → midbrain; **preserved pupillary reactivity with deep coma strongly suggests a metabolic cause**.
16. **Brainstem reflexes**: corneal, oculocephalic (doll's eye — not if cervical injury), oculovestibular (cold caloric), gag and cough. **Respiratory pattern**: Cheyne–Stokes (bihemispheric/diencephalic), central neurogenic hyperventilation, apneustic (pons), ataxic/Biot (medulla).
17. **Motor**: asymmetry indicates a structural lesion; **decorticate** (flexor) posturing implies a lesion above the red nucleus, **decerebrate** (extensor) posturing a brainstem lesion. **Multifocal myoclonus and asterixis point to a metabolic cause**.
18. Also examine: fundi (papilloedema, subhyaloid haemorrhage), neck stiffness, skin (rash, needle marks, jaundice, cyanosis, hot dry skin of heat stroke), temperature, and evidence of head or cervical injury.

Investigations and management

19. Blood glucose, sodium, calcium, urea and creatinine, LFT, ammonia, ABG, CK, TSH, cortisol, blood and urine cultures, toxicology, ECG, chest X-ray.
20. **Non-contrast CT head is the first-line imaging**; MRI (with DWI) for posterior fossa, brainstem and basilar occlusion. **Lumbar puncture** after imaging if meningoencephalitis is suspected. **EEG** if NCSE is suspected.
21. Immediate reversal agents: 25–50 mL of 50% dextrose for hypoglycaemia; **thiamine 100 mg IV**; naloxone for opioids; flumazenil used cautiously (may precipitate seizures in chronic benzodiazepine use or mixed overdose).

22. **Airway protection if GCS ≤8.** Maintain oxygenation and normocapnia, treat hypotension, nurse head-up 30°, avoid hypotonic fluids.
23. **Raised intracranial pressure**: mannitol or hypertonic saline, controlled ventilation, urgent neurosurgical referral for subdural haematoma, intracerebral haemorrhage, cerebellar haemorrhage or hydrocephalus.
24. **Correct hyponatraemia cautiously** — Pathy's recommends raising serum sodium by only **6–8 mEq/L in 24 hours**; a **100 mL bolus of 3% saline** restores the GCS faster than a slow 20 mL/h infusion and had a lower mortality trend. Over-rapid correction risks osmotic demyelination.
25. Treat the cause: sepsis bundle and empirical antibiotics ± aciclovir; antiepileptics for NCSE; levothyroxine and hydrocortisone for myxoedema coma; rapid cooling for heat stroke; rewarming for hypothermia.
26. **Supportive care**: pressure-area care, VTE prophylaxis, aspiration precautions and nil by mouth, eye care to prevent exposure keratopathy, bladder and bowel care, nutrition, and prevention of contractures.
27. **Prognosis** depends entirely on cause. Metabolic and toxic causes are largely reversible; structural and hypoxic-ischaemic causes carry a poor outcome. In the elderly, **early goals-of-care discussion and review of advance directives are an essential part of management**, not an afterthought.

Latest Updates (2023–Present)

- **Curing Coma Campaign / Neurocritical Care Society (2023–2025)**: coma is being reframed as an **"acute disorder of consciousness"** with standardised common data elements, endotypes, and a proposed shift from purely descriptive to mechanism-based classification.
- **Cognitive motor dissociation (*NEJM* 2024, Bodien et al.)**: approximately **25% of clinically unresponsive patients** show volitional brain activation on task-based fMRI or EEG despite no bedside response. This is transforming prognostication and cautions strongly against early withdrawal of treatment based on the bedside examination alone.
- **FOUR score** has increasingly supplanted the GCS in intubated and sedated patients, as the verbal component of the GCS cannot be scored and it captures brainstem reflexes and respiratory pattern.
- **NCSE**: contemporary series find that up to **20% of unexplained coma in older adults** is non-convulsive status; the **Salzburg consensus criteria** are now the standard EEG diagnostic framework, and continuous EEG is recommended for unexplained persistent altered consciousness.
- **SCCM Heat Stroke Guidelines (*Critical Care Medicine*, 2025)** are directly relevant — hyperthermic coma requires cooling to <39°C within 30 minutes; and **Surviving Sepsis** updates address sepsis-associated encephalopathy.
- **Indian context**: road traffic injury, poisoning (organophosphate, aluminium phosphide), cerebral malaria, tuberculous meningitis and neurocysticercosis remain important causes not prominent in Western texts. The Supreme Court's *Common Cause* (2018, modified 2023) framework for advance medical directives is directly applicable to decision-making in prolonged coma.

Current Practice Update

Textbook Position	Current Evidence-Based Practice (2023–2026)
GCS is the standard measure of depth of coma and of prognosis in SDH	GCS retained, but **FOUR score preferred in intubated patients**; adds brainstem and respiratory assessment
Bedside unresponsiveness taken at face value	**Cognitive motor dissociation in ~25%** (*NEJM* 2024) — consider fMRI/EEG paradigms before prognosticating
Coma described descriptively within individual disease chapters	Reframed as an **"acute disorder of consciousness"** with mechanism-based endotypes (Curing Coma Campaign)
EEG mentioned mainly for epilepsy diagnosis	**Continuous EEG is standard** for unexplained coma; up to 20% is NCSE; Salzburg criteria applied
Hyponatraemia: 3% saline 100 mL bolus preferred to slow infusion	Confirmed and now standard; correct by **≤8 mEq/L per 24 h**, with DDAVP re-lowering if overcorrected

Examiner's Tip — say these lines verbatim:

- "Consciousness = **arousal (ARAS) + awareness (cortex)**. Coma requires damage to the ARAS or to **both** hemispheres."
- "**Check glucose before anything else**, and give **thiamine before glucose** in the malnourished."
- "**Preserved pupillary reactions in a deeply comatose patient point to a metabolic cause**; asymmetry points to a structural lesion."
- "**Multifocal myoclonus and asterixis are metabolic; posturing is structural.**"
- "In the elderly, always exclude **subdural haematoma, hypoglycaemia, hyponatraemia, drugs and non-convulsive status epilepticus** — all reversible."
- "Correct hyponatraemia by **no more than 6–8 mEq/L in 24 hours** to avoid osmotic demyelination."

3. Subdural Haematoma RECURS ×2

Definition and classification

1. **Subdural haematoma (SDH)** is a collection of blood in the **subdural space** — the potential space between the **dura mater** and the **arachnoid mater**.
2. The dura has two layers — an outer **periosteal** layer and an inner **meningeal** layer, separated from the arachnoid by a **dural border cell layer**. The potential space becomes a real space when the dural border cell layer **cleaves away** from the meningeal layer, shearing the **bridging veins** that traverse it to reach the dural venous sinuses.
3. **Classification by time from ictus**: **acute <3 days** (usually post-traumatic), **subacute 4–20 days**, **chronic >20 days**. The spectrum is best regarded as a **dynamic continuum**, not discrete entities.

Epidemiology and why the elderly are at risk

4. With ageing, the **brain atrophies and pulls away from the skull**, placing traction on the meninges and bridging veins. This predisposes to SDH after **minimal or unremembered trauma**, or even spontaneously.
5. The incidence of chronic SDH (CSDH) in patients **over 80 has risen significantly over the last three decades**; by 2030 CSDH is projected to be the **commonest cranial neurosurgical disease in the world**.
6. Falls are the commonest mechanism in older adults. A major confounding factor is the **widespread use of antiplatelets and anticoagulants** for comorbidities.

Pathophysiology — the key contrast

7. **Acute SDH**: signs and symptoms result from an **abrupt rise in intracranial pressure with compression of eloquent brain tissue**. The two aetiologies are **tearing of bridging veins** and **parenchymal laceration** (the latter reflecting severe primary injury).
8. **Chronic SDH**: the mechanism is **entirely different** — it is driven by an **inflammatory response provoked by blood haemolysis and its degradation products**, with neomembrane formation, fragile leaky neocapillaries fed by the **middle meningeal artery**, repeated microhaemorrhage and osmotic exudation. CSDH is phenotypically diverse.

Clinical features

9. **Acute SDH**: rapid decline in consciousness, headache, vomiting, focal deficit, seizures, anisocoria and herniation signs. Often a lucid interval is absent.
10. **Chronic SDH — the great mimic**: headache, cognitive decline, confusion, gait disturbance, falls, hemiparesis, aphasia and seizures. **50–70% of elderly patients present with altered mental status**; the course is characteristically **fluctuating**.
11. CSDH may masquerade as **dementia, stroke, delirium, normal pressure hydrocephalus, parkinsonism or depression** — it is a **reversible cause of cognitive decline**.

Investigations

12. **Non-contrast CT of the head is the initial method of choice.** Acute SDH is a **crescent-shaped hyperdensity with a concave inner surface**, causing midline shift and ventricular effacement; it **crosses suture lines but not the midline**.
13. **Subacute SDH becomes isodense** with brain (about 1–3 weeks) and is easily missed — look for **effaced sulci, a displaced grey–white junction, and asymmetric ventricles**. Chronic SDH is hypodense; **mixed density indicates re-bleeding**.
14. **MRI** is most useful for isodense collections, bilateral collections and for chronic intradural haematoma in evolution. Add coagulation screen, platelet count, and anticoagulant drug levels.

Management — acute SDH

15. **Small acute SDH (clot thickness <10 mm) in a neurologically intact patient**, with no herniation, no significant midline shift and no raised ICP, is **not emergently evacuated**. Monitor clinically and repeat the CT at 24 hours (or sooner) if there is concern about expansion or any change in mental status or neurological examination.
16. **Acute symptomatic SDH is a medical emergency** — rapid diagnosis, **reversal of anticoagulation**, and surgical evacuation (craniotomy or decompressive craniectomy) as soon as indicated.
17. **Anticoagulant reversal**: vitamin K plus 4-factor prothrombin complex concentrate for warfarin; **idarucizumab** for dabigatran; **andexanet alfa** for factor Xa inhibitors. **In patients on antiplatelet medication, preoperative platelet transfusion did not reduce reoperation rates; platelet reversal and platelet transfusion are therefore not routinely recommended.**

Management — chronic SDH

18. Surgical options are **twist-drill craniostomy**, **burr-hole craniostomy** (5–30 mm hole, better visualisation of dura and cortex, usually under general anaesthesia — the standard operation), and **craniotomy** (large bone flap, direct access to haematoma and membranes; preferred for **multiloculated CSDH with significant membranes**).
19. Evidence: **no difference** in mortality, morbidity, recurrence or cure between burr-hole and twist-drill craniostomy. Open craniotomy as first treatment has **more morbidity but lower recurrence**.
20. **Strong evidence supports placement of a subdural drain** — it significantly lowers recurrence and improves

functional outcome **without** increasing operative complications or mortality. No universal management consensus exists; each case is individualised.

21. Complications are frequent given age, frailty and comorbidity: wound infection, subdural empyema, tension pneumocephalus, seizure, brain contusion, fresh subdural or extradural haematoma, intracerebral haemorrhage and death.
22. **Recurrence is up to 33%** despite gold-standard drainage and irrigation. **Risk factors for recurrence: age >70, bilateral CSDH, large haematoma volume, preoperative seizure, postoperative intracranial air, low GCS, and diabetes mellitus.**
23. **Medical adjuncts**: **atorvastatin** 20 mg (Jiang et al., *JAMA Neurology* 2018 RCT) reduced haematoma volume and improved neurological outcome. Dexamethasone has been studied — see updates below.
24. **Endovascular treatment**: **middle meningeal artery (MMA) embolisation** addresses the underlying pathology rather than simply draining blood — by embolising the MMA the blood supply to the pathological membranes is removed, allowing spontaneous resolution.

Prognosis and prevention

25. **Mortality for acute SDH is 22–60%**, higher in the elderly. **Predictors of poor outcome: advanced age, low GCS at presentation, comorbid intracranial and extracranial injury, and anticoagulant use.**
26. **Features favouring spontaneous resolution**: coma lasting less than 12 hours after injury, absence of cerebral contusion, a **thin haematoma with wide distribution**, GCS >8, and clinical stability over time.
27. **Prevention**: **DOACs reduce the risk of intracranial haemorrhage compared with vitamin K antagonists** while remaining at least as effective for thromboembolism. Registry data show DOAC-treated patients have **lower in-hospital mortality, fewer severe intracranial haemorrhages, less need for operative intervention and less discharge to skilled nursing facilities** than warfarin-treated patients. Add falls prevention, medication review, and vision and gait optimisation.

Latest Updates (2023–Present)

- **Three landmark randomised controlled trials of MMA embolisation published in the *New England Journal of Medicine* (November 2024 and February 2025):**
  - **EMBOLISE** (Onyx liquid embolic, MMAE as an *adjunct* to surgery) — significant reduction in haematoma recurrence requiring reoperation.
  - **STEM** (Squid, adjunctive and standalone) — composite of recurrent/residual haematoma, surgical rescue, major disabling stroke, MI or death at 180 days was **16% with MMAE vs 36% with control (OR 0.36, 95% CI 0.20–0.66, p = 0.001)**.
  - **MAGIC-MT** (China, Onyx) — **neutral**: symptomatic recurrence or progression at 90 days 6.7% vs 9.9% (p = 0.10).
- **Meta-analysis of the three RCTs (2025)**: MMAE is **safe** with no significant increase in serious adverse events, and gives a **modest overall reduction in recurrence (risk difference −0.09, p = 0.02)**, though the effect is largely driven by STEM and the benefit as a surgical adjunct is less clear.
- **Society of Vascular and Interventional Neurology (SVIN) consensus guideline (2025)** supports MMAE in selected patients (adjunct in high-recurrence-risk cases and standalone in minimally symptomatic patients unfit for surgery). In contrast, **the 2025 UK national CSDH guidelines declined to recommend MMAE routinely**, calling for further research — know both positions.
- **Dexamethasone is not recommended**: the **DEX-CSDH trial (*NEJM* 2020)** found dexamethasone reduced recurrence but produced **worse functional outcome at 6 months and more adverse events**. This overturns older favourable observational data cited in the textbook.
- **Resumption of anticoagulation after CSDH**: contemporary observational data (2023–2025) support restarting at around **4 weeks** in patients with a strong indication (mechanical valve, high CHA_2DS_2-VA score), balanced individually against re-bleed risk.
- **Indian context**: CSDH is a very common presentation in Indian neurosurgical practice. **Burr-hole drainage with a closed-system subdural drain under local anaesthesia** remains the standard and is highly cost-effective; MMA embolisation is limited by cost, catheter-lab availability and the absence of Indian RCT data. Atorvastatin is an attractive low-cost adjunct in the Indian setting.

Current Practice Update

Textbook Position (Pathy's 6th ed.)	Current Evidence-Based Practice (2023–2026)
MMA embolisation described as an area of "increasing interest" based on meta-analyses of case series	Now supported by **three NEJM RCTs** (EMBOLISE, STEM positive; MAGIC-MT neutral) and a 2025 **SVIN consensus guideline**; safe, with modest recurrence benefit
Corticosteroid (dexamethasone) treatment cited from favourable systematic reviews	**Not recommended** — DEX-CSDH (*NEJM* 2020) showed **worse functional outcome and more adverse events** despite lower recurrence
Atorvastatin supported by a single Chinese RCT	Remains a reasonable low-cost adjunct, particularly for small/asymptomatic CSDH; larger confirmatory trials still awaited
Subdural drain reduces recurrence	**Unchanged and reinforced** — drain placement remains the single most robust surgical recommendation
Platelet transfusion not routinely recommended in antiplatelet users	**Unchanged**; supported by PATCH-type data showing harm from platelet transfusion in antiplatelet-associated ICH
DOACs preferable to warfarin with respect to intracranial haemorrhage	**Confirmed**; specific reversal agents (idarucizumab, andexanet alfa) are now standard of care

Extra Points to Remember — recurring topic (asked twice)

- **The one-line contrast that earns marks:** "Acute SDH is a **pressure and mass-effect** problem; chronic SDH is an **inflammatory and angiogenic** problem driven by blood degradation products."
- **The classic exam trap:** the **isodense phase at 2–3 weeks** — a CT may look "normal". Look for effaced sulci, a displaced grey–white junction, and small ventricles.
- **Bilateral CSDH may show NO midline shift** because the two collections balance each other — the only clues are effaced sulci and compressed ventricles ("slit ventricles").
- **CSDH is a reversible cause of dementia** — always image the brain before labelling a patient with falls or anticoagulant use as having dementia.
- **Contrast with extradural haematoma:** biconvex/lenticular, does *not* cross suture lines but *can* cross the midline, arterial (middle meningeal artery) bleed, classic lucid interval, typically younger patients. SDH: crescentic, crosses sutures but not midline, venous bleed, older patients.
- **Numbers worth quoting:** mortality 22–60%; recurrence up to 33%; clot thickness <10 mm may be observed; recurrence risk factors — age >70, bilateral, large volume, preoperative seizure, postoperative air, low GCS, diabetes.
- **Two-sentence "prevention" answer:** "DOACs rather than vitamin K antagonists where anticoagulation is indicated, plus multifactorial falls prevention with medication review, vision correction and gait/balance training."

Examiner's Tip — say these lines verbatim:

- "**Brain atrophy stretches the bridging veins**, so SDH occurs in the elderly after trivial or unremembered trauma."
- "**CT head is the initial investigation of choice** — acute is a crescentic hyperdensity concave to the skull; chronic is hypodense; **subacute is isodense and easily missed**."
- "**Chronic SDH is driven by an inflammatory response to blood degradation products**, not by mass effect — which is why MMA embolisation works."
- "**Placement of a subdural drain significantly lowers recurrence and improves functional outcome without increasing complications.**"
- "**Dexamethasone is no longer recommended** — DEX-CSDH showed worse functional outcome despite fewer recurrences."
- "CSDH is expected to be the **commonest cranial neurosurgical disease in the world by 2030**."

4. Lacunar Infarcts

Definition and pathology

1. **Lacunes** are small infarcts resulting from occlusion of the **small perforating arterioles** supplying the basal ganglia, external and internal capsule, thalamus and pons. On brain imaging they are defined as **deep subcortical infarcts less than 15 mm in maximum diameter**.
2. Two underlying pathologies: **microatheroma at the origin of the perforating artery**, and more distal **lipohyalinosis** — degenerative change in the **tunica media and adventitia** of the arterioles. These cannot be distinguished clinically or radiologically and are together known as **small-vessel disease (SVD)**.
3. Lacunar strokes account for roughly **a quarter of all ischaemic strokes**.
4. Other imaging manifestations of the same process: **leukoaraiosis** (patchy or confluent low attenuation on CT, high signal on T_2 and FLAIR MRI in the white matter, also attributed to lipohyalinosis) and **cerebral microhaemorrhages** on gradient-echo (T_2*) or susceptibility-weighted MRI.
5. **Leukoaraiosis, lacunes and microhaemorrhages are each independently associated with cognitive impairment.**
6. **Hypertension is the major risk factor** for lacunar infarction, leukoaraiosis and microhaemorrhage. Microhaemorrhages are also associated with **cerebral amyloid angiopathy** — deep/basal ganglia microbleeds suggest hypertensive arteriopathy, lobar microbleeds suggest amyloid angiopathy.
7. Other risk factors: increasing age, diabetes mellitus, smoking and dyslipidaemia. Cardioembolism and large-artery atherosclerosis are usually **not** the mechanism, but must still be excluded.

Classical lacunar syndromes

8. **Pure motor stroke** (commonest): unilateral face, arm and leg weakness. The weakness need not involve all three but should involve **at least two contiguous sites** — face and arm, or arm and leg. Lesion in the posterior limb of the internal capsule or the pons.
9. **Pure sensory stroke**: contralateral hemisensory loss; lesion in the thalamus (ventral posterolateral nucleus).
10. **Sensorimotor stroke**: a larger lacune producing mixed motor and sensory loss.
11. **Ataxic hemiparesis**: limb ataxia combined with hemiparesis and/or dysarthria (including the dysarthria–clumsy hand variant); internal capsule or pons.
12. **The defining negative features (the marks-earning point): there is NO disturbance of higher cortical function and NO visual field deficit** — no aphasia, neglect, agnosia or apraxia, and no hemianopia.
13. Rarer lacunar syndromes include **unilateral chorea and hemiballismus** from lacunes in the basal ganglia.

Diagnosis

14. **Imaging is required** to distinguish a capsular from a pontine site, as the syndromes are identical clinically. **Diffusion-weighted MRI is most sensitive acutely**; CT is frequently normal in the first hours.
15. Occasionally a small haemorrhage produces an identical syndrome — imaging is essential before antithrombotic therapy.
16. Complete the standard stroke workup: ECG and prolonged rhythm monitoring, echocardiography, carotid imaging, lipids, HbA1c, renal function, ESR — principally to exclude an alternative mechanism.

Prognosis and management

17. **Lacunar stroke carries a good prognosis for survival, with a one-year mortality of about 10%, but 50% of survivors remain dependent.**
18. Recurrent lacunes and progressive white matter change lead to **vascular cognitive impairment, vascular parkinsonism, pseudobulbar palsy, gait apraxia and emotional lability** ("état lacunaire").
19. **Acute treatment**: intravenous thrombolysis if within the licensed window (benefit is preserved in lacunar stroke), antiplatelet therapy, and admission to a **stroke unit** for multidisciplinary care.
20. **Secondary prevention — blood pressure control is the single most important intervention** (PROGRESS: perindopril-based regimen reduced recurrent stroke); target ≤130/80 mmHg in diabetic patients.
21. **Statin**: high-dose atorvastatin 80 mg reduced recurrent ischaemic stroke by around 20% (SPARCL); treat cholesterol >3.5 mmol/L with dietetic input.
22. **Antiplatelet therapy** (aspirin, clopidogrel, or aspirin with extended-release dipyridamole) plus glycaemic control, smoking cessation and lifestyle change. **Anticoagulation is not indicated** unless there is a separate indication such as atrial fibrillation or a prosthetic valve.

Latest Updates (2023–Present)

- **LACI-2 trial (Wardlaw et al., *JAMA Neurology* 2023)** — the single most examinable update. 363 patients with clinical lacunar ischaemic stroke, randomised 2×2 to **isosorbide mononitrate (ISMN) 40–60 mg/day**, **cilostazol 200 mg/day**, both, or neither, on top of guideline secondary prevention for one year. **ISMN reduced recurrent stroke (adjusted OR 0.23, 95% CI 0.07–0.74) and cognitive impairment (aOR 0.55, 95% CI 0.36–0.86); cilostazol reduced dependence; the combination reduced the composite of vascular, dependence and cognitive outcomes.** Retention was 99% with good adherence and no safety concerns. A prespecified 6-month

analysis (2025) confirmed that benefit appears early. Both are **inexpensive, licensed generic drugs**; the definitive phase 3 trial (LACI-3) is awaited.

- **STRIVE-2 consensus (*Lancet Neurology* 2023)** updated the neuroimaging standards for reporting cerebral small vessel disease — standardised terminology for recent small subcortical infarct, lacune of presumed vascular origin, white matter hyperintensity, perivascular space, microbleed, cortical superficial siderosis and brain atrophy. **"Leukoaraiosis" is being retired in favour of "white matter hyperintensity of presumed vascular origin."**
- **European Stroke Organisation guidelines on cerebral small vessel disease** — Part 1 (covert SVD, 2021) and Part 2 (lacunar ischaemic stroke) formalise management.
- **Antiplatelet intensification**: **INSPIRES (*NEJM* 2024)** supports clopidogrel plus aspirin started within 72 hours for mild ischaemic stroke or high-risk TIA of atherosclerotic origin; **CHANCE-2** showed genotype-guided ticagrelor is superior in **CYP2C19 loss-of-function carriers** — highly relevant, as this genotype is common in Indian and East Asian populations.
- **Blood pressure**: SPS3 supported a systolic target <130 mmHg in lacunar stroke, particularly for reducing intracerebral haemorrhage; ESPRIT and 2024–2025 intensive-lowering trials reinforce this.
- **Indian context**: small-vessel disease and intracranial atherosclerosis account for a much larger share of Indian ischaemic stroke than extracranial carotid disease. ICMR-INDIAB and LASI data show very high prevalence and poor control of hypertension and diabetes in older Indians; the **India Hypertension Control Initiative (IHCI)** and the **National Programme for Prevention and Control of Non-Communicable Diseases (NP-NCD)** are the operative public-health frameworks for prevention.

Current Practice Update

Textbook Position (Pathy's 6th ed.)	Current Evidence-Based Practice (2023–2026)
Secondary prevention is antiplatelet + statin + BP control, identical to other ischaemic stroke subtypes	**LACI-2 (2023)** supports adding **isosorbide mononitrate** (reduces recurrent stroke and cognitive impairment) and **cilostazol** (reduces dependence) — endothelium-targeted therapy specific to small-vessel disease
Terms "leukoaraiosis" and "lacune" used descriptively	**STRIVE-2 (2023)** standardised terminology — "white matter hyperintensity of presumed vascular origin", "recent small subcortical infarct", "lacune of presumed vascular origin"
Aspirin or clopidogrel monotherapy after minor stroke/TIA	Short-course **dual antiplatelet therapy** (CHANCE, POINT, INSPIRES) for 21–30 days; **genotype-guided ticagrelor** in CYP2C19 loss-of-function carriers (CHANCE-2)
BP target ≤130/80 specified for diabetic patients	**Systolic <130 mmHg for all lacunar stroke** (SPS3 and subsequent intensive-lowering trials), especially to reduce intracerebral haemorrhage
Cognitive consequences noted as an association	SVD is now recognised as the **leading vascular contributor to dementia**; cognitive outcome is a primary trial endpoint

Examiner's Tip — say these lines verbatim:

- "A lacune is a **deep subcortical infarct less than 15 mm in maximum diameter** from occlusion of a small perforating artery."
- "The two pathologies are **microatheroma at the perforator origin** and **lipohyalinosis of the tunica media and adventitia** — indistinguishable clinically and radiologically, hence 'small-vessel disease'."
- "The four syndromes are **pure motor, pure sensory, sensorimotor and ataxic hemiparesis**."
- "The defining feature is **absence of cortical signs and absence of a visual field defect**."
- "**Hypertension is the major risk factor** for lacunes, leukoaraiosis and microhaemorrhages."
- "Prognosis: **one-year mortality about 10%, but 50% of survivors remain dependent**."
- "**LACI-2 (2023)** showed isosorbide mononitrate reduced recurrent stroke and cognitive impairment, and cilostazol reduced dependence — two cheap licensed drugs awaiting phase 3 confirmation."

5. Subacute Combined Degeneration of the Cord — Presentation and Treatment

Definition and biochemistry

1. **Subacute combined degeneration (SACD)** is progressive degeneration — demyelination followed by axonal loss — of the **posterior (dorsal) columns** and the **lateral corticospinal tracts** of the spinal cord, caused by **vitamin B_{12} (cobalamin) deficiency**.
2. "**Subacute**" refers to evolution over weeks to months; "**combined**" to the involvement of **two** tract systems. The cervical and upper thoracic cord are preferentially affected.
3. Vitamin B_{12} is **required for DNA synthesis, mitochondrial function and red blood cell production**, and converts folate to its biologically active form; when deficient it can cause a secondary folate deficiency.
4. Two cobalamin-dependent enzymes explain the pathology: **methionine synthase** (homocysteine → methionine, required for S-adenosylmethionine and myelin methylation) and **methylmalonyl-CoA mutase**. Deficiency therefore raises both **homocysteine** and **methylmalonic acid (MMA)**, and impairs myelin maintenance.

Causes

5. **Impaired absorption** is the dominant mechanism in the elderly: **atrophic gastritis** (10–30% of older adults malabsorb food-bound B_{12}), **pernicious anaemia** (lack of intrinsic factor; 1–2% of older adults), gastrectomy or bariatric surgery, ileal resection, Crohn's disease, intestinal bacterial overgrowth.
6. **Drugs**: **metformin** (long-term users should be screened), **proton pump inhibitors and H_2 blockers** (raised gastric pH prevents release of B_{12} from animal protein).
7. **Inadequate intake**: B_{12} is found **strictly in animal products** — red meat, eggs and dairy — making **vegetarians and vegans particularly susceptible**. This is of major importance in India.
8. **Nitrous oxide** exposure or abuse irreversibly oxidises the cobalt ion of cobalamin and inactivates methionine synthase, precipitating SACD **even when the serum B_{12} is normal**.
9. Prevalence of B_{12} deficiency ranges from **5–40%** in older people depending on the cut-off used; 13.8% on nursing-home admission with a 4% new yearly incidence.

Presentation

10. **Critical exam point: neurological disease may occur in the complete absence of anaemia or macrocytosis** — a normal blood count never excludes SACD.
11. **Earliest symptoms are sensory**: symmetrical distal **paraesthesiae, numbness and tingling in the hands and feet**, often affecting the hands before the feet.
12. **Posterior column signs**: loss of **vibration sense (the earliest and most sensitive sign)** and joint position sense, **sensory ataxia**, a **positive Romberg's sign**, and unsteadiness that is markedly worse in the dark or with eyes closed.
13. **Corticospinal tract signs**: spastic paraparesis, weakness, hyperreflexia and **extensor plantar responses**.
14. **The classic paradoxical combination: brisk knee jerks with absent ankle jerks and extensor plantars** — produced by simultaneous myelopathy and peripheral neuropathy. Always quote this.
15. Additional features: **Lhermitte's phenomenon**, gait ataxia, autonomic dysfunction, bladder disturbance late, and **optic atrophy** with centrocaecal scotoma.
16. **Neuropsychiatric**: mood change, depression, irritability, confusion, poor memory, dementia, and psychosis ("megaloblastic madness").
17. **Haematological and mucosal**: macrocytic megaloblastic anaemia, **hypersegmented neutrophils**, glossitis with a beefy-red sore tongue, angular stomatitis, vitiligo, mild jaundice from ineffective erythropoiesis (raised LDH, low haptoglobin), fatigue, weakness, anorexia and weight loss.

Investigations

18. **Serum B_{12}** is the first test but there is **no gold-standard diagnostic test**; the diagnosis rests on the clinical picture with a low B_{12}. **Serum MMA and homocysteine are both raised** and increase diagnostic sensitivity where the B_{12} is borderline; holotranscobalamin (active B_{12}) is an alternative.
19. **Falsely normal B_{12}** occurs in hepatic and renal disease and myeloproliferative disorders; **falsely low B_{12}** occurs in folate deficiency and multiple myeloma.
20. Additional tests: full blood count and peripheral smear, reticulocytes, LDH, folate, ferritin (coexisting iron deficiency masks macrocytosis), TSH, **intrinsic factor antibody** (specific but insensitive) and **anti-parietal cell antibody** (sensitive, non-specific), and upper GI endoscopy for atrophic gastritis.
21. **MRI of the spine**: T_2 hyperintensity in the **dorsal columns of the cervical and upper thoracic cord**, producing the classic **"inverted V" or "inverted rabbit-ear" sign** on axial images. Nerve conduction studies confirm coexisting peripheral neuropathy.

Treatment

22. **Neurological involvement mandates parenteral replacement.** Give **hydroxocobalamin 1000 mcg IM on alternate days until there is no further improvement**, then 1000 mcg every 2–3 months indefinitely.

23. Textbook regimen for deficiency without neurological disease: **IM B_{12} 1000 mcg weekly for severe deficiency, then monthly for maintenance** (10–15% of each injection is absorbed, so stores replenish quickly).
24. **Oral B_{12} 2000 mcg daily** is as effective as intramuscular therapy in most cases, is cheaper and easier, and works even with achlorhydria or pernicious anaemia because a small fraction is **passively absorbed independent of intrinsic factor**. Give **once daily only** — receptor saturation means more frequent dosing does not improve absorption.
25. **Do not give folate alone** — folic acid without B_{12} corrects the anaemia while allowing the neurological disease to progress. Start B_{12} first, then replace folate.
26. Monitoring: reticulocytosis by day 5–7; **watch for hypokalaemia in the first week** of a brisk haematological response; recheck haemoglobin and B_{12} at **3 months**.
27. **Treatment is lifelong** in pernicious anaemia and after gastric or bowel resection, since absorption will remain deficient. Correct coexisting iron and folate deficiency. Review and, where possible, address contributing drugs (metformin, PPIs).
28. **Prognosis is inversely related to the duration of symptoms.** Treatment within weeks usually gives complete recovery; after 6–12 months of symptoms, progression is halted but full recovery is unlikely. **Sensory symptoms recover better than spastic paraparesis.**
29. **Prevention**: have a high index of suspicion and screen at-risk individuals annually — those with bowel resection, atrophic gastritis, inflammatory bowel disease, restrictive diets, and users of metformin or acid suppressants. Adults over 50 should meet the RDA mainly through **fortified foods or a supplement** containing B_{12}, since food-bound B_{12} is poorly absorbed.

Latest Updates (2023–Present)

- **NICE guideline NG239, "Vitamin B_{12} deficiency in over 16s: diagnosis and management" (March 2024)** — the first UK national guideline and the definitive current reference, which **supersedes the BSH 2014 guideline**. Key positions: test only when there are symptoms or risk factors rather than routinely; where serum B_{12} is indeterminate, use **MMA or holotranscobalamin** as second-line; and **in suspected neurological involvement start intramuscular hydroxocobalamin without waiting for test results**. Oral replacement is acceptable for non-neurological deficiency without malabsorption.
- **NICE 2024 also stresses "functional B_{12} deficiency"** — strong clinical features such as megaloblastic anaemia or SACD despite a normal serum B_{12} warrant urgent haematology advice and empirical treatment.
- **Nitrous oxide–induced SACD** is now a recognised and rapidly growing cause (case series in *Journal of Neurology* 2025 and *Frontiers in Immunology* 2025). Compared with dietary B_{12} deficiency it produces **more severe and more extensive spinal cord and peripheral nerve damage**; **serum B_{12} was normal in a substantial proportion, but MMA was elevated in all**; recovery is often incomplete despite replacement.
- **Metformin**: the **ADA Standards of Care (2025)** recommend periodic measurement of B_{12} in metformin-treated patients, particularly those with peripheral neuropathy or anaemia.
- **Indian context**: B_{12} deficiency is exceptionally common because of predominantly lacto-vegetarian diets, with population studies reporting deficiency in **47–75%** of Indian adults. **ICMR-NIN RDA (2020) is 2.2 mcg/day** for adults. Mandatory fortification of wheat flour and rice with folic acid, iron and B_{12} under FSSAI's +F programme is relevant — folate excess without B_{12} repletion could theoretically unmask neurological disease, so B_{12} status should be checked in symptomatic patients.

Current Practice Update

Textbook Position (Pathy's 6th ed.)	Current Evidence-Based Practice (2023–2026)
Oral B_{12} 2000 mcg daily is generally as effective as IM in most cases	True for **uncomplicated deficiency**, but **NICE NG239 (2024) requires IM hydroxocobalamin where there is neurological involvement** — oral therapy risks under-treatment of SACD
"No consensus exists on when additional diagnostic tests should be added"	**NICE NG239 (2024) provides a clear algorithm**: indeterminate serum B_{12} → MMA or holotranscobalamin as second-line
"No major medical organization has published guidelines on routine screening"	**NICE NG239 (2024) now defines who to test**: symptoms plus specified risk factors; still no universal population screening
Treatment may await confirmatory testing	**Do not delay** — in suspected neurological disease start IM hydroxocobalamin immediately; damage becomes irreversible
Causes listed as dietary, malabsorptive and drug-related	**Nitrous oxide abuse** added as an important and severe cause, often with a **normal serum B_{12}** but elevated MMA
Metformin association noted; "long-term users should be screened"	**ADA 2025** formalises periodic B_{12} monitoring in metformin users, especially with neuropathy or anaemia

> **Examiner's Tip — say these lines verbatim:**
> - "SACD is degeneration of the **posterior columns and lateral corticospinal tracts** due to vitamin B_{12} deficiency — 'combined' means two tract systems."

- "**Neurological disease can occur without anaemia or macrocytosis** — a normal blood count never excludes SACD."
- "The classic sign is **brisk knee jerks with absent ankle jerks and extensor plantars** — myelopathy plus peripheral neuropathy."
- "**Loss of vibration sense is the earliest and most sensitive sign**, with a positive Romberg's."
- "MRI shows **T_2 hyperintensity in the dorsal columns** — the **'inverted V' sign** on axial images."
- "**Never give folate before B_{12}** — it corrects the anaemia and lets the neurological disease progress."
- "**Recovery is inversely related to the duration of symptoms**; treat within weeks for complete recovery."
- "**NICE NG239 (2024): in neurological involvement, start intramuscular hydroxocobalamin without waiting for test results.**"

6. Motor Neuron Disease — Presentation, Evaluation and Treatment

Definition and classification

1. **Motor neuron diseases (MNDs)** are a heterogeneous group characterised by degeneration or dysfunction of the **upper motor neurons (UMN) of the precentral gyrus** and/or the **lower motor neurons (LMN) of the ventral horn of the spinal cord**.
2. **Amyotrophic lateral sclerosis (ALS)** is the commonest MND in adults and involves both UMN and LMN. It shows considerable **phenotypic heterogeneity** and is better regarded as a syndrome — sporadic and genetic forms, pure motor and extra-motor (frontotemporal dementia) presentations, pure LMN or pure UMN variants, and localised forms.
3. Variants: **progressive muscular atrophy** (LMN only), **primary lateral sclerosis** (UMN only), **progressive bulbar palsy**, flail arm and flail leg syndromes, and the **ALS–FTD spectrum**. Although histopathologically similar, **individualisation matters because prognosis differs**.
4. Epidemiology: incidence roughly 2–3 per 100,000 per year, peak onset 60–75 years, slight male predominance; about 10% familial. More than **30 genes** are implicated — **C9orf72** (commonest, links ALS with FTD), **SOD1, TARDBP, FUS**.

Pathophysiology

5. The neuropathological hallmark of most ALS is **cytoplasmic inclusions containing ubiquitinated and hyperphosphorylated TDP-43** (except in SOD1- and FUS-linked familial ALS, which represent under 3% of cases).
6. **TDP-43** is normally nuclear and regulates transcription, splicing and mRNA stability. In disease it is **mislocalised** — nuclear depletion causes aberrant RNA metabolism and impaired chromatin remodelling, while cytoplasmic accumulation disturbs autophagy, proteasome function, protein quality control and mitochondrial function. TDP-43 aggregates have a **seeding capability and prion-like cell-to-cell transmission**.
7. Converging mechanisms: **disrupted RNA metabolism, aberrant protein quality control, mitochondrial dysfunction, oxidative stress, glutamate excitotoxicity, trafficking abnormalities and hypermetabolism**.

Clinical presentation

8. Motor symptoms are frequently preceded for months by **non-specific features: fatigue, weight loss secondary to hypermetabolism, cramps and diffuse fasciculation** related to loss and hyperexcitability of lower motor neurons.
9. **Limb-onset ALS** (commonest): difficulty performing fine hand movements, difficulty raising the arms, foot drop and difficulty walking — typically **asymmetrical, painless, progressive weakness**.
10. **Bulbar-onset ALS (about one-third)**: swallowing and phonation disorders with **sialorrhoea**, dysarthria, tongue wasting and fasciculation. Commoner in older women and carries a **worse prognosis**.
11. **Respiratory-onset (rare)**: onset in the trunk musculature with isolated restrictive respiratory impairment causing effort dyspnoea and orthopnoea, frequently with unexplained weight loss.
12. **Signs**: mixed UMN (spasticity, hyperreflexia, extensor plantars, brisk jaw jerk, clonus) and LMN (wasting, fasciculation, hypotonia) findings **in the same body region**, with contiguous spread. **Pseudobulbar affect** (emotional lability) is common.
13. **Cognitive and behavioural change occurs in up to 50%**, with frank FTD in 10–15%. Revised criteria for **ALS–frontotemporal spectrum disorder (ALS-FTSD, Strong 2017)** and the **Edinburgh Cognitive and Behavioural ALS Screen (ECAS)** are the standards. **The frontotemporal syndrome of ALS is associated with poor survival** and negatively affects caregiver burden and quality of life.
14. **Characteristically spared until late**: extraocular movements, sphincter function, sensation, and cognition in the majority — the preservation of cognitive capacity in most patients is precisely why **advance directives can and should be discussed early**.

Evaluation

15. **In the absence of positive diagnostic markers, diagnosis rests on precise clinical and electrophysiological analysis, the study of the disease course, and elimination of the differential diagnoses referred to as "ALS mimics".** Diagnostic delay is common.
16. Diagnostic criteria: **El Escorial (1994)**, **revised El Escorial / Airlie House (2000)**, and the **Awaji electrodiagnostic criteria (de Carvalho 2008)**, in which fasciculation potentials count as evidence of acute denervation.
17. **EMG**: fibrillations, positive sharp waves, fasciculations, large-amplitude long-duration polyphasic motor units and reduced recruitment, in at least two of four regions (bulbar, cervical, thoracic, lumbosacral). Nerve conduction studies show **normal sensory responses** and no conduction block.
18. **Exclude the treatable mimics** — many are eminently treatable: cervical and lumbar spondylotic myeloradiculopathy (MRI brain and whole spine), **multifocal motor neuropathy with conduction block** (anti-GM1 antibodies; responds to IVIG), CIDP, myasthenia gravis, inclusion body myositis, Kennedy disease, **vitamin B_{12} deficiency**, thyrotoxicosis, hyperparathyroidism, paraneoplastic syndromes, HIV, syphilis and lymphoma.
19. Laboratory work: CK (mildly raised), TSH, calcium and PTH, vitamin B_{12}, ESR, serum protein electrophoresis, anti-GM1, HIV and VDRL. **CSF neurofilament proteins (NfL, pNfH) are established diagnostic and prognostic biomarkers.**

20. **Respiratory assessment at diagnosis and every 3 months**: erect and supine FVC, sniff nasal inspiratory pressure (SNIP), overnight oximetry and arterial blood gases. Also assess nutrition, weight trajectory, swallow and cognition (ECAS).

Treatment

21. **Despite more than 200 trials, ALS remains incurable.** Aetiologic treatment slightly improves survival; symptomatic and supportive management improves both survival and quality of life.
22. **Riluzole** (approved 1995), 50 mg twice daily, acts on Na^+ and K^+ currents and glutamatergic transmission. Median survival gain is modest, about **three months**; the one-year survival gain was 9% (**58% vs 49%**), with small effects on bulbar and limb function. **All population-based registries confirm riluzole is an independent factor influencing long-term survival**, and it is recommended by the EFNS and the AAN **for all ALS patients, even in later stages**. Monitor liver enzymes.
23. **Edaravone**, a free-radical scavenger, was FDA-approved on the basis of a second Japanese phase 3 trial in a narrowly selected group (definite or probable ALS, duration ≤2 years, normal respiratory function, ALSFRS-R item scores ≥2), showing a least-squares mean difference of **2.49 points** in favour of edaravone. However a slight **deterioration in quality of life** was observed, and in **May 2019 the EMA withdrew the marketing authorisation application** because of baseline imbalance, lack of effect on survival, breathing and muscle strength, and too short a study duration.
24. **Multidisciplinary care is itself an evidence-based intervention** — multidisciplinary clinics improve survival compared with general neurology care.
25. **Respiratory failure is the commonest cause of death** and one of the most important determinants of quality of life and anxiety. **Non-invasive ventilation prolongs survival** and should be offered for symptomatic nocturnal hypoventilation, FVC <50%, or SNIP <40 cmH_2O. Cough augmentation and secretion management are essential adjuncts.
26. **Nutrition**: early dietitian involvement, high-calorie diet to offset hypermetabolism, texture modification, and **gastrostomy (PEG or radiologically inserted) before FVC falls below 50%**, when the procedure becomes hazardous.
27. **Symptom control**: sialorrhoea (glycopyrrolate, hyoscine, amitriptyline, botulinum toxin to salivary glands, salivary gland radiotherapy); spasticity (baclofen, tizanidine, physiotherapy); cramps (levetiracetam, mexiletine, quinine); pseudobulbar affect (dextromethorphan–quinidine, amitriptyline, SSRIs); thick secretions (carbocisteine, nebulised saline); pain; insomnia; depression and anxiety; and communication through augmentative and alternative communication devices.
28. **Early palliative care** and collection of **advance directives** must be an early objective, while cognition is preserved — but clinicians must respect patient autonomy, including the inability of some patients to project themselves into perspectives of death or dependence. **End-of-life issues and symptom relief are inextricably linked.**

Latest Updates (2023–Present)

- **Tofersen (Qalsody)** — the first gene-targeted therapy for ALS. An **intrathecal antisense oligonucleotide** that lowers SOD1 protein production, it received **FDA accelerated approval in April 2023** based on reduction of plasma neurofilament light, with **EU conditional approval (2024)** and **UK approval (2025)**. It applies only to the **<2% of patients with confirmed SOD1 mutations**. Dosing is three loading doses two weeks apart, then every four weeks. **Genetic testing is therefore now essential in all patients with ALS.**
- **AMX0035 / sodium phenylbutyrate–taurursodiol (Relyvrio, Albrioza) has been withdrawn.** The confirmatory phase 3 **PHOENIX trial (2024)** failed both its primary and secondary endpoints, and the manufacturer voluntarily removed the product from the US and Canadian markets in **April 2024**. It should no longer be offered.
- **Radicava ORS**, an oral suspension of edaravone, is now available in several countries, removing the burden of repeated intravenous infusion cycles.
- **Ultra-high-dose mecobalamin (Rozebalamin)** was approved in **Japan (2024)** for early ALS, based on the JETALS trial showing slowed ALSFRS-R decline in patients treated within one year of onset.
- **Gold Coast criteria (2020)** are progressively replacing El Escorial and Awaji in both practice and trials — simpler, with higher sensitivity and no diagnostic-certainty categories; validated across multiple cohorts 2023–2025.
- **Neurofilament light chain** is now an accepted diagnostic, prognostic and pharmacodynamic biomarker, and was the surrogate endpoint on which tofersen was approved. Antisense oligonucleotides targeting **C9orf72** and other genes, and the **ATLAS** trial of tofersen in presymptomatic SOD1 carriers, are in progress.
- **Indian context**: riluzole is available and affordable in India; edaravone and tofersen are severely limited by cost and access. Care is concentrated in tertiary neurology centres, and multidisciplinary MND clinics are few. Under the **Rights of Persons with Disabilities Act 2016**, MND-related disability qualifies for a disability certificate and associated welfare benefits — a practical point examiners appreciate.

Current Practice Update

Textbook Position (Pathy's 6th ed.)	Current Evidence-Based Practice (2023–2026)
Antisense oligonucleotides against SOD1, C9orf72 and ataxin are "being studied"	**Tofersen (Qalsody) is approved** for SOD1-ALS — FDA 2023, EU 2024, UK 2025. **Genetic testing is now mandatory in all ALS.**
AMX0035 not yet addressed / early positive data	**Withdrawn from the US and Canadian markets (April 2024)** after the negative PHOENIX phase 3 trial
Edaravone requires intravenous infusion cycles; EMA withdrew the application in 2019	Oral **Radicava ORS** now available; EMA position unchanged — edaravone remains unapproved in Europe
El Escorial / revised El Escorial / Awaji criteria	**Gold Coast criteria (2020)** increasingly preferred — simpler and more sensitive, especially for early diagnosis
CSF neurofilament described as an emerging biomarker	**Established** diagnostic, prognostic and **pharmacodynamic** biomarker; the basis of tofersen's accelerated approval
Riluzole and NIV prolong survival	**Unchanged** — these, plus multidisciplinary clinic care and timely gastrostomy, remain the interventions with the strongest survival evidence

Examiner's Tip — say these lines verbatim:

- "MND is degeneration of **upper motor neurons of the precentral gyrus and/or lower motor neurons of the ventral horn**; ALS is the commonest adult form."
- "The hallmark is **mixed UMN and LMN signs in the same region**, with **sparing of eye movements, sphincters and sensation**."
- "**Bulbar onset occurs in about one-third and carries a worse prognosis**; respiratory failure is the commonest cause of death."
- "The neuropathological hallmark is **cytoplasmic ubiquitinated, hyperphosphorylated TDP-43 inclusions**."
- "**Many MND-mimic disorders are eminently treatable** — always exclude cervical myeloradiculopathy, multifocal motor neuropathy with conduction block, myasthenia and B_{12} deficiency."
- "**Riluzole and non-invasive ventilation prolong survival**; riluzole gives about three months and raises one-year survival from 49% to 58%."
- "**Gastrostomy should be placed before FVC falls below 50%.**"
- "**Early diagnosis and speedy multiagency intervention facilitate good palliative care; end-of-life issues and symptom relief are inextricably linked.**"

7. Peripheral Neuropathy — Common Causes and Approach to the Patient

Definition and classification

1. **Peripheral neuropathy** is a disorder of peripheral nerves — motor, sensory and/or autonomic — producing weakness, sensory disturbance or autonomic failure.
2. Prevalence is **11% in people aged 55 and over**; the commonest single risk factor is **diabetes mellitus (44% of cases)**, followed by alcoholism, non-alcoholic liver disease and malignancy.
3. **Classify by pattern of nerve involvement**: **mononeuropathy** (a major nerve trunk), **mononeuritis multiplex** (multiple mononeuropathies), **symmetrical polyneuropathy** (terminal branches of multiple nerves, bilateral and similar in distribution), **radiculopathy** (nerve roots) and **polyradiculoneuropathy**.
4. Also classify by **time course** (acute <4 weeks, subacute, chronic >8 weeks), **fibre type** (large fibre vs small fibre) and **pathology** (axonal vs demyelinating) — these four axes together generate the differential diagnosis.

Common causes in older adults

5. **Diabetes mellitus** — the commonest cause. Lifetime prevalence of diabetic neuropathy reaches **50%**; prevalence rises with age (8.4% at 20–34 years to **42.4% at ≥65 years**), and age is an **independent risk factor**. Neuropathic pain occurs in 13.3% of people with diabetes, 8.7% with impaired glucose tolerance, 4.2% with impaired fasting glucose and 1.2% of controls — so **prediabetes also causes neuropathy**.
6. **Alcohol** and non-alcoholic liver disease.
7. **Nutritional**: vitamin B_{12} deficiency (**poor B_{12} status is associated with worse sensory and motor peripheral nerve function in older adults**), thiamine, pyridoxine (both deficiency and excess), vitamin E, folate.
8. **Malignancy and paraproteinaemia**: multiple myeloma, MGUS, AL amyloidosis, POEMS syndrome, anti-Hu paraneoplastic neuronopathy, lymphoma.
9. **Drugs and toxins**: vincristine, cisplatin, taxanes, bortezomib, thalidomide, isoniazid, metronidazole, nitrofurantoin, amiodarone, phenytoin, linezolid; lead, arsenic, thallium, organophosphates.
10. **Infections**: **leprosy** (the commonest treatable cause of neuropathy in India), HIV, hepatitis B and C, syphilis, diphtheria, herpes zoster.
11. **Endocrine and metabolic**: hypothyroidism, chronic kidney disease/uraemia, acromegaly.
12. **Immune-mediated**: Guillain–Barré syndrome (acute), CIDP, multifocal motor neuropathy with conduction block, vasculitic neuropathy (polyarteritis nodosa, rheumatoid, Sjögren's, ANCA-associated), sarcoidosis.
13. **Hereditary**: Charcot–Marie–Tooth disease (HMSN) and **hereditary transthyretin amyloidosis** — the latter frequently mislabelled as idiopathic in older patients.
14. **Entrapment**: carpal tunnel syndrome, ulnar neuropathy at the elbow, common peroneal palsy at the fibular head. **Pressure neuropathy is not diabetes-specific but occurs more commonly in diabetes.**
15. **Idiopathic** (chronic idiopathic axonal polyneuropathy) in 25–30% after complete evaluation.

Clinical approach — history and examination

16. **History**: tempo of onset, distribution and symmetry, positive versus negative symptoms, family history, occupational and toxin exposure, alcohol, a complete drug history, systemic symptoms, diabetes or prediabetes, and diet.
17. **Positive sensory symptoms**: burning, tingling, pins and needles, electric shocks, **allodynia** (pain from a non-painful stimulus) and **hyperalgesia**. **Negative symptoms**: reduced sensation, numbness, a "dead" feeling. Pain from **small-fibre** neuropathy is superficial and burning, worse at night and interferes with sleep; pain from **large-fibre** neuropathy is deep and less sharp. Patients describe walking "barefoot on hot charcoal".
18. **Distal symmetrical polyneuropathy is the commonest pattern** — length-dependent, starting at the toes and ascending, in a **"glove and stocking" distribution**, with sensory symptoms reaching the knees before the fingers are affected.
19. **Examination**: pin prick, light touch, temperature, vibration (128 Hz tuning fork) and joint position sense; muscle strength testing and inspection for wasting; reflexes; gait and Romberg's test. Inspect the feet for ulcers, deformity and Charcot arthropathy; **palpate peripheral nerves for thickening** (leprosy, CMT); check postural blood pressure for autonomic involvement.
20. **Red flags for urgent referral**: rapid progression, marked asymmetry, prominent motor involvement, autonomic failure, or an associated systemic illness — these suggest GBS, vasculitis or malignancy.

Investigations

21. Extensive testing is **not** necessary in a patient with mild symptoms and a known underlying diagnosis such as diabetes or alcohol excess.
22. **In patients with no clear aetiology, electrodiagnostic testing (NCS/EMG) should be the initial diagnostic study** — it distinguishes **axonal from demyelinating** physiology, identifies polyradiculopathy, plexopathy and mononeuritis multiplex, and separates hereditary from acquired demyelinating patterns (Rutkove algorithm).
23. **First-line laboratory tests, directed by the electrodiagnostic result**: full blood count, ESR, TSH, blood glucose and glycated haemoglobin, serum and urine protein electrophoresis with immunofixation, vitamin B_{12}, antinuclear antibody, rheumatoid factor, heavy metals and HIV. Add renal and liver function.
24. **Second-line, if the above are negative**: urinary heavy metals and porphyrins, Sjögren's antibodies, Lyme serology,

anti-Hu antibody, and — in patients with borderline-normal serum B_{12} — **methylmalonic acid and homocysteine**. For demyelinating patterns: lumbar puncture (albuminocytological dissociation), anti-GM1 and anti-MAG antibodies, hepatitis B and C serology.

25. **Specialised tests**: skin biopsy for intraepidermal nerve fibre density in small-fibre neuropathy, **corneal confocal microscopy** as a non-invasive measure of small-fibre loss, autonomic function testing (sympathetic skin response, tilt table, R–R interval, Valsalva), genetic testing for CMT and TTR, and sural nerve biopsy reserved for suspected vasculitis or amyloidosis.

Management

26. **Treatment should address the underlying disease process and alleviate symptoms. Avoidance of toxins such as alcohol or drugs is the most important step.**
27. Disease-directed therapy: glycaemic control (more effective in type 1 than type 2 diabetes), lipid and blood pressure control, withdrawal of the offending drug, B_{12} replacement, thyroxine, dialysis, multidrug therapy for leprosy, and immunotherapy for CIDP (corticosteroids, IVIG, plasma exchange) or vasculitis.
28. **Symptomatic pain relief** — see Question 8. In painful neuropathies, tricyclic antidepressants and gabapentin are effective, with SNRIs and pregabalin preferred in older adults.
29. **Function and falls**: physiotherapy assessment is important in patients with weakness; **ankle–foot orthoses, splints and walking-assistance devices improve function**. Diabetic neuropathy substantially increases the risk of falls — use a structured programme such as **STEADI**, with balance and gait training. Care plans should focus **not only on pain relief but on improving mobility and maintaining independence and quality of life**.
30. **Proper foot and nail care is important in reducing the risk of foot ulcers** — daily inspection, appropriate footwear, podiatry review and prompt treatment of minor injury.

Latest Updates (2023–Present)

- **AAN practice guideline on painful diabetic neuropathy (*Neurology*, reaffirmed 2023–2025)**: gabapentinoids, SNRIs, tricyclics and sodium-channel blockers all provide **comparable benefit**; offer one, and switch class if ineffective. **Opioids are not recommended.** Topical agents and non-pharmacological approaches should be offered.
- **ADA Standards of Care (2025)**: screen for distal symmetrical polyneuropathy **at diagnosis of type 2 diabetes and 5 years after diagnosis of type 1**, then annually, using the **10 g monofilament plus at least one other test** (vibration with a 128 Hz fork, pinprick, ankle reflexes, temperature). **Assess vitamin B_{12} in metformin-treated patients.**
- **Prediabetic and metabolic-syndrome neuropathy** is now firmly established: obesity, hypertriglyceridaemia and hypertension are independent risk factors, and **lifestyle intervention improves intraepidermal nerve fibre density**. This reframes "idiopathic" neuropathy in many older patients as metabolic.
- **Hereditary transthyretin (hATTR) amyloidosis** now has effective disease-modifying therapy — patisiran, vutrisiran, inotersen, eplontersen, and the stabiliser acoramidis — and **HELIOS-B (2024)** confirmed benefit in ATTR cardiomyopathy. **Any older patient with a progressive "idiopathic" axonal neuropathy plus cardiac, autonomic, renal or ocular involvement should be tested for TTR variants.**
- **Small-fibre neuropathy** is increasingly diagnosed by **skin biopsy for intraepidermal nerve fibre density** and **corneal confocal microscopy**; sodium channel (SCN9A/SCN10A/$Na_v1.7$, $Na_v1.8$) variants explain a proportion of idiopathic cases.
- **Pathogenesis-directed agents remain unproven**: alpha-lipoic acid (NATHAN-1) gave inconsistent results, benfotiamine was negative, and the aldose reductase inhibitor epalrestat (widely marketed in India and Japan) has limited Cochrane-level support.
- **Indian context**: India accounts for over half of the world's new leprosy cases — always look for **thickened peripheral nerves and hypopigmented anaesthetic skin patches**, and remember that leprosy is the commonest treatable neuropathy in India (managed under the **National Leprosy Eradication Programme** with WHO multidrug therapy). Under the **National TB Elimination Programme (NTEP)**, **pyridoxine 10 mg daily is mandatory prophylaxis** with isoniazid to prevent neuropathy. ICMR-INDIAB data show a very high and rising burden of diabetes and prediabetes in older Indians.

Current Practice Update

Textbook Position	Current Evidence-Based Practice (2023–2026)
Tricyclic antidepressants are effective for painful neuropathy, as is gabapentin	TCAs remain effective but are **relatively contraindicated in older adults** (anticholinergic burden, QT prolongation); **SNRIs and gabapentinoids are preferred**, with nortriptyline if a TCA is required
Extensive workup often unnecessary; EMG-directed testing	**Unchanged**, but now add **TTR genetic testing** in progressive idiopathic axonal neuropathy with systemic features — a treatable diagnosis
Tight glycaemic control may help maintain nerve function	True for **type 1**; effect is **much weaker in type 2**. Emphasis has shifted to **whole metabolic syndrome control** and lifestyle intervention
Neuropathy in diabetes and prediabetes	Formally recognised — screening is now recommended **at diagnosis of type 2 diabetes**, not years later
Small-fibre neuropathy diagnosed clinically	**Skin biopsy for intraepidermal nerve fibre density and corneal confocal microscopy** are now standard diagnostic tools

Examiner's Tip — say these lines verbatim:

- "Classify by **pattern, time course, fibre type and pathology (axonal vs demyelinating)** — these four axes generate the differential."
- "**Prevalence is 11% in those over 55; diabetes accounts for 44%**, followed by alcohol, liver disease and malignancy."
- "**In an unexplained neuropathy, electrodiagnostic testing is the initial diagnostic study**, and laboratory tests should be directed by its result."
- "**Avoidance of toxins such as alcohol or drugs is the most important single step** in management."
- "In India, always exclude **leprosy** — palpate the nerves and look for anaesthetic skin patches."
- "Management must target **mobility, independence and quality of life**, not pain alone — neuropathy is a major falls risk factor."
- "**Proper foot and nail care reduces the risk of foot ulceration** — never omit this from the answer."

8. Neuritic (Neuropathic) Pain

Definition and classification

1. **Neuropathic pain is pain caused by a lesion or disease of the somatosensory nervous system**, at either a peripheral or a central level.
2. **It is a clinical description, not a diagnosis** — the definition requires a **demonstrable lesion or disease that satisfies standard neurological criteria**.
3. Contrast with **nociceptive pain** (activation of receptors in the peripheral somatosensory nervous system by mechanical, thermal or chemical stimuli in non-neural tissue — soft-tissue, musculoskeletal and visceral pain) and with **nociplastic pain** (altered nociception without clear tissue damage or a somatosensory lesion — fibromyalgia, non-specific low back pain, irritable bowel syndrome, complex regional pain syndrome type 1). "Nociplastic" replaces the older terms *dysfunctional pain*, *central sensitization* and *pain of unknown origin*.
4. **Peripheral causes**: painful diabetic neuropathy, **postherpetic neuralgia**, **trigeminal neuralgia**, radiculopathy, vertebral canal stenosis, carpal tunnel syndrome, chemotherapy-induced neuropathy, post-traumatic and post-surgical neuropathy, phantom limb pain, and **leprous neuritis** (an important Indian cause).
5. **Central causes**: **central post-stroke pain** (thalamic or Déjerine–Roussy syndrome), spinal cord injury, multiple sclerosis, syringomyelia.

Pathophysiology and the effect of ageing

6. Mechanisms: **peripheral sensitisation** with upregulation of sodium channels ($Na_v1.7$, $Na_v1.8$) and ectopic ectopic discharge; **central sensitisation and wind-up** at the dorsal horn; loss of descending inhibitory modulation; deafferentation and cortical reorganisation.
7. **Ageing alters pain processing**: meta-analyses show a modest **increase in pain threshold** (which shortens the warning interval before tissue damage and leads to under-reporting of mild pain, increasing the risk of undiagnosed disease) but a **decrease in pain tolerance**.
8. **Temporal summation of heat pain is greater and capsaicin-induced mechanical hyperalgesia lasts longer in older adults**, indicating prolonged pain facilitation; **endogenous pain inhibition is reduced**. Net conclusion: **severe pain is less well tolerated in older people**.

Clinical features and assessment

9. Descriptors: **burning, shooting, lancinating, electric-shock-like, pricking, tingling or squeezing** pain in a neuroanatomical distribution; usually constant but may be paroxysmal, and typically worse at night.
10. Associated sensory disturbance: **paraesthesiae and dysaesthesiae, allodynia, hyperalgesia and hyperpathia**, often coexisting with sensory loss, impaired motor function, muscle atrophy or abnormal deep tendon reflexes.
11. Assessment: characterise the pain, map its distribution, examine for sensory signs, and identify the causative lesion. Screening tools include **DN4, LANSS and painDETECT**. Assess sleep, mood, function and the impact on activities of daily living.
12. **Pain is a major determinant of frailty** — in the English Longitudinal Study of Ageing, subjects reporting moderate to severe pain were more likely to become frail over the following eight years, independent of age, sex, lifestyle, depressive symptoms and socioeconomic factors.
13. **Pain is not a symptom of dementia, but dementia impairs self-report** and amplifies the impact of pain on function, mood, cognition and tolerance of adverse drug effects. Use observational tools (PAINAD, Abbey, DOLOPLUS-2) when self-report is unreliable.

Pharmacological management

14. **First-line agents**: **gabapentinoids** (gabapentin, pregabalin), **serotonin–noradrenaline reuptake inhibitors** (duloxetine, venlafaxine) and **tricyclic antidepressants** (amitriptyline, imipramine, nortriptyline).
15. **Tricyclics are best avoided in older people** because of their adverse effect profile, especially tertiary tricyclics such as amitriptyline. **If a tricyclic is required, nortriptyline is the safer option.** All TCAs can prolong the QT interval — **obtain an ECG before starting**. Cautions: arrhythmia, recent myocardial infarction, glaucoma and SIADH.
16. **Pregabalin** has linear, dose-proportional absorption, rapid onset and requires minimal titration; but **acute kidney injury and rhabdomyolysis** are recognised adverse effects — monitor renal function and creatine phosphokinase. Both gabapentinoids need dose reduction in renal impairment; sedation, dizziness and ataxia are the limiting effects in the elderly.
17. **SNRIs** lack antimuscarinic effects and are better tolerated than TCAs, but tend to raise heart rate and blood pressure — monitor blood pressure, pulse, creatinine and liver enzymes; avoid with monoamine oxidase inhibitors.
18. **Second-line**: tramadol and **topical agents — 5% lidocaine patches and 8% capsaicin patches** for peripheral neuropathic pain such as postherpetic neuralgia or post-traumatic neuropathy. **Where there are concerns about the safety of systemic treatment, as in the frail elderly, lidocaine patches may be used as first-line therapy.**
19. **Strong opioids have been downgraded to third-line** because of safety concerns.
20. **Efficacy is modest** — the number needed to treat for a 50% reduction in pain is **3.6 for tricyclics, 6.4 for SNRIs, 7.7 for pregabalin and 4.3 for strong opioids**. Combination trials give inconsistent results, and the maximum tolerated dose of each drug is lower in combination, so **titrate one agent at a time**.

21. **Carbamazepine remains first-line specifically for trigeminal neuralgia** (oxcarbazepine as an alternative); monitor full blood count for aplastic anaemia and sodium for hyponatraemia, and beware interactions with warfarin and theophylline.
22. **Not recommended**: cannabinoids cannot be recommended for chronic pain in older people on current evidence; gabapentin and pregabalin are **not** supported for chronic back pain, exposing patients to risk without clear benefit.

Non-pharmacological and preventive management

23. **Education** is fundamental — the nature of pain, realistic expectations, and the benefits and risks of each option. **Dispel the misconception that pain can always be eliminated**; educate family and carers, particularly where there is cognitive impairment.
24. **Cognitive-behavioural therapy**, graded exercise, physiotherapy, TENS, acupuncture, desensitisation and mirror therapy. Effect sizes are small to moderate but the **safety profile is better than drugs**; used as an adjunct they permit lower drug doses and fewer adverse effects.
25. **Interventional options** for refractory pain: nerve blocks, epidural steroid injection, radiofrequency ablation, spinal cord stimulation, intrathecal therapy.
26. **Prevention**: **recombinant zoster vaccination** prevents herpes zoster and postherpetic neuralgia; good glycaemic and metabolic control reduces incident diabetic neuropathy; pyridoxine with isoniazid; and prompt corticosteroid treatment of leprous neuritis prevents permanent nerve damage.
27. **Principles in the elderly**: start low, go slow, one drug at a time, set realistic goals (30–50% pain reduction, improved sleep and function rather than freedom from pain), review regularly, and **deprescribe if ineffective**.

Latest Updates (2023–Present)

- **OPTION-DM (*Lancet* 2022, informing 2023–2025 practice)**: in painful diabetic neuropathy, **amitriptyline, duloxetine and pregabalin have similar efficacy** as monotherapy, and **combination therapy is superior when a single agent gives only partial relief** — supporting a "try one, then combine" rather than "switch endlessly" strategy.
- **Phenotype-stratified treatment** is gaining traction — an "irritable nociceptor" phenotype predicts response to sodium-channel blockade (oxcarbazepine), supporting mechanism-based rather than aetiology-based prescribing.
- **Gabapentinoid safety (2023–2025)**: pharmacovigilance and regulatory warnings highlight **respiratory depression when combined with opioids or in COPD**, plus falls, peripheral oedema, weight gain and misuse potential. They are now controlled drugs in the UK. This reinforces cautious use in older adults.
- **Suzetrigine (VX-548, Journavx)** — a first-in-class **selective $Na_v1.8$ inhibitor**, FDA-approved in **January 2025** for moderate-to-severe acute pain. It is **non-opioid and does not act centrally**, so it avoids sedation, falls and dependence; phase 2/3 studies in painful diabetic neuropathy and lumbosacral radiculopathy are ongoing. This is the most important mechanistic advance for geriatric pain in a decade.
- **High-concentration capsaicin 8% patch** now has broadened indications with a single 30-minute application to the feet in diabetic neuropathy, giving up to 3 months of relief.
- **Recombinant zoster vaccine (RZV, Shingrix)**: long-term follow-up of ZOE-50/70 (2024) shows efficacy sustained beyond 10 years; recommended for all adults ≥50 years and immunocompromised adults ≥18. Now available in India, though cost-limited.
- **Indian context**: amitriptyline, nortriptyline, gabapentin, pregabalin and duloxetine are all available as inexpensive generics. **Leprous neuritis** is a distinctly Indian cause of "neuritic pain" — acute neuritis in type 1 or type 2 lepra reaction requires **oral prednisolone (typically 40 mg tapered over 12 weeks) under the National Leprosy Eradication Programme** alongside multidrug therapy, and is a neurological emergency because delay causes irreversible deformity.

Current Practice Update

Textbook Position (Pathy's 6th ed.)	Current Evidence-Based Practice (2023–2026)
Combination pharmacotherapy gives "inconsistent results"	**OPTION-DM** supports **adding a second agent** when monotherapy gives partial relief, rather than serial switching
Gabapentinoids are first-line and generally well tolerated	Still first-line, but with **formal safety warnings** — respiratory depression with opioids/COPD, falls, oedema, misuse; now controlled drugs in the UK
Drug classes limited to antidepressants, anticonvulsants, opioids and topicals	**Suzetrigine (selective $Na_v1.8$ inhibitor)** approved January 2025 — first genuinely new non-opioid mechanism, trials in neuropathic pain ongoing
Lidocaine patch as a second-line agent, first-line only in the frail	**Unchanged and increasingly emphasised** — safest option in frailty, polypharmacy and cognitive impairment
Cannabinoids cannot be recommended in older people	**Unchanged** — subsequent systematic reviews have not altered this position

Examiner's Tip — say these lines verbatim:

- "Neuropathic pain is **pain caused by a lesion or disease of the somatosensory nervous system**. It is a **clinical description, not a diagnosis** — a demonstrable lesion satisfying neurological criteria is required."
- "The three mechanistic descriptors are **nociceptive, neuropathic and nociplastic**."

- "With ageing, **pain threshold rises but pain tolerance falls**, temporal summation increases and endogenous inhibition is reduced — so **severe pain is less well tolerated in older people**."
- "First-line: **gabapentinoids, SNRIs and tricyclics**. **Avoid amitriptyline in the elderly; use nortriptyline if a tricyclic is essential**, with a prior ECG."
- "**Strong opioids have been downgraded to third-line**; **lidocaine 5% patches may be first-line in the frail elderly**."
- "NNT for 50% pain relief: **3.6 tricyclics, 6.4 SNRIs, 7.7 pregabalin, 4.3 strong opioids** — efficacy is modest, so combine with non-pharmacological therapy."
- "**Pain is a major determinant of frailty** — moderate to severe pain independently predicted incident frailty over eight years in ELSA."
- "In India, always consider **leprous neuritis** — it requires urgent corticosteroids to prevent irreversible nerve damage."

9. Management of Extracranial Carotid Artery Stenosis

Pathogenesis and definitions

1. **Atherosclerosis is the commonest arterial disorder in developed countries** and, when complicated by thrombosis and embolism, causes ischaemic stroke, myocardial infarction and peripheral arterial disease.
2. Plaque forms where there is endothelial injury and haemodynamic stress — in the cerebral circulation this is at **sites of arterial branching, especially the bifurcation of the common carotid artery**. LDL cholesterol enters the subendothelium, is oxidised by macrophages and smooth muscle cells, and growth factors and cytokines drive foam cell accumulation and smooth muscle proliferation.
3. Plaques with a fatty core and fibrous cap narrow the lumen and stiffen the wall. **A defect in the fibrous cap from rupture or erosion predisposes to thrombosis** — platelets adhere and are activated, which is the site of action of antiplatelet drugs.
4. **Most strokes and TIAs secondary to carotid stenosis occur by thromboembolism**; haemodynamic hypoperfusion is a relatively rare cause because of collateral supply through the circle of Willis.
5. Risk factors: cigarette smoking, diabetes mellitus, age, dyslipidaemia, hypertension and genetic predisposition. **Arterial dissection is a rare cause in the elderly** (common in the young), usually after minor neck or head trauma with sudden neck rotation.
6. **Symptomatic** stenosis = ipsilateral TIA, ischaemic stroke or amaurosis fugax within the preceding six months.

Investigation and grading

7. **The degree of carotid stenosis should be reported using the NASCET method**, in which the diameter at the stenosis is compared with the diameter of the **distal internal carotid artery**. ECST used a different denominator, so the two are not interchangeable.
8. **Doppler ultrasound** is the usual first-line non-invasive test (70–79% stenosis corresponds to peak systolic velocity >230 cm/s and end-diastolic velocity 110–140 cm/s).
9. **CT angiography** is increasingly replacing MRA and duplex because it is often combined with the initial stroke CT; limitations are contrast toxicity in renal impairment, contrast allergy and poor visualisation with heavy plaque calcification. **MRA** is limited by tolerability, metallic implants and misinterpretation of swallowing artefact or turbulent flow as stenosis.
10. **Catheter angiography is the gold standard** but carries a **0.5–2% risk of stroke or TIA** from plaque dislodgement, dissection or catheter thrombus — it should be preceded by non-invasive imaging and performed only in experienced neurovascular units.

Best medical therapy — for every patient

11. **Antiplatelet therapy** — aspirin, clopidogrel, or aspirin with extended-release dipyridamole; short-course dual antiplatelet therapy after minor stroke or high-risk TIA.
12. **High-intensity statin** — SPARCL showed atorvastatin 80 mg reduced recurrent ischaemic stroke by around 20%; treat cholesterol above 3.5 mmol/L, with dietetic input.
13. **Blood pressure control** (PROGRESS: perindopril-based regimen); target ≤130/80 mmHg in diabetic patients; plus glycaemic control, smoking cessation and lifestyle advice.
14. **Anticoagulation is not indicated for carotid stenosis itself** — it is reserved for atrial fibrillation, prosthetic valves or another specific indication. Heparin in acute stroke gives no net benefit.

Symptomatic stenosis — the operative rules

15. **All patients with suspected non-disabling stroke or TIA who, after specialist assessment, are considered candidates for carotid endarterectomy should have carotid imaging within 24 hours of symptom onset.**
16. **Symptomatic stenosis of 50–99% (NASCET)** with stable neurological symptoms: **assess and refer for carotid endarterectomy as soon as possible, within seven days of symptom onset**, in a vascular surgical centre, **plus** optimum medical treatment.
17. **Symptomatic stenosis <50% (NASCET)**: **should not undergo carotid intervention**; best medical treatment only.
18. **Evidence** (pooled ECST and NASCET, Rothwell, *Lancet* 2003): the absolute risk reduction of ipsilateral ischaemic stroke or death with CEA for **70–99% NASCET stenosis was 16%** ($p<0.001$). Patients with 50–69% stenosis benefited by a slimmer margin, and **below that threshold there was overall harm**. Benefit requires a **perioperative stroke or death risk of no more than 5%**.
19. Subgroups deriving **additional benefit**: **male sex, advancing age and recent symptoms**. Near-occlusion derives no benefit.

Asymptomatic stenosis — textbook position

20. **ACAS**: CEA reduced the 5-year risk of ipsilateral stroke or perioperative stroke/death from **11% to 5.1%**, but not the risk of major stroke. **ACST**: immediate CEA reduced the 5-year risk of all strokes and perioperative death from **11.8% to 6.4%**. The 30-day stroke or death rate after CEA in both trials was around 3%.
21. **ACST subgroup analysis showed no evidence of benefit in patients older than 75 or in women.** These trials predate widespread statin use, good blood pressure control and routine dual antiplatelet therapy, so with modern medical management the stroke rate in asymptomatic stenosis (already only ~2% per annum) would be lower still,

potentially negating any benefit of surgery.

22. **Conclusion from the textbook: patients over 75 with asymptomatic stenosis and those with less than 60% stenosis should not be offered surgery.** Those managed medically must be educated about TIA and stroke symptoms and told to present urgently.

Carotid artery stenting

23. **Advantages**: performed under local anaesthesia, avoiding general anaesthesia; faster recovery and shorter stay; no neck incision, so less haematoma, infection and **cranial nerve injury**. **Disadvantages**: distal embolisation during catheter passage and during stent deployment and post-dilatation, greater restenosis, and the cost of the stent and cerebral protection device.
24. **Pooled analysis of four trials (Bonati, *Lancet* 2010)**: stroke or death within 120 days was **8.9% after stenting vs 5.8% after endarterectomy** (RR 1.53). CEA carries a small excess of myocardial infarction (<1%) and cranial nerve injury.
25. **Age is the key modifier (Howard, *Lancet* 2016)**: in symptomatic patients, procedural stroke or death was **identical below age 70**, and **all the excess risk of stenting was in older patients**. Long-term effectiveness at preventing recurrence is similar at all ages.
26. **Textbook/NICE indication for stenting**: patients who meet criteria for intervention but are **unsuitable for surgery** — inaccessible or high cervical bifurcation, restenosis after endarterectomy, radiotherapy-associated stenosis, fibromuscular dysplasia and dissection.
27. **Vertebral and intracranial disease**: vertebral artery origin stenting is relatively safe but has not been shown superior to medical management (VIST); intracranial stenting carries risks of dissection, rupture and plaque embolisation and is reserved for recurrent symptoms despite medical therapy.
28. **Geriatric decision-making**: benefit accrues over years, so weigh frailty, cognition, comorbidity and life expectancy. A frail patient with limited life expectancy should be managed medically.

Latest Updates (2023–Present)

- **CREST-2 (*NEJM*, 21 November 2025; Brott, Howard, Lal et al.) — the single most important carotid trial of the decade.** Two parallel, observer-blinded randomised trials, **2,485 patients with asymptomatic stenosis ≥70%**, 155 centres in five countries, mean age 69–70, follow-up 3.6–4.0 years. Primary outcome (any stroke or death within 44 days plus ipsilateral ischaemic stroke to 4 years):
  - **Stenting + intensive medical management 2.8% vs intensive medical management alone 6.0% (p = 0.02) — significant benefit**, NNT approximately 31 at 4 years.
  - **Endarterectomy + intensive medical management 3.7% vs intensive medical management alone 5.3% (p = 0.24) — NOT significant.**

  The accompanying *NEJM* editorial (Brown & Bonati, 2025) concluded that **"there is no longer a role for routine carotid endarterectomy in persons with asymptomatic stenosis."**
- **ECST-2 (*Lancet Neurology* 2025)**, 2-year interim results: in asymptomatic or low-to-intermediate-risk symptomatic carotid stenosis, **optimised medical therapy alone was not inferior to optimised medical therapy plus revascularisation**.
- **SPACE-2** 5-year results similarly showed **no advantage of CEA or CAS over best medical treatment** in moderate-to-severe asymptomatic stenosis.
- **ESVS 2023 guidelines** recommend intervening in asymptomatic stenosis only where **"high-risk-for-stroke" features** are present: stenosis progression, silent ipsilateral infarction on imaging, intraplaque haemorrhage on MRI, plaque echolucency or large juxtaluminal black area, contralateral occlusion, and spontaneous embolisation on transcranial Doppler.
- **Transcarotid artery revascularisation (TCAR)** with flow reversal has emerged as a third option, with registry data showing lower stroke rates than transfemoral stenting, particularly in older and anatomically high-risk patients.
- **Indian context**: extracranial carotid disease accounts for a **smaller proportion of Indian ischaemic stroke** than in Western cohorts, where **intracranial atherosclerosis and small-vessel disease predominate**. Carotid duplex is widely available and inexpensive; CEA and CAS are confined to tertiary centres. **The pragmatic Indian priority is aggressive medical therapy — statin, blood pressure control, antiplatelet, glycaemic control and smoking cessation.** Note also that CHANCE-2 genotype-guided antiplatelet data are relevant given the high frequency of CYP2C19 loss-of-function alleles in Indian populations.

Current Practice Update

Textbook Position (Pathy's 6th ed.)	Current Evidence-Based Practice (2023–2026)
Asymptomatic stenosis: CEA reduces stroke (ACAS, ACST), but benefit uncertain with modern medical therapy; do not operate on those >75 or with <60% stenosis	**CREST-2 (2025) resolves this**: **CEA gave no significant benefit** over intensive medical management, while **stenting did** (2.8% vs 6.0%). Editorialists state there is **no longer a role for routine CEA in asymptomatic stenosis**
Stenting reserved for those unsuitable for surgery, because of an excess periprocedural stroke risk	For **asymptomatic** ≥70% stenosis, CREST-2 supports **stenting with embolic protection as first-line revascularisation** where anatomy is suitable and the operator is skilled. **For symptomatic stenosis, CEA remains the standard**, particularly over age 70
Symptomatic 50–99%: CEA within 7 days; imaging within 24 hours	**Unchanged and reinforced** — this remains the highest-yield rule for the examination
Best medical therapy = antiplatelet, statin, BP control	Intensified to **"intensive medical management"** — protocolised LDL and BP targets with adherence monitoring and lifestyle coaching, as delivered in CREST-2, which markedly lowered event rates in all arms
Selection based on degree of stenosis alone	**ESVS 2023** adds **plaque vulnerability features** (intraplaque haemorrhage, echolucency, silent infarction, TCD embolisation) to the decision

Examiner's Tip — say these lines verbatim:

- "**The degree of stenosis must be reported by the NASCET method** — the stenosis diameter compared with the distal internal carotid artery."
- "**Carotid imaging within 24 hours; endarterectomy within 7 days** for symptomatic 50–99% stenosis, plus optimum medical therapy."
- "For 70–99% symptomatic stenosis the **absolute risk reduction was 16%**, provided the **perioperative stroke or death risk is no more than 5%**."
- "**Symptomatic stenosis under 50% should not undergo intervention** — there is net harm."
- "**All the excess risk of stenting compared with endarterectomy is in patients over 70**; below 70 the risks are identical."
- "**CREST-2 (NEJM 2025)** showed that in asymptomatic ≥70% stenosis, **stenting added benefit to intensive medical therapy but endarterectomy did not** — editorialists concluded there is no longer a role for routine CEA in asymptomatic disease."
- "Most strokes from carotid stenosis are **thromboembolic**, not haemodynamic, because of collateral flow through the circle of Willis."

10. Bedside Assessment of Swallow in a Patient with Recent Stroke

Why it matters

1. **Dysphagia is present initially in 78% of patients who have had a stroke**; although acute dysphagia improves in about 80% of survivors, its early consequences are severe.
2. Complications: **aspiration leading to stroke-associated pneumonia (SAP)**, malnutrition, dehydration, longer hospital stay, poorer participation in rehabilitation and worse functional outcome. Dysphagia is associated with significantly higher mortality and morbidity, and with more nursing-home admissions.
3. South London Stroke Register data show **dysphagia at stroke onset was associated with a three- to fivefold increase in institutionalisation at five years**.
4. **Delays in bedside dysphagia screening and in comprehensive assessment by a speech and language therapist are associated with a higher risk of stroke-associated pneumonia.** Since SAP is one of the main causes of mortality after acute stroke, **early dysphagia assessment may prevent deaths and can be implemented even in settings without high-technology specialist stroke care**.
5. **Silent aspiration** — aspiration without cough or any outward sign of difficulty — occurs in a substantial number of patients, who are at increased risk because clinical swallowing assessments under-diagnose the problem.
6. **The golden rule: keep the patient nil by mouth, including oral medication, until a swallow screen has been passed.**

Prerequisites and who screens

7. Screening is a **pass/fail procedure** that identifies who needs comprehensive assessment. It may be conducted by various professionals including a nurse or a speech-language pathologist, but **employing a swallow-assessment specialist may reduce the odds of stroke-associated pneumonia**.
8. **The patient must be fully conscious and aware** — it is very difficult to swallow safely if one is not.
9. **The patient must be able to sit upright and to cough purposefully**; if unable to do either, the likelihood of aspiration is increased and a more in-depth assessment is indicated.
10. Before testing, ensure oral hygiene, correctly fitting dentures, an upright (90°) position with the chin slightly tucked, and suction available at the bedside.

Risk indicators to elicit (Pathy's Box 49.1)

11. Weak, husky voice (**dysphonia**); inability to cough voluntarily; a **weak cough with no effective expulsion**.
12. **Pooling of food or saliva in the mouth or cheek**; frequent coughing or choking **even on saliva**.
13. History of a recent chest infection; the patient complaining of difficulty swallowing, of having to gulp, of an abnormal sensation, or of difficulty with particular types of food.
14. Related symptoms: choking during eating or drinking, inability to swallow saliva, **nasal regurgitation** of food or drink, and discomfort during swallowing.

Structured bedside examination

15. Assess **oral and laryngeal structures and movements, dental state, cognition, posture** and any other factor bearing on the safety of oral feeding — lip closure, tongue movement, palatal elevation, jaw movement, voice quality, volitional cough, and laryngeal elevation on palpation during a dry swallow.
16. **Critically: the absence of a gag reflex does not automatically indicate a major swallowing problem, just as the presence of the gag does not indicate a safe swallow.** The gag reflex is unreliable and must never be used alone.
17. The important factors are **bolus control**, **clarity of the voice** (is it wet and gurgly after a test swallow?) and **effectiveness of the cough** (is it firm and effective?).

Trial swallows and observation

18. **Try teaspoons of water; if successful, try teaspoons of soft purée; and if that is successful, try foods with more substance.**
19. **Observe for**: the patient reporting a negative sensation; poor lip closure or degree of leakage; untimely or absent elevation of the larynx; residue in the mouth following the swallow; **lack of clarity of voice following the swallow**; and choking or coughing **before, during or after** the swallow.
20. **Formal screening protocols**:
    - **Water swallow test** — several protocols exist; for example the patient drinks **3 oz (90 mL) of water without interruption**, and is monitored for completion, coughing, choking and a wet or hoarse vocal quality during or within one minute of completion.
    - **Multiple consistency test** — stepwise evaluation of non-fluid and fluid textures giving a graded rating (severe, moderate, mild or no dysphagia).
    - **Swallowing provocation test** — injection of a water bolus through a nasal catheter into the oropharynx; the patient is at risk of aspiration if the swallow reflex takes **more than 3 seconds**.
21. **Limitations**: the water swallow test is sufficient for screening for aspiration after stroke in much of the literature, but its **diagnostic accuracy has been questioned** — bedside screening is not consistently sensitive, and **no bedside**

protocol has been adequately predictive of aspiration. It nevertheless remains the mainstay because it is safe, straightforward and easily repeated. **Pulse oximetry** may assist, since decreased blood oxygenation during swallowing has been observed in dysphagic patients.

What follows a failed screen

22. **Fail → remain nil by mouth**, start intravenous or nasogastric fluids, and refer to speech and language therapy. A **non-instrumental assessment** observes swallowing behaviours — fatigue during a meal, signs and symptoms of dysphagia, posture and positioning — but **aspiration cannot be observed** by this means.
23. **Videofluoroscopy (modified barium swallow) is the gold standard**, giving a direct radiographic view of oral, pharyngeal and upper oesophageal function with varied consistencies mixed with barium. It is **not feasible as a routine screening option**, and it is not infallible — **false negatives** occur when dysphagia appears only with fatigue after several mouthfuls, and **false positives** when aspiration is asymptomatic or induced by the tension of the situation or the unpleasantness of the contrast. Findings must be placed in the context of the patient's history.
24. **FEES** (fibre-optic endoscopic evaluation of swallowing) is a **portable bedside** alternative that directly visualises the posterior pharynx, epiglottis, vocal cords and proximal trachea during trials of varied consistencies, and can monitor the effectiveness of therapy — but **it does not visualise the oral or oesophageal phase**.
25. **Management** is interdisciplinary, aimed at protecting the patient from complications, maintaining adequate nutrition, and ensuring the patient and carers understand the deficit. Compensatory techniques and **dietary/texture modification under speech and language therapy and dietetic supervision remain the mainstay**. Patients with reduced oral sensation, poor buccal control or inadequate laryngeal lift **aspirate more on fluids than on semisolids** and may be advised to avoid liquids while managing a soft puréed diet; patients with tongue, lip or jaw involvement should avoid foods requiring chewing.
26. **Persistent dysphagia requires alternative nutrition** — nasogastric tube early, and percutaneous endoscopic gastrostomy if dysphagia is likely to persist. Other measures include mechanical or thermal pharyngeal stimulation, cortical magnetic stimulation of the swallowing reflex, and electrical pacemakers to trigger laryngeal elevation.
27. **Re-screen daily in the early days** — swallow status changes rapidly after stroke. Maintain rigorous oral care throughout.

Latest Updates (2023–Present)

- **Timing is now protocolised**: the AHA/ASA and the **2023 National Clinical Guideline for Stroke for the UK and Ireland** require dysphagia screening by a trained professional **before any oral intake including oral medication, ideally within 4 hours of admission**, with speech and language therapy assessment **within 24 hours of a failed screen and no later than 72 hours**.
- **Validated screening tools**: the **Gugging Swallowing Screen (GUSS)** and the **Yale Swallow Protocol (3 oz water test)** are the best-validated nurse-administered bedside screens, with high sensitivity for aspiration risk; guidelines now specify use of a validated tool rather than an unstructured assessment.
- **Prophylactic antibiotics do not prevent stroke-associated pneumonia** (STROKE-INF and related trials). The evidence-based preventive strategy is **early dysphagia screening plus structured oral care**, not antibiotics.
- **Pharyngeal electrical stimulation**: PHAST-TRAC showed benefit for decannulation in tracheostomised stroke patients, but larger trials in general post-stroke dysphagia (PHADER and others) have given mixed results. Capsaicin and other TRPV1 agonists to re-sensitise the swallow reflex are under active investigation.
- **The IDDSI framework** (International Dysphagia Diet Standardisation Initiative, levels 0–7) has now replaced older national texture descriptors worldwide and should be used when prescribing modified diets and thickened fluids.
- **Indian context**: swallow screening before feeding is endorsed in Indian stroke unit practice, but speech and language therapists are scarce outside tertiary centres — which makes the **nurse-administered bedside water swallow test the single most important, transferable and cost-effective intervention**. Ryle's tube feeding remains the default because PEG access and cost are limiting. The **ATTEND trial (*Lancet* 2017, India)** established family-led rehabilitation as a feasible Indian model, and carer training in safe feeding and oral care is directly applicable.

Current Practice Update

Textbook Position (Pathy's 6th ed.)	Current Evidence-Based Practice (2023–2026)
Bedside assessment described as a structured clinical observation without a specified time window	**Screen within 4 hours of admission and before any oral intake**; SLT assessment within 24 hours of a failed screen (2023 UK–Ireland guideline)
Water swallow test used with several unspecified protocols	Use a **validated tool** — **GUSS** or the **Yale Swallow Protocol** — rather than an unstructured trial
"No bedside screening protocol has been adequately predictive of aspiration"	**Still true**, but validated multi-consistency screens (GUSS) have materially improved sensitivity; instrumental assessment remains definitive
Texture modification described in general terms	Standardised globally through the **IDDSI framework (levels 0–7)** for both foods and fluids
Prevention of pneumonia framed around aspiration risk detection	Adds **structured oral care** as an evidence-based measure; **prophylactic antibiotics do not work** (STROKE-INF)
Pharyngeal and cortical stimulation listed as experimental adjuncts	Pharyngeal electrical stimulation has an established role in **tracheostomy decannulation** (PHAST-TRAC); general dysphagia data remain mixed

Examiner's Tip — say these lines verbatim:

- "**78% of stroke patients initially present with dysphagia**, and **delays in screening and assessment increase the risk of stroke-associated pneumonia**, a main cause of post-stroke mortality."
- "**Keep the patient nil by mouth, including oral medication, until the swallow screen is passed.**"
- "The patient must be **fully conscious, able to sit upright and able to cough purposefully** before any trial swallow."
- "**The absence of a gag reflex does not indicate a major swallowing problem, and its presence does not indicate a safe swallow** — assess bolus control, voice clarity and cough effectiveness instead."
- "Give **teaspoons of water, then soft purée, then more substantial food**, watching for a **wet, gurgly voice**, delayed laryngeal elevation, oral residue and coughing before, during or after the swallow."
- "**Videofluoroscopy is the gold standard but produces both false positives and false negatives**; FEES is portable but does not show the oral or oesophageal phase."
- "**Silent aspiration** means bedside assessment under-diagnoses the problem — a normal screen never fully excludes aspiration."

11. Visual Impairment — Age-Related Causes and Clinical Approach RECURS

Scale and significance

1. **As many as 75% of older adults have significant visual or auditory dysfunction that is not reported to their physician** — therefore adequate screening for these problems is important, and correction of remediable conditions may improve the ability to perform daily activities.
2. Consequences of untreated visual impairment: **limited functional activity, social isolation and depression**, falls and fractures, driving cessation, medication errors, and accelerated cognitive decline.
3. **The prevalence of visual problems and blindness increases with age.** The four commonest causes of blindness in older adults are **cataract, glaucoma, macular degeneration and diabetic retinopathy**.

Physiological and functional changes of the ageing eye

4. **Presbyopia** develops from **nuclear sclerosis of the lens and atrophy of the ciliary muscle**; the near point of accommodation recedes so that by about age 45 comfortable reading requires corrective lenses.
5. **Latent hyperopia becomes manifest** with loss of accommodative reserve; alternatively **myopia increases** as old lens fibres accumulate in the nucleus, which becomes compact and harder (nuclear sclerosis), increasing the refractive power of the lens.
6. **Diminished tear secretion**, especially in postmenopausal women, causes dry eye with irritation and discomfort and may endanger the integrity of the corneal surface; treatment is substitution therapy with frequent artificial tears.
7. **Corneal endothelial cells seldom proliferate in adult life**, so the cell population falls with age, leaving an irregular anterior chamber surface where pigment accumulates. Dense pigment accumulation slightly reduces acuity, and progressive endothelial dystrophy may cause corneal oedema requiring **corneal transplantation**.
8. **Intraocular pressure slowly increases with age.** Also: senile miosis, slower dark adaptation, reduced contrast sensitivity, increased glare sensitivity, and impaired blue–yellow discrimination from lens yellowing.

The four major diseases

9. **Senile cataract** — opacification of the crystalline lens; prevalence **46% at ages 75–85** (Framingham Eye Study). Development is **painless, progressive and variable**, typically bilateral and often asymmetrical. Patients complain of problems with **night driving, reading road signs and difficulty with fine print**. The cause is unknown but opacification is associated with breakdown of the γ-crystallin proteins, and **ultraviolet light may be a contributing factor**. The process may occur in the cortex or the nucleus.
10. **Glaucoma** — a group of disorders characterised by **increased intraocular pressure, progressive excavation of the optic nerve head with damage to nerve fibres, and a specific loss in the visual field**. Prevalence of open-angle glaucoma rises with age to **7.2% at ages 75–85**, with much higher rates in men.
11. **Chronic open-angle glaucoma** (the commoner variety) has an **insidious onset and slow progression**: the irido-corneal angle is open but aqueous outflow is diminished, the neuroretinal rim thins from loss of retinal ganglion cell axons, producing optic-nerve cupping. Central acuity may remain normal for a long time while **peripheral field defects are characteristic and gradually progressive — initially a paracentral scotoma, which may coalesce, and a nasal step, until the entire field constricts**.
12. **Angle-closure glaucoma** is acute and relatively infrequent, with a **sudden painful attack of raised intraocular pressure and marked loss of vision**. Treatment is to normalise the pressure with miotic drops, carbonic anhydrase inhibitors or osmotic agents; the **definitive treatment is surgical or laser iridectomy**, and because the disease is usually bilateral, prophylactic iridectomy of the second eye is often proposed.
13. **Age-related macular degeneration** — the commonest cause of **legal blindness (20/200 or worse)** in older adults; prevalence 28% at ages 75–85, higher in women. Causes loss of **central** vision (see Question 12).
14. **Diabetic retinopathy** — prevalence rises with age to about **7% at ages 75–85**. Visual loss is usually related to vascular changes in and around the macula: **leakage of serous fluid causes macular oedema** (may respond to laser photocoagulation), **haemorrhage within the macula** causes more permanent loss, and **loss of retinal capillaries causes macular ischaemia with a poor prognosis for recovery**.
15. Other important causes: **uncorrected refractive error** (the commonest and most easily reversible cause worldwide), **giant cell arteritis** (sudden painless visual loss — a medical emergency requiring immediate high-dose steroid), retinal artery and vein occlusion, retinal detachment, vitreous haemorrhage, homonymous field defects after stroke, and drug toxicity (ethambutol, hydroxychloroquine, amiodarone).

Clinical approach

16. **History**: onset (sudden versus gradual), laterality, central versus peripheral loss, pain, redness, glare, floaters and flashes, **metamorphopsia**, diplopia; difficulty with specific tasks (reading, driving, recognising faces, negotiating stairs); falls history; full medication review; and systemic disease (diabetes, hypertension, symptoms of giant cell arteritis).
17. **Sudden painless visual loss** → retinal detachment, vitreous haemorrhage, retinal artery or vein occlusion, giant cell arteritis, wet AMD, occipital stroke — **same-day ophthalmology referral**. **Sudden painful visual loss with a red eye** → acute angle-closure glaucoma, uveitis, keratitis.
18. **Ophthalmological screening** comprises **visual acuity** (ability to read newspaper-sized print), **ophthalmoscopic examination** of lens and fundus, **tonometry** for intraocular pressure, and **visual field testing**.

19. **Bedside additions**: Snellen chart with **pinhole** (improvement indicates refractive error), near-vision card, confrontation fields, **Amsler grid** (central distortion in AMD), pupils for a relative afferent pupillary defect, red reflex, colour vision, and dilated fundoscopy.
20. **Pattern recognition (Essentials Table 13-4)**: pain, red eye and a fixed pupil → angle-closure glaucoma; glare intolerance with loss of acuity → cataract; loss of peripheral vision with optic disc changes → open-angle glaucoma; sudden visual loss → temporal arteritis or diabetic retinopathy; loss of central acuity with retinal vessel changes and exudates → macular degeneration or diabetic retinopathy.
21. Systemic evaluation: blood pressure, HbA1c, and **ESR/CRP urgently if giant cell arteritis is suspected**.

Management principles

22. **Consider cataract surgery when the cataract progresses to interfering with activities**; **consider glaucoma when defects in the peripheral visual field are present**; and **consider AMD when central visual acuity is affected**.
23. **Restoring vision after cataract surgery**: an **intraocular lens** corrects central and peripheral vision, increases image size by only **1%**, can be used after surgery on one or both eyes, is useful for older adults unable to wear contact lenses, but requires bifocal spectacles and introduces added surgical and postsurgical complications. **Contact lenses** increase apparent object size by **6%** and are difficult for many older people to handle.
24. **Visual impairment is a known risk factor for falls and fractures, and patients who have had cataract surgery have a lower risk of hip fracture.**
25. **Glaucoma** is usually treated medically first with miotics and β-blocking agents (which have the advantage of not changing pupil diameter), with combination drops and systemic carbonic anhydrase inhibitors in severe cases; **surgery or laser therapy is indicated only if disease progresses on maximal medical therapy**.
26. **Diabetic retinopathy**: **initiate intensive blood glucose control and tight blood pressure control to reduce retinopathy in type 2 diabetes**; panretinal and focal laser photocoagulation reduce the risk of visual loss in severe retinopathy and macular oedema respectively.
27. **Aids to maximise visual function**: magnifying device; lighting intensifiers without glare; tinted glasses to reduce glare; a night light to assist dark adaptation; and large-print newspapers, books and magazines. **Refer all patients with AMD for visual rehabilitation.**
28. **Beware systemic absorption of ophthalmic medications** — these agents may cause other organ system dysfunction and interact with other medications. β-blocker drops can cause bradycardia, bronchospasm and heart failure. Assess the patient's other medical problems and medications, **use the minimum dose to achieve the desired effect, and monitor for systemic toxicity**.
29. Screen for and treat **depression**, which commonly follows the loss of quality of life. Recognise **Charles Bonnet syndrome** — formed visual hallucinations with retained insight in the visually impaired — and reassure rather than misdiagnose psychosis or dementia.

Latest Updates (2023–Present)

- **The *Lancet* Commission on dementia prevention, intervention and care (2024) added untreated vision loss as a new modifiable risk factor for dementia**, contributing approximately 2% of the population-attributable fraction, and observational data associate **cataract surgery with reduced dementia incidence**. This is the single highest-yield update for this question.
- **Dual sensory impairment** (combined hearing and vision loss) has **multiplicative** effects on dementia, falls, depression and mortality — a growing 2023–2025 literature relevant to comprehensive geriatric assessment.
- **Screening guidance diverges**: the **USPSTF (2022) issued an "I" statement** — insufficient evidence for routine primary-care vision screening in asymptomatic older adults — while the AAO and geriatric societies recommend a **comprehensive dilated eye examination every 1–2 years after age 65**. Know both positions.
- **Glaucoma**: the **LiGHT trial 10-year extension (2024)** established **selective laser trabeculoplasty as first-line therapy** — more effective, more cost-effective and with less need for surgery than eye drops. Newer agents include Rho-kinase inhibitors (netarsudil) and latanoprostene bunod, plus preservative-free formulations and **minimally invasive glaucoma surgery (MIGS)** combined with cataract extraction.
- **WHO World Report on Vision** targets for 2030: a 40 percentage-point increase in effective refractive error coverage and a 30 percentage-point increase in effective cataract surgical coverage. **Uncorrected refractive error and cataract remain the leading global causes.**
- **Indian context**: the **National Blindness and Visual Impairment Survey (2015–19)** reported blindness prevalence of **1.99% in those aged 50 and over**, with **cataract responsible for about 66%** and **93% of blindness avoidable**. The **National Programme for Control of Blindness and Visual Impairment (NPCBVI)** targets a blindness prevalence of 0.25% and provides free cataract surgery with intraocular lens; **Vision Centres under Ayushman Bharat Health and Wellness Centres** extend refraction services; and the **NPHCE** mandates vision screening for older persons. **LASI Wave 1** documented a very high burden of self-reported visual difficulty and unmet cataract need in Indian elders.

Current Practice Update

Textbook Position	Current Evidence-Based Practice (2023–2026)
Visual impairment causes functional limitation, isolation and depression	Add: **untreated vision loss is now a *Lancet* Commission (2024) modifiable dementia risk factor**; cataract surgery is associated with reduced dementia incidence
Glaucoma treated medically first; laser or surgery only on progression	**LiGHT (10-year, 2024): selective laser trabeculoplasty as first-line** — better disease control, more cost-effective, fewer glaucoma surgeries than drops
Insufficient evidence for VEGF antagonists in diabetic retinopathy	**Anti-VEGF therapy is now first-line for centre-involving diabetic macular oedema**, superior to laser (Protocol T and successors)
Screen for visual dysfunction in all older adults	Still recommended by AAO and geriatric societies, but the **USPSTF (2022) found insufficient evidence** for routine primary-care screening — present both views
Cataract surgery reduces hip fracture risk	**Confirmed and extended** — also associated with reduced motor-vehicle collisions, less depression and lower dementia incidence

Extra Points to Remember — recurring topic

- **Four causes of blindness in the elderly — "CGMD": C**ataract, **G**laucoma, **M**acular degeneration, **D**iabetic retinopathy. Quote the Framingham prevalences: cataract 46%, AMD 28%, open-angle glaucoma 7.2%, diabetic retinopathy 7% (all at ages 75–85).
- **The one-line pattern discriminator:** "Cataract = glare and gradual blurring; glaucoma = **peripheral** field loss; AMD = **central** loss with metamorphopsia; diabetic retinopathy = macular oedema, haemorrhage or ischaemia."
- **The pinhole test** is the fastest bedside discriminator — improvement means refractive error, no improvement means organic disease.
- **Never miss giant cell arteritis** — sudden painless monocular loss with headache, jaw claudication and scalp tenderness needs immediate high-dose steroid before biopsy.
- **Charles Bonnet syndrome** — formed hallucinations *with insight* in the visually impaired. Reassure; do not prescribe antipsychotics.
- **Systemic toxicity of eye drops** is a classic geriatric viva question — timolol drops causing bradycardia, bronchospasm and falls.
- **Indian data to quote:** blindness prevalence 1.99% in those ≥50 years, cataract responsible for 66%, **93% of blindness in India is avoidable**; NPCBVI target 0.25%.
- **The examiner's favourite closing line:** "Vision loss is one of the few dementia risk factors that is **cheaply and completely reversible** — a pair of spectacles or a cataract operation."

Examiner's Tip — say these lines verbatim:

- "**As many as 75% of older adults have visual or auditory dysfunction that is not reported to their physician** — hence screening is essential."
- "Screening comprises **visual acuity, ophthalmoscopy, tonometry and visual fields**."
- "**Presbyopia arises from nuclear sclerosis of the lens and atrophy of the ciliary muscle**."
- "**Visual impairment is a risk factor for falls and fractures, and cataract surgery lowers the risk of hip fracture.**"
- "**Refer all patients with age-related macular degeneration for visual rehabilitation.**"
- "Ophthalmic solutions are **significantly absorbed systemically** — use the minimum effective dose and monitor for toxicity."
- "The ***Lancet* Commission 2024 added untreated vision loss as a modifiable dementia risk factor**."

12. Macular Degeneration — Presentation, Aetiology, Diagnosis, Treatment and Prevention

Definition and anatomical basis

1. **Age-related macular degeneration (AMD)** is degeneration of the macula — the area of the retina lying at the **posterior pole of the globe**, which is the **site of highest visual acuity** — producing progressive loss of **central** vision with preservation of peripheral vision.
2. **The macula depends entirely on the choriocapillaris for nutrition.** Any disturbance in the vessel wall of the choroidal capillaries, in the **permeability or thickness of Bruch's membrane**, or in the **retinal pigment epithelium**, interferes with the exchange of nutrients and oxygen from the choroidal blood to the central retina. Such disturbances occur frequently in older patients.
3. **AMD is one of the most frequent causes of visual loss in older adults and is the commonest cause of legal blindness (20/200 or worse)**; Framingham Eye Study prevalence was **28% at ages 75–85, with a higher rate in women than in men**.

Aetiology and risk factors

4. Modifiable and non-modifiable risks: **older age, family history of the disorder, cigarette smoking, low dietary intake or low plasma concentrations of antioxidant vitamins and zinc, and white race (for "wet" lesions)**. **High lifetime exposure to sunlight** is also a risk factor, as are obesity and hypertension.
5. **AMD is caused by the interplay of genetic predisposition and exposure to modifiable risk factors.** Two susceptibility genes have been identified — **CFH (complement factor H)** and **LOC387715/ARMS2**.
6. **Carrying the susceptibility allele of either gene increases risk three- to eightfold; having two copies of the susceptibility alleles in both genes increases the risk 50-fold.** The combined effect of these polymorphisms carries an **attributable risk of 60%**. **Cigarette smoking and obesity multiply the risks associated with these variants.**
7. The central role of **complement factor H** identifies **complement dysregulation and chronic low-grade inflammation** as the pathogenic core — the rationale for modern complement-inhibitor therapy.

Types and presentation

8. **Dry (non-exudative, atrophic) AMD — 85–90% of cases.** Early and intermediate stages show **drusen** and pigmentary change. In the **geographic atrophy** form there are **areas of depigmentation alternating with zones of hyperpigmentation, caused mainly by changes in the retinal pigment epithelium**; in another form the degeneration involves **Bruch's membrane**, producing pigmentation of **well-circumscribed, roundish yellow areas**. **As lesions coalesce and involve the central macula, there is progressive worsening of vision to legal blindness.**
9. **Wet (exudative, neovascular) AMD — 10–15% of cases**, but responsible for most severe visual loss. There is **acute exudative pathology as a consequence of neovascularisation** — choroidal neovascular membranes breaching Bruch's membrane, with subretinal fluid, haemorrhage, lipid exudate and eventual disciform scarring.
10. **Symptoms**: gradual (dry) or rapid (wet) **painless loss of central vision**; **metamorphopsia** — straight lines appearing wavy or bent, the earliest and most characteristic symptom of wet AMD; a central scotoma; difficulty reading and recognising faces; reduced contrast sensitivity; need for brighter light; and micropsia. **Peripheral vision and navigational vision are preserved — patients never become totally blind from AMD.**
11. **Ophthalmoscopic findings vary and do not always parallel the loss of vision** — a key clinical caution.

Diagnosis

12. Visual acuity (distance and near) and an **Amsler grid** for metamorphopsia and central scotoma — also used for home self-monitoring with instructions to report new distortion urgently.
13. **Dilated fundoscopy and slit-lamp biomicroscopy**: hard and soft drusen, RPE mottling and clumping, geographic atrophy, subretinal fluid, haemorrhage, hard exudate, and disciform scar.
14. **Optical coherence tomography (OCT)** is the current standard for detecting intraretinal and subretinal fluid, pigment epithelial detachment, and drusen volume — it guides initiation, retreatment and extension of anti-VEGF therapy.
15. **Fundus fluorescein angiography** defines classic and occult choroidal neovascular membranes; **indocyanine green angiography** is required to diagnose **polypoidal choroidal vasculopathy**, which is far commoner in Indian and other Asian eyes.
16. **OCT angiography** gives non-invasive detection of neovascularisation including subclinical (non-exudative) CNV; **fundus autofluorescence** is used to measure and monitor geographic atrophy lesion area.

Treatment

17. **Dry/intermediate AMD — AREDS2 antioxidant supplementation**: vitamin C, vitamin E, zinc, copper, plus **lutein and zeaxanthin** (which replaced β-carotene because of increased lung cancer risk in smokers). It reduces progression to advanced AMD by roughly **25% over 5 years** in patients with intermediate AMD or with advanced AMD in one eye. It does **not** prevent AMD in those who do not have it, and Cochrane reviews find no benefit for cataract prevention.

18. **Wet AMD — intravitreal anti-VEGF injection is the standard of care**: ranibizumab, **bevacizumab** (off-label but far cheaper; the CATT trial showed it equivalent to ranibizumab), aflibercept, brolucizumab and **faricimab**. Typically three monthly loading doses followed by a **treat-and-extend** or pro-re-nata regimen guided by OCT.
19. Older modalities — **photodynamic therapy with verteporfin** and thermal laser photocoagulation — are now largely superseded, retained for polypoidal choroidal vasculopathy and selected extrafoveal lesions.
20. **Visual rehabilitation is an important part of therapy for AMD. Patients may benefit from reading glasses or magnification devices — refer all patients with AMD for visual rehabilitation.** Add high-intensity task lighting without glare, large print, eccentric viewing training, high-contrast markings and home hazard modification.
21. **Clinical depression may occur as a consequence of reductions in quality of life and should be treated.** Screen actively; also recognise Charles Bonnet syndrome.

Prevention

22. **Smoking cessation is the single most important modifiable intervention** — smoking both causes AMD and multiplies genetic risk.
23. **Sunlight protection** with ultraviolet-blocking sunglasses and a wide-brimmed hat, given high lifetime sunlight exposure as a risk factor.
24. **Diet** rich in dark green leafy vegetables (lutein and zeaxanthin) and oily fish (omega-3), with control of obesity, hypertension and dyslipidaemia.
25. **Amsler grid self-monitoring** of the fellow eye with clear instructions to report new distortion immediately, plus regular dilated fundus examination — the second eye is at high risk once one eye is affected.
26. **Prognosis**: dry AMD progresses slowly over years; **untreated wet AMD causes severe central visual loss within weeks to months**, which makes urgent referral for anti-VEGF therapy essential.

Latest Updates (2023–Present)

- **The first-ever treatments for geographic atrophy were approved in 2023. Pegcetacoplan (Syfovre)**, a **C3 inhibitor**, was FDA-approved in February 2023 on the basis of **OAKS and DERBY (*Lancet* 2023)** — in OAKS at 12 months monthly dosing slowed GA lesion growth by **21%** and every-other-month dosing by **16%**. **Avacincaptad pegol (Izervay)**, a pegylated RNA aptamer inhibiting **C5**, was FDA-approved in August 2023 on **GATHER1 and GATHER2 (*Lancet* 2023)**, with **27% and 14%** reductions in GA growth at 12 months.
- **The critical caveat, and the most examinable nuance:** these agents slow **anatomical** lesion growth but have **not demonstrated a clinically meaningful functional benefit**. GATHER2 two-year data (*Ophthalmology* 2025) showed persistent loss of ≥15 ETDRS letters in **18% with avacincaptad versus 19% with sham (HR 0.90, p = 0.64)**. **The EMA's CHMP issued a negative opinion and both manufacturers withdrew their European marketing applications — as of 2025 there is no approved treatment for geographic atrophy in Europe.** Both carry a risk of conversion to exudative AMD, and pegcetacoplan carries a risk of retinal and occlusive retinal vasculitis.
- **Faricimab (Vabysmo)** — a bispecific antibody targeting **VEGF-A and angiopoietin-2**; TENAYA and LUCERNE showed non-inferiority to aflibercept with dosing intervals extended to **16 weeks**, and 2024–2025 real-world data confirm reduced injection burden.
- **Aflibercept 8 mg (Eylea HD)** — FDA-approved 2023 and EU 2024; the PULSAR trial showed **12- to 16-week dosing** non-inferior to 2 mg every 8 weeks.
- **Ranibizumab port delivery system (Susvimo)** — a refillable surgical ocular implant giving continuous delivery with refills every 24 weeks; reintroduced in 2024 after a 2022 recall.
- **Gene therapy** — ixoberogene soroparvovec (Ixo-vec) and RGX-314 are in phase 3, aiming for sustained intraocular anti-VEGF expression from a single administration. **Home OCT monitoring** (Notal Vision Home OCT, FDA-cleared 2024) enables patient-performed weekly scans for fluid detection.
- **AREDS2 10-year follow-up (*JAMA Ophthalmology* 2022)** confirmed **lutein/zeaxanthin is superior to β-carotene** for reducing progression, with no excess lung cancer — consolidating the current formulation.
- **Indian context**: population-based studies (INDEYE, CIEMS, APEDS) report AMD prevalence of roughly **2–3% in those aged 60 and over**, lower than Western figures but rising. **Polypoidal choroidal vasculopathy is substantially commoner in Indian eyes** and must be excluded with ICG angiography in wet AMD that responds poorly to anti-VEGF. **Bevacizumab and the Indian-developed ranibizumab biosimilar (Razumab, the world's first) are the mainstay because of cost**; aflibercept biosimilars became available in 2024–2025. AMD care falls under the NPCBVI, and low-vision aids are supplied through its low-vision programme.

Current Practice Update

Textbook Position (Essentials 7th ed.)	Current Evidence-Based Practice (2023–2026)
No treatment exists for the atrophic (dry/geographic atrophy) form beyond antioxidant supplementation	**Two complement inhibitors approved in the US in 2023** — pegcetacoplan (C3) and avacincaptad pegol (C5) — slowing lesion growth by 14–27%. **But no proven visual-function benefit, and both were rejected in Europe**
Ranibizumab and bevacizumab for neovascular AMD, given monthly	**Faricimab and aflibercept 8 mg allow 12–16-week intervals**; treat-and-extend guided by OCT is standard; **biosimilars have transformed affordability in India**
Antioxidant formulation includes β-carotene	**AREDS2 formulation replaces β-carotene with lutein and zeaxanthin** — more effective and safe in smokers
Diagnosis by ophthalmoscopy and fluorescein angiography	**OCT is the standard** for diagnosis and monitoring; OCT-angiography detects subclinical CNV; **home OCT** FDA-cleared 2024
Genetics: CFH and LOC387715 confer risk	Confirmed, and now **therapeutically actionable** — complement dysregulation is the target of the new geographic atrophy drugs
Refer all patients for visual rehabilitation; treat depression	**Unchanged and still under-performed in practice** — state it explicitly in the answer

Examiner's Tip — say these lines verbatim:

- "**The macula depends entirely on the choriocapillaris for nutrition**; AMD results from disturbance of the choroidal capillaries, Bruch's membrane or the retinal pigment epithelium."
- "AMD is the **commonest cause of legal blindness (20/200 or worse)** in older adults; prevalence 28% at ages 75–85, higher in women."
- "**CFH and LOC387715/ARMS2** — one susceptibility allele raises risk 3–8 fold, two copies of both raise it **50-fold**, with a **60% attributable risk**; **smoking and obesity multiply these genetic risks**."
- "**Metamorphopsia is the earliest symptom of wet AMD** — the Amsler grid is the bedside test."
- "**Ophthalmoscopic findings vary and do not always parallel the loss of vision.**"
- "**Peripheral vision is always preserved — patients never become totally blind from AMD.**"
- "**Smoking cessation is the single most important preventive measure**, and **AREDS2 supplementation reduces progression by about 25% over 5 years** in intermediate disease."
- "**Refer all patients with AMD for visual rehabilitation, and treat the depression that commonly follows.**"
- "The 2023 complement inhibitors **slow lesion growth but have not shown visual benefit**, and were **rejected by the EMA**."

13. Heat Stroke

Definition and epidemiology

1. **Heat stroke is defined as a failure to maintain body temperature, and is characterised by a core temperature of >40.6°C (>105°F), severe central nervous system dysfunction (psychosis, delirium, coma), and anhidrosis (hot, dry skin).** These are the **three defining criteria**.
2. **The two groups primarily affected are older adults who are chronically ill (classic or non-exertional heat stroke) and the young undergoing strenuous exercise (exertional heat stroke).**
3. **Mortality is as high as 80% once this syndrome is manifest.**
4. **Hypo- and hyperthermic states are predominantly disorders of older adults.** Morbidity and mortality increase during particularly hot or cold periods, especially among the ill elderly, and much of this illness is caused by an increased incidence of **cardiovascular disorders (myocardial infarction and stroke) or infectious disease (pneumonia)** during these periods.

Pathophysiology — why older people are vulnerable

5. The major pathophysiological mechanisms of temperature dysregulation in ageing are **impaired temperature perception** and **diminished sweating in hyperthermia**.
6. **Dermal vascularisation and the number of sweat glands both decrease with age**, along with sebum production. **Since dermal vascularisation and sweating are the major mechanisms for balancing body temperature and losing heat from the skin, and both are impaired with age, elderly people tend to experience hyperthermia in hot weather.**
7. **There are multiple predisposing factors for heat stroke in older adults, but most often there is a prolonged heat wave.** Others: cardiovascular disease, diabetes, dementia, immobility, social isolation, living alone without cooling, obesity, dehydration and blunted thirst.
8. **Drugs**: anticholinergics, antihistamines, tricyclic antidepressants and antipsychotics (impair sweating); diuretics (volume depletion); β-blockers (limit the cardiac output response); alcohol.
9. Mechanism of injury: heat produces **direct cytotoxic injury plus a systemic inflammatory response with cytokine release**, causing endothelial damage, coagulopathy and multi-organ failure — the syndrome behaves like sepsis.

Clinical features

10. **The diagnosis requires a high level of suspicion**, because the **early manifestations are non-specific**.
11. **Early signs (Table 12-14)**: **dizziness, weakness, sensation of warmth, anorexia, nausea, vomiting, headache and dyspnoea.**
12. **Later signs**: **central nervous system dysfunction, psychosis, delirium, coma, anhidrosis, and hot dry skin.**
13. Differential diagnosis: sepsis, thyroid storm, neuroleptic malignant syndrome, serotonin syndrome, malignant hyperthermia, anticholinergic toxidrome, meningitis and status epilepticus. In older people the presentation overlaps heavily with delirium and infection.

Complications (Table 12-15)

14. **Cardiac**: myocardial damage, congestive heart failure, arrhythmias. **Renal**: renal failure in **20–25%**.
15. **Neurological**: cerebral oedema, seizures, diffuse and focal findings. **Hepatic**: hepatocellular necrosis, jaundice, liver failure.
16. **Muscle**: rhabdomyolysis with myoglobinuria. **Haematological**: bleeding diathesis, disseminated intravascular coagulation.
17. **Metabolic**: electrolyte disturbance, acid–base disturbance (metabolic acidosis, respiratory alkalosis). **Infective**: aspiration pneumonia, sepsis. **Circulatory**: dehydration and shock.
18. Investigations: **rectal core temperature** (most accurate), full blood count, electrolytes, urea and creatinine, liver function, creatine kinase, coagulation screen, arterial blood gases, urinary myoglobin, ECG, chest radiograph, blood cultures, and CT head if there are focal signs.

Management

19. **Once the full syndrome has developed for any length of time the prognosis is very poor.** Management at this stage requires intense multisystem care, but **the key is rapid specific therapy consisting of cooling to 38.9°C (102°F) within the first hour.**
20. **Ice packs and ice-water immersion are superior to convection cooling with alcohol sponge baths or electric fans.** This is the single most important therapeutic statement in the chapter.
21. **ABC**: airway protection if comatose, high-flow oxygen, large-bore intravenous access, continuous cardiac monitoring, and careful fluid resuscitation guided by haemodynamics — older patients are at risk of precipitating pulmonary oedema.
22. Adjunctive measures: remove clothing, cold intravenous fluids, cold gastric or bladder lavage, benzodiazepines to control shivering and seizures, fluids and urinary alkalinisation for rhabdomyolysis, and correction of electrolyte disturbance and coagulopathy.

23. **Do not give antipyretics** (paracetamol or NSAIDs) — the hypothalamic set point is normal, so they are ineffective and may worsen hepatic and renal injury. **Do not give dantrolene** — it has no role in heat stroke, unlike malignant hyperthermia.

Prevention

24. **In view of the poor survival, efforts must be directed toward prevention. Prevention appears to be the most appropriate approach to management of temperature dysregulation in older adults.**
25. **Older patients should be cautioned about the dangers of hot weather. For those at particularly high risk, temporary relocation to more protected environments should be considered.**
26. **Education of older adults about their susceptibility to hypo- and hyperthermia in extremes of environmental temperature, education as to appropriate behaviour in such conditions, and close monitoring of the most vulnerable older adults should help reduce morbidity and mortality.**
27. Practical measures: remain indoors and in shade between noon and 3 pm; **drink fluids on a schedule rather than waiting for thirst**, since thirst is blunted with age; light, loose cotton clothing; cool showers; avoid alcohol, caffeine and heavy meals; pre-summer medication review; buddy or check-in systems for those living alone; and access to cooling centres.

Latest Updates (2023–Present)

- **Society of Critical Care Medicine Guidelines for the Treatment of Heat Stroke (*Critical Care Medicine*, February 2025)** — the first dedicated critical care guideline. **Active cooling is recommended over passive cooling, and cold- or ice-water immersion achieves the fastest cooling rate and should be prioritised where available.** The target is a core temperature **below 39°C within 30 minutes**. Where immersion is not feasible, select the method or combination achieving the fastest cooling rate — evaporative cooling with misting and fanning (well tolerated in classic heat stroke), ice packs to neck, axillae and groin, cooling blankets, and cold intravenous fluids. The governing principle is **"cool first, transport second."** Antipyretics and dantrolene are explicitly not recommended.
- Contemporary outcome data: **mortality approaches 60% in ICU patients, and approximately 30% of survivors have long-term cognitive or motor dysfunction** — the central nervous system is particularly vulnerable, with cell death increasing exponentially with the duration of temperature exposure.
- **India — National Programme on Climate Change and Human Health (NPCCHH), NCDC/MoHFW**: the **National Action Plan on Heat Related Illnesses** (2021, revised subsequently) and the **"Emergency Cooling for Severe Heat-Related Illnesses" guidance (March 2024)**. Key positions: **conductive cooling with ice or cold water immersion is the gold standard**; where a tub is unavailable a **tarpaulin-based portable cold-water immersion method** can be improvised; and **cooling should not be delayed in order to remove all clothing or equipment**. Standard case definitions and first aid for heat rash, heat cramps, heat syncope, heat exhaustion and heat stroke are provided.
- **MoHFW heatwave advisory (2025)**: every health facility must have a designated cooling area, ice packs and a heat stroke protocol; medical officers must submit **patient-level data daily on the IHIP (Integrated Health Information Platform) portal** using standardised checklist criteria to classify heat stroke and heat-related illness deaths.
- **Global burden**: approximately **61,672 heat-related deaths occurred in Europe in the summer of 2022**; 2023 saw a substantial global increase in heat-related illness; India recorded severe heatwaves in 2024 and 2025 with several hundred confirmed heat-stroke deaths. The *Lancet* Countdown reports that **heat-related mortality in adults over 65 has risen by roughly 85% since the 1990s**. Older adults, outdoor workers and the urban poor are disproportionately affected, and South Asian populations with inadequate access to cooling, healthcare and safe water are at particularly high risk.
- Under investigation: extracorporeal and endovascular cooling catheters, and biomarkers (heat shock proteins, IL-6) for prognostication.

Current Practice Update

Textbook Position (Essentials 7th ed.)	Current Evidence-Based Practice (2023–2026)
Cool to **38.9°C within the first hour**	**SCCM 2025 tightens this to <39°C within 30 minutes** — "cool first, transport second"
Ice packs and ice-water immersion superior to alcohol sponging and fans	**Confirmed and formalised**: cold/ice-water immersion (≤12°C) is the gold standard; evaporative cooling with misting and fanning is the preferred alternative in classic heat stroke
Mortality "as high as 80% once the syndrome is manifest"	Contemporary ICU mortality is around **60%**, with **~30% of survivors left with cognitive or motor dysfunction**
Prevention through education, caution in hot weather, relocation of the high-risk	**Institutionalised in India** through NPCCHH Heat Action Plans, facility-level cooling protocols and mandatory **IHIP daily reporting**
Antipyretics and dantrolene not specifically addressed	**Explicitly not recommended** in current guidelines — ineffective and potentially harmful

> **Examiner's Tip — say these lines verbatim:**
>
> - "Heat stroke is defined by the **triad of core temperature >40.6°C, severe CNS dysfunction, and anhidrosis with hot**

dry skin."

- "The two affected groups are **chronically ill older adults (classic) and young people exercising strenuously (exertional)**; **mortality is as high as 80%**."
- "The mechanisms in ageing are **impaired temperature perception and diminished sweating** — dermal vascularisation and sweat gland numbers both fall with age."
- "**The key is rapid cooling to 38.9°C within the first hour; ice packs and ice-water immersion are superior to alcohol sponge baths and electric fans.**"
- "**Do not give antipyretics or dantrolene** — the hypothalamic set point is normal."
- "Renal failure occurs in **20–25%**; also expect rhabdomyolysis, DIC, hepatic necrosis and cerebral oedema."
- "**Prevention is the most appropriate approach to management** — caution about hot weather and temporary relocation of the highest-risk older adults."
- "**SCCM 2025: cool to below 39°C within 30 minutes — cool first, transport second.**"

14. Heat Exhaustion

Definition and place in the spectrum

1. **Heat exhaustion** is a moderate heat-related illness caused by **water and/or salt depletion** during heat exposure, characterised by a core temperature that is **normal or only mildly elevated (usually below 40°C)**, with **preserved sweating** and an **intact mental state**.
2. It occupies the stage immediately before heat stroke on the continuum of heat-related illness: **heat rash (miliaria) → heat oedema → heat cramps → heat syncope → heat exhaustion → heat stroke**. **Untreated, heat exhaustion progresses to heat stroke, which carries up to 80% mortality** — this is why recognising it matters.
3. **Early manifestations of heat exhaustion are non-specific**; later, severe central nervous system dysfunction and anhidrosis develop, which signifies progression to heat stroke.

Pathophysiology

4. Prolonged heat exposure causes sustained **cutaneous vasodilatation and profuse sweating**, producing loss of water and sodium, **hypovolaemia and reduced cardiac output**, and hence inadequate tissue perfusion. Crucially, **thermoregulation remains intact** — which is why sweating continues and consciousness is preserved.
5. Two overlapping types: **water-depletion heat exhaustion** (inadequate fluid intake; hypernatraemia; rapid onset — the dominant form in the elderly, in whom thirst is blunted) and **salt-depletion heat exhaustion** (losses replaced with water alone; hyponatraemia; prominent cramps; more insidious onset).
6. **Why older adults are vulnerable**: impaired temperature perception, diminished sweating from reduced sweat gland numbers and dermal vascularisation, blunted thirst sensation, reduced renal concentrating ability, lower total body water, diuretic and anticholinergic use, immobility and dependence on carers for fluids, and dementia limiting self-protective behaviour.

Clinical features

7. **Early/non-specific symptoms (Table 12-14)**: **dizziness, weakness, sensation of warmth, anorexia, nausea, vomiting, headache and dyspnoea.**
8. Additional features: **profuse sweating with cool clammy skin**, intense thirst, fatigue, myalgia and muscle cramps, irritability, tachycardia, **postural hypotension and near-syncope**, and oliguria with concentrated urine.
9. **The cardinal distinction from heat stroke**: in heat exhaustion the core temperature is **below 40°C**, the **skin is moist because sweating is preserved**, and there is **no severe CNS dysfunction** — no psychosis, delirium, coma or seizures. Mild irritability or feeling faint is acceptable; **any frank alteration of sensorium means heat stroke until proved otherwise**.
10. In older people the picture is easily mistaken for gastroenteritis, urinary sepsis, drug adverse effects or delirium — **a high index of suspicion during a heatwave is essential**, exactly as for heat stroke.

Investigations

11. **Rectal core temperature** (essential to separate exhaustion from stroke), capillary glucose, serum electrolytes (**sodium may be high or low depending on the type**), urea and creatinine (pre-renal pattern), creatine kinase (to exclude rhabdomyolysis), urine specific gravity and ECG.
12. Investigate to exclude infection or another acute illness whenever the diagnosis is not clear-cut, since heat illness and sepsis coexist and mimic each other in the elderly.

Management

13. **Move the patient immediately to a cool, shaded or air-conditioned environment** and lay them supine with the legs elevated; remove excess clothing.
14. **Active external cooling** — cool water spray with fanning, cool wet cloths, and ice packs to the neck, axillae and groin. Cooling is less aggressive than for heat stroke but should still be prompt.
15. **Oral rehydration with cool electrolyte-containing fluid (ORS)** if the patient is alert and not vomiting. **Intravenous isotonic saline** if there is vomiting, marked postural hypotension or failure to improve; in older patients infuse cautiously with monitoring for fluid overload and heart failure.
16. Correct electrolyte abnormalities; **correct hyponatraemia slowly** to avoid osmotic demyelination. Rest and avoid further heat exposure for 24–48 hours.
17. **Do not give antipyretics** — the hypothalamic set point is normal, so they are ineffective.
18. **Monitor core temperature, blood pressure, urine output and mental status. Any deterioration in mental status, or a core temperature reaching 40°C, means the patient has progressed to heat stroke — begin aggressive cooling immediately.**
19. **Indications for admission in older adults**: advanced age with comorbidity, persistent symptoms after 30–60 minutes of cooling and rehydration, significant electrolyte derangement, renal impairment, rhabdomyolysis, inability to take oral fluids, and an unsafe or unairconditioned home environment.
20. **Prognosis is excellent with prompt treatment** — heat exhaustion resolves fully without sequelae. Its clinical importance lies entirely in being the **recognisable and completely reversible stage** before the very high mortality of heat stroke.

Prevention

21. Identical to heat stroke prevention: **education of older adults about their susceptibility and about appropriate behaviour in extremes of environmental temperature, and close monitoring of the most vulnerable**.
22. Practical measures: pre-summer medication review (diuretics, anticholinergics, antipsychotics, β-blockers); **scheduled fluid intake rather than thirst-driven drinking**; avoid outdoor activity between noon and 3 pm; light loose clothing; cool showers; use of cooling centres; buddy check-in systems; and **temporary relocation of very high-risk older people to a protected environment**.

Latest Updates (2023–Present)

- **SCCM Heat Stroke Guidelines (*Critical Care Medicine*, 2025)** emphasise that the distinction between heat exhaustion and heat stroke rests on **core temperature and CNS dysfunction**, and that **where there is any doubt the patient should be treated as heat stroke and cooled** — the risk of over-treating exhaustion is trivial compared with the risk of under-treating stroke.
- **India — NCDC/NPCCHH "National Action Plan on Heat Related Illnesses"** provides **standard case definitions, symptoms and first aid for heat rash, heat cramps, heat syncope, heat exhaustion and heat stroke** (Annexure 2), and mandates **daily reporting of heat-related illness and deaths on the IHIP portal**. The **March 2024 "Emergency Cooling for Severe Heat-Related Illnesses"** document supplements this for severe cases, and the NPCCHH Programme Implementation Plan funds procurement of emergency cooling equipment.
- **MoHFW 2025 heatwave advisory** requires facility preparedness — a designated cooling area, ice packs and a written heat illness protocol at every health facility.
- **Oral rehydration solution is superior to plain water** in salt-depletion heat exhaustion (2024–2025 occupational and sports medicine literature); ice-slurry or "slushy" ingestion provides useful internal cooling and is being adopted for outdoor workers.
- **WHO–WMO heat and health guidance (2024)** and India's state and city **Heat Action Plans** (with the Ahmedabad Heat Action Plan as the internationally cited model) identify **older adults as a priority vulnerable group**, with early warning systems, cooling shelters and targeted outreach.
- ***Lancet* Countdown (2025)**: heat exposure days and heat-attributable mortality in adults over 65 continue to rise in India. **ICMR-NIN** guidance advises older adults to take roughly **2–2.5 litres of fluid daily in summer** unless fluid-restricted for heart failure or chronic kidney disease.

Current Practice Update

Textbook Position (Essentials 7th ed.)	Current Evidence-Based Practice (2023–2026)
Heat exhaustion described only as the "early manifestations of hyperthermia", without a separate management protocol	**Now a distinct entity with its own case definition and first-aid protocol** under India's NPCCHH National Action Plan on Heat Related Illnesses
Distinction from heat stroke implied by the early/late signs table	**Explicit criteria**: core temperature <40°C, preserved sweating, intact sensorium. **When in doubt, treat as heat stroke and cool** (SCCM 2025)
Rehydration not specified in detail	**Oral rehydration solution preferred over plain water** in salt depletion; IV isotonic saline with cautious monitoring for fluid overload in older adults
Prevention through education and relocation of the high-risk	**Operationalised** through state and city **Heat Action Plans**, early warning systems, cooling shelters and mandatory IHIP reporting

Examiner's Tip — say these lines verbatim:

- "Heat exhaustion is **water and/or salt depletion during heat exposure, with a core temperature below 40°C, preserved sweating and an intact sensorium**."
- "**The early manifestations of heat exhaustion are non-specific** — dizziness, weakness, a sensation of warmth, anorexia, nausea, vomiting, headache and dyspnoea."
- "**The single discriminator from heat stroke is CNS dysfunction with anhidrosis** — if the sensorium is altered and the skin is hot and dry, it is heat stroke."
- "Two types: **water-depletion (hypernatraemic, rapid, commoner in the elderly because thirst is blunted)** and **salt-depletion (hyponatraemic, insidious, prominent cramps)**."
- "Treatment is **cool environment, supine with legs elevated, active external cooling, and oral rehydration solution or cautious isotonic saline**."
- "**Do not give antipyretics** — the hypothalamic set point is normal."
- "**Heat exhaustion is the completely reversible stage before heat stroke**, which carries up to 80% mortality — recognising it is the whole point."
- "**Prevention is the most appropriate approach to management of temperature dysregulation in older adults.**"

Rapid Revision Grid — Night Before the Examination

Topic	One-line textbook anchor	Single most examinable 2023–2026 update
Epilepsy in the elderly	3rd commonest neurological disorder; stroke causes 40–53%; treat after first unprovoked seizure	Choose ASM by **tolerability, not efficacy**; no prophylaxis after stroke; lacosamide/lamotrigine/levetiracetam
Coma	Arousal (ARAS) + awareness (cortex); GCS ≤8; preserved pupils = metabolic	**Cognitive motor dissociation in ~25%** (*NEJM* 2024); FOUR score; cEEG for NCSE
Subdural haematoma	Acute = mass effect; chronic = inflammation from blood degradation products; subdural drain lowers recurrence	**MMA embolisation** — EMBOLISE and STEM positive, MAGIC-MT neutral; **dexamethasone harmful (DEX-CSDH)**
Lacunar infarct	<15 mm deep infarct; microatheroma or lipohyalinosis; **no cortical signs, no field defect**	**LACI-2 (2023)**: isosorbide mononitrate reduces recurrence and cognitive impairment; cilostazol reduces dependence
SACD	Posterior columns + lateral corticospinal tracts; brisk knees, absent ankles, extensor plantars	**NICE NG239 (2024)**: IM hydroxocobalamin immediately in neurological disease; nitrous oxide SACD
Motor neuron disease	Mixed UMN + LMN in one region; eyes, sphincters and sensation spared; riluzole + NIV prolong survival	**Tofersen approved** for SOD1-ALS; **AMX0035 withdrawn (2024)**; Gold Coast criteria
Peripheral neuropathy	11% over 55; diabetes 44%; NCS/EMG is the initial study in unexplained cases	**Test for hATTR** in progressive "idiopathic" neuropathy; screen for DPN at diagnosis of T2DM
Neuritic pain	Lesion or disease of the somatosensory nervous system; NNT 3.6 TCA, 7.7 pregabalin	**Suzetrigine ($Na_v1.8$ inhibitor), FDA-approved January 2025**; OPTION-DM supports combination
Carotid stenosis	NASCET method; image within 24 h; CEA within 7 days for symptomatic 50–99%	**CREST-2 (*NEJM* 2025)**: in asymptomatic ≥70%, stenting helped, **CEA did not**
Bedside swallow	78% dysphagic after stroke; gag reflex unreliable; water → purée → solids; watch for wet gurgly voice	**Screen within 4 hours**, before any oral intake; use **GUSS or Yale**; oral care not antibiotics
Visual impairment	75% unreported; cataract, glaucoma, AMD, diabetic retinopathy; cataract surgery lowers hip fracture risk	***Lancet* Commission 2024: vision loss is a modifiable dementia risk factor**; LiGHT — SLT first-line for glaucoma
Macular degeneration	Macula depends on choriocapillaris; CFH + LOC387715 give 50-fold risk; metamorphopsia; refer for rehabilitation	**Pegcetacoplan and avacincaptad** approved 2023 for GA — but **no functional benefit and rejected by EMA**
Heat stroke	>40.6°C + CNS dysfunction + anhidrosis; mortality up to 80%; cool to 38.9°C within 1 hour	**SCCM 2025: cool to <39°C within 30 minutes; "cool first, transport second"**; India NPCCHH/IHIP reporting
Heat exhaustion	<40°C, sweating preserved, sensorium intact; the reversible stage before heat stroke	Distinct case definition and first aid under India's **National Action Plan on Heat Related Illnesses**

Prepared from Pathy's Principles and Practice of Geriatric Medicine (6th edition) and Essentials of Clinical Geriatrics (7th edition) as primary sources, supplemented with peer-reviewed literature and guidelines published between 2023 and 2026. Where the textbook position and current evidence differ, the difference is set out explicitly in the Current Practice Update table for that question.

MD GERIATRIC MEDICINE — EXAM REVISION

Genitourinary System Continence, Prostate & Urinary Infection

High-Yield Structured Answers · 15 Questions

Prepared for: Dr. Sudarsan · MD Geriatric Medicine

Module: Urinary Incontinence · LUTS/BPH · Catheters · UTI

Format: University 15-mark & 7-mark answer patterns

Compiled: August 2026

Core text: Pathy's Principles and Practice of Geriatric Medicine (6th ed.) · Kane's Essentials of Clinical Geriatrics

Supplemented with guideline and trial evidence up to 2026 · Indian units and Indian programme context throughout

Contents

1	Classify urinary incontinence; evaluation and treatment **(RECURS)**	**15 marks**
2	Types, diagnosis and management of incontinence in the elderly, including reversible causes	**15 marks**
3	Case: 65-year-old woman with frequency, nocturia and leakage for 6 months	**15 marks**
4	Urinary incontinence in males — types, epidemiology, pathogenesis, risk factors, screening, evaluation, management	**15 marks**
5	Algorithm to investigate and treat an elderly male with lower urinary tract symptoms	**15 marks**
6	Medical management of benign prostatic hypertrophy	**7 marks**
7	Overactive bladder	**7 marks**
8	Bladder retraining protocol	**7 marks**
9	Behavioural intervention in urinary incontinence	**7 marks**
10	Uroflowmetry	**7 marks**
11	Prostate specific antigen	**7 marks**
12	Obstructive uropathy — clinical features, investigation and treatment	**15 marks**
13	Chronic indwelling catheter usage in the elderly	**7 marks**
14	Catheter-associated urinary tract infections — prevention and management	**7 marks**
15	Recommendations for treatment of complicated urinary tract infection in males	**7 marks**

Each answer is arranged as: high-yield numbered points → Latest Updates (2023–present) → Current Practice Update → Exam One-Liners. Recurring / high-yield topics additionally carry mnemonics, numerical anchors, examiner traps and a 15-second answer skeleton.

QUESTION 1

Classify urinary incontinence; discuss its evaluation and treatment. 15 MARKS RECURS

High-Yield Points

A. Definition and Burden

1. **Definition:** Urinary incontinence (UI) is the **involuntary loss of urine that is objectively demonstrable**, in an amount or frequency sufficient to be a social and/or health problem.
2. **Prevalence:** About **one in three women** and **15–20% of men** above 65 years are affected; rises to **60–80% in nursing homes**. In women with comorbidity, cognitive or physical impairment, or polypharmacy, prevalence may reach 70–80%.
3. **Ageing is not the cause.** Age-related changes only predispose: reduced bladder capacity, higher residual urine, more involuntary detrusor contractions, fall in urethral closure pressure (women), prostatic enlargement (men), and loss of the normal night-time rise in **vasopressin** with higher **ANP** causing **nocturnal polyuria**.
4. **Consequences:** skin breakdown, recurrent UTI, **falls and fractures** (especially nocturnal), isolation, depression, dependency, caregiver strain, and **institutionalisation**.

B. Classification

5. **Physiological classification (two basic faults):** (i) **failure to store** — overactive or poorly compliant bladder, or weak outlet; (ii) **failure to empty** — poorly contractile bladder, or increased outlet resistance.
6. **By time course: Acute (transient) incontinence** — sudden onset with an acute illness or drug, resolves when the cause is treated; **Persistent incontinence** — unrelated to acute illness, continues over time.
7. **Persistent UI has four basic clinical types** (they overlap in the same patient):

Type	Definition	Common causes
Stress	Small-volume leak on rise in intra-abdominal pressure — cough, laugh, exercise	Pelvic floor weakness with urethral hypermobility; bladder outlet or sphincter weakness (intrinsic sphincter deficiency); obesity, chronic cough, prior childbirth/ surgery, oestrogen lack
Urge	Larger-volume leak because voiding cannot be delayed once fullness is felt	**Detrusor overactivity**, alone or with local disease (stone, tumour, diverticulum, obstruction) or CNS disease (stroke, dementia, parkinsonism, cord injury)
Overflow (incomplete emptying)	Nonspecific symptoms; classically small-volume dribble from an over-distended bladder	Prostatic enlargement, urethral stricture, cystocele; acontractile bladder in diabetes or cord injury; detrusor–sphincter dyssynergia (multiple sclerosis, suprasacral cord lesion)
Functional	Cannot or will not reach the toilet in time despite an intact lower tract	Severe dementia, other neurological disease, depression/hostility, immobility, restraints, inaccessible toilet

8. **Mixed incontinence** (stress + urge) is the commonest pattern in older women. **Frail older patients** typically combine urge incontinence with functional disability.

9. **Special subtype — DHIC** (**detrusor hyperactivity with impaired contractility**): involuntary contractions but emptying of less than one-third of bladder volume; patients strain to void and are difficult to treat because of retention. **Very high-yield.**
10. **Overactive bladder (OAB)** is a symptom syndrome (urgency ± frequency >8/24 h ± nocturia), with or without incontinence; ~30% of women and ~40% of men aged ≥75 report these symptoms.

C. Evaluation

11. **Case-finding first** — patients rarely volunteer it. Ask: "Do you have trouble with your bladder?", "Do you ever lose urine when you don't want to?", "Do you wear padding in case you lose urine?"
12. **Basic evaluation in ALL patients (four components):** focused **history** (with bladder record), targeted **physical examination**, **urinalysis**, and **post-void residual (PVR)**.
13. **History:** onset and type, frequency/timing/volume, irritative vs voiding symptoms, fluid and caffeine pattern, obstetric/surgical/GU history, comorbidity (diabetes, heart failure, neurological disease), **drug review**, bowel habit, environment, and the **most bothersome symptom**.
14. **Bladder diary / voiding chart** for 3 days characterises the pattern and monitors response to treatment.
15. **Examination:** mobility and dexterity, cognition and mood, focal neurology and **sacral reflexes**, abdomen for distended bladder, rectal for impaction/tone/prostate, and pelvic examination for **atrophic vaginitis** and prolapse (cystocele grading).
16. **Urinalysis:** persistent microscopic haematuria (>5 RBC/hpf) without infection needs cytology and cystoscopy. Bacteriuria in a community patient with new or worsening incontinence should be treated before further evaluation; in nursing-home residents, eradicating bacteriuria does **not** improve chronic stable incontinence.
17. **PVR:** by bladder ultrasound or in–out catheter. **>200 mL is abnormal** and warrants further evaluation. PVR may be omitted in a carefully selected patient with pure stress or pure urgency symptoms, no voiding difficulty and no risk factor for retention.
18. **Uroflowmetry** is a useful non-invasive screen in older men: **peak flow <10 mL/s on a voided volume >150 mL** suggests obstruction or impaired contractility.
19. **Referral criteria** for urology/gynaecology/urodynamics: recent pelvic surgery or irradiation (<6 months), **≥2 symptomatic UTIs in 12 months**, risk factors for bladder cancer (new irritative symptoms, heavy smoking, aniline dye exposure), **marked pelvic prolapse**, significant retention, prostatic nodule, or failure of an adequate therapeutic trial.

D. Treatment

20. **Step 1 — correct reversible factors** (mnemonic **DRIP**: Delirium; Restricted mobility/ Retention; Infection, Inflammation, Impaction; Polyuria, Pharmaceuticals). Relief of faecal impaction alone may restore continence.
21. **Step 2 — behavioural therapy for everyone.** Patient-dependent: **pelvic floor (Kegel) exercises** — the single most effective component — bladder training, bladder retraining. Caregiver-dependent: scheduled toileting, habit training, **prompted voiding** (25–40% of nursing-home residents become dry by day).

22. **Step 3 — drugs, by type:**
    - **Urge/OAB:** antimuscarinics — darifenacin 7.5–15 mg, solifenacin 5–10 mg, tolterodine, fesoterodine, oxybutynin, trospium. Full effect takes **1–2 months**; warn about dry mouth, constipation, glaucoma, reflux and **cognitive impairment/delirium**.
    - **Men with BPH-related irritative symptoms:** α-blocker (tamsulosin 0.4 mg, alfuzosin 10 mg, silodosin 4 mg) ± antimuscarinic; add a 5-α-reductase inhibitor if prostate >30 g.
    - **Stress:** no drug is licensed; α-agonist (pseudoephedrine) ± topical oestrogen, or duloxetine, are off-label options.
    - **Atrophic vaginitis: topical vaginal oestrogen** only — **oral oestrogen is ineffective and may worsen incontinence.**
    - **Refractory nocturia:** low-dose desmopressin with **close sodium monitoring** — hyponatraemia risk is high in the elderly.
23. **Step 4 — devices and surgery:** pessary for prolapse; periurethral bulking or sling for intrinsic sphincter deficiency; colposuspension/autologous fascial sling for stress UI; TURP or other prostatic surgery for obstruction; **artificial urinary sphincter** for post-prostatectomy sphincter damage; intradetrusor **botulinum toxin** and **sacral neuromodulation (S3)** for refractory OAB.
24. **Catheters only for specific indications.** Condom catheter only for intractable incontinence in a dependent man without retention; intermittent catheterisation keeps volumes <400 mL; chronic indwelling catheters are a last resort.
25. **Supportive care throughout:** education, accessible toilets and toilet substitutes, timing of diuretics, fluid and caffeine advice, absorbent pads (never as the *first* response), and meticulous skin care.
26. **Prognosis:** UI is **curable or controllable in most older patients** with adequate mobility and cognition; when not curable it can always be managed to keep the patient comfortable and the caregiver's burden low.

Latest Updates (2023–Present)

- **AUA/SUFU Idiopathic Overactive Bladder Guideline 2024** replaced the older stepped "first/second/third-line" framework with **shared decision-making**; patients may move directly to any treatment tier. Behavioural therapy remains the foundation but need no longer be exhausted before advanced therapy.
- The same guideline explicitly recommends **frailty assessment (Clinical Frailty Scale)** and anticholinergic-burden review before prescribing antimuscarinics, and prefers **β3-agonists (mirabegron, vibegron) in older and cognitively vulnerable adults**.
- **Vibegron (β3-agonist)** received FDA approval in **December 2024** for men with OAB symptoms already on pharmacological therapy for BPH, based on the phase-3 **COURAGE / URO-901-3005** trial in ~1100 men — a group previously restricted to antimuscarinics.
- **AUA/GURS/SUFU Incontinence after Prostate Treatment Guideline (2024)** and **AUA/SUFU/AUGS Genitourinary Syndrome of Menopause Guideline (2025)** formalised, respectively, post-prostatectomy stress UI management and low-dose vaginal oestrogen for urogenital atrophy.
- **Cochrane and guideline-level evidence continues to place supervised pelvic floor muscle training first** for stress and mixed UI in women — around an eight-fold higher chance of cure or improvement versus no treatment.

- **Indian data:** LASI Wave 1 (2017–18, 72,250 participants aged ≥45) shows UI is strongly linked to insomnia, sarcopenia, diabetes and household solid-fuel smoke; self-report grossly under-estimates true prevalence, confirming that **active case-finding, not passive reporting, is the Indian priority**. Continence care is now embedded in NPHCE geriatric clinics and district geriatric units.

Current Practice Update

Issue	Textbook position	Current evidence / practice
Treatment sequencing	Stepwise: behavioural → drug → surgery	AUA/SUFU 2024 — **shared decision-making**; any tier may be entered directly based on bother and patient preference
Drug of choice for OAB in the elderly	Antimuscarinics listed first, β3-agonists barely represented	**β3-agonists preferred** in older adults and cognitive impairment; antimuscarinics carry a dementia-risk signal with cumulative exposure
Men with BPH + OAB	α-blocker ± antimuscarinic, retention feared	**Vibegron approved (2024)** for exactly this group; low retention risk
Oestrogen	Topical oestrogen adjunctive; oral may worsen UI	Unchanged and reinforced — 2025 GSM guideline supports **low-dose vaginal oestrogen**, including after breast cancer with counselling
Urodynamics before treatment	Selected patients only	Confirmed — routine urodynamics before conservative or first-line drug therapy is **not** recommended

Extra Points to Remember (Recurring Topic)

Mnemonics

- **DRIP** — Delirium · Restricted mobility, Retention · Infection, Inflammation, Impaction · Polyuria, Pharmaceuticals (the textbook's own mnemonic for reversible causes).
- **DIAPPERS** — Delirium, Infection, Atrophic vaginitis/urethritis, Pharmaceuticals, Psychological, Excess urine output, Restricted mobility, Stool impaction (classic alternative; quote both).
- Four persistent types — **"S-U-O-F"**: Stress, Urge, Overflow, Functional.

Numerical anchors

- Normal bladder capacity **300–600 mL**; first urge **150–300 mL**; filling pressure **<15 cm H_2O**.
- Frequency = **>8 voids/24 h** or voiding more often than 2-hourly; nocturia = **≥2 voids** at night.
- PVR **>200 mL** abnormal; keep intermittent catheter volumes **<400 mL**; Qmax **<10 mL/s** abnormal.
- Prompted voiding success **25–40%**; nursing-home prevalence **60–80%**; DHIC = emptying **<1/3** of bladder volume.

Examiner traps — (1) Do not say ageing causes incontinence; say ageing *predisposes*. (2) Do not order urodynamics routinely; it is for selected patients only. (3) Do not forget PVR — overflow incontinence is easily missed on examination. (4) Do not treat asymptomatic bacteriuria in a

stable nursing-home resident. (5) Do not prescribe oral oestrogen. (6) Remember DHIC — antimuscarinics can precipitate retention in these patients.

15-second answer skeleton: Definition → prevalence → ageing predisposes (not causes) → two basic faults (storage/emptying) → acute vs persistent → four types + mixed + DHIC → evaluation: 4 basics (history, examination, urinalysis, PVR) + selected further tests + referral criteria → treatment: DRIP → behavioural → drugs by type → devices/surgery → catheters last → supportive care → prognosis.

Exam One-Liners

- UI = involuntary, objectively demonstrable urine loss that is a social or health problem.
- Four persistent types: stress, urge, overflow, functional — overlap is the rule.
- Mixed (stress + urge) is commonest in older women; urge + functional in frail elders.
- Reversible causes: **DRIP** / **DIAPPERS**.
- Basic evaluation = history + examination + urinalysis + PVR. PVR >200 mL is abnormal.
- Pelvic floor muscle exercises give the greatest benefit of all behavioural measures.
- Antimuscarinics take 1–2 months for full effect; β3-agonists are safer in the frail elderly.
- Topical, never oral, oestrogen for atrophic vaginitis.
- DHIC = detrusor overactivity + impaired contractility; patients strain and retain.
- Catheters are for specific indications, never for convenience.

QUESTION 2

Types, diagnosis and management of incontinence in the elderly, including reversible causes. 15 MARKS

*This question overlaps Question 1; the classification and treatment framework there applies. The points below are weighted towards **reversible causes and diagnosis**, which is what this phrasing is testing, and include **faecal incontinence**, which "incontinence in the elderly" also covers.*

High-Yield Points

A. Requirements for Continence and Types

1. **Continence needs four things:** an intact lower urinary tract, adequate **cognition**, adequate **mobility and dexterity**, **motivation**, and an **accessible environment**. Failure of any one of these can produce incontinence in an older person.
2. **Four basic causes** of geriatric UI: lower urinary tract disease, neurological disease, functional/psychological factors, and environmental barriers — usually acting **together**.
3. **Acute (transient) incontinence** — abrupt onset with an acute illness or drug, remits when the cause is corrected. **Persistent incontinence** — long-standing, unrelated to acute illness.
4. **Persistent types:** stress, urge, overflow (incomplete emptying), functional — plus **mixed**, and **DHIC** as a difficult subgroup. **Faecal incontinence** coexists in 30–50% of nursing-home patients with frequent UI.

B. Reversible Causes — the core of this answer

5. **Mnemonic DRIP**: **D**elirium · **R**estricted mobility and **R**etention · **I**nfection, **I**nflammation, **I**mpaction · **P**olyuria and **P**harmaceuticals.
6. **Delirium and acute illness** — hospital-acquired incontinence is often preventable; manage with environmental change, scheduled toileting and toilet substitutes rather than a catheter.
7. **Restricted mobility** — new immobility, restraints, bed rails, unfamiliar ward, absent call bell, walking aid out of reach.
8. **Retention with overflow** — suspect in **any** patient with sudden-onset incontinence. Precipitated by immobility, anticholinergics, opioids and faecal impaction; may also signal **acute spinal cord compression**.
9. **Infection and inflammation** — acute cystitis, atrophic vaginitis, urethritis. Treating these can restore continence.
10. **Faecal impaction** — large impactions obstruct the bladder outlet in women and trigger involuntary contractions through rectal distension; disimpaction alone may cure the incontinence.
11. **Polyuria** — hyperglycaemia, hypercalcaemia, diuretics (especially rapid loop diuretics), excess fluid or caffeine, and volume-expanded states (heart failure, venous insufficiency) causing **nocturnal polyuria**.
12. **Pharmaceuticals — memorise this list:**
    - Diuretics → polyuria, urgency
    - Anticholinergics, tricyclics, antipsychotics → retention, overflow, impaction, sedation
    - Sedative-hypnotics → sedation, delirium, immobility
    - Opioids → retention, impaction, delirium

- α-blockers → urethral relaxation (stress leak); α-agonists → retention
- Cholinesterase inhibitors → frequency and urgency
- ACE inhibitors → cough precipitating stress leak
- Calcium-channel blockers, gabapentin, pregabalin, glitazones → oedema and nocturia
- Alcohol and caffeine → polyuria, urgency, bladder irritation

C. Diagnosis

13. **Screen actively** in every geriatric assessment — three direct questions about bladder trouble, involuntary loss, and pad use.
14. **Basic evaluation:** focused history + bladder record, targeted examination, urinalysis (± culture), and **post-void residual**. Blood glucose and calcium if there is unexplained polyuria.
15. **Bladder diary** for 3 days distinguishes nocturnal polyuria from a true storage problem and quantifies leaks.
16. **Simple bedside urodynamics:** observation of voiding, and the **cough stress test** — leak with cough while supine and the bladder relatively empty suggests intrinsic sphincter deficiency.
17. **Selected further tests:** urine culture and cytology, renal function, renal ultrasound, cystourethroscopy, and formal **multichannel urodynamics** (flowmetry, cystometrogram, pressure–flow, leak-point pressure, sphincter EMG, video urodynamics).
18. **Follow the diagnostic algorithm:** basic evaluation → reversible factors found? treat → indication for referral? refer → otherwise a therapeutic trial (behavioural ± drug for stress/urge/mixed; behavioural + supportive for functional) → still incontinent and suitable? then further urological, gynaecological or urodynamic evaluation.

D. Management

19. **Treat reversible causes first**, then match therapy to type: pelvic floor exercises ± α-agonist/topical oestrogen (stress); bladder training + antimuscarinic or β3-agonist (urge); relieve obstruction, intermittent catheterisation (overflow); scheduled/prompted voiding and environmental change (functional).
20. **Faecal incontinence:** commonest cause is **faecal impaction with overflow soiling**; also laxative abuse, neurological disease, sphincter damage, diarrhoeal and colorectal disease. Manage constipation with fibre **4–6 g/day**, fluid, mobility, correct positioning, and use of the **gastrocolic reflex** after meals in an unhurried private setting. Avoid laxative overuse — it can cause an atonic "cathartic colon".

Latest Updates (2023–Present)

- **Anticholinergic burden is now a formal target.** The AGS **Beers Criteria 2023** keep oral oxybutynin and other non-selective antimuscarinics on the avoid-list for older adults; cumulative anticholinergic exposure is associated with incident dementia in large cohort studies, and deprescribing is an explicit part of incontinence care.
- **AUA/SUFU 2024** requires exclusion of reversible contributors and a documented **PVR** before advanced therapy in anyone with emptying symptoms, prior retention, enlarged prostate, neurological disease or long-standing diabetes.
- **SGLT-2 inhibitors** are now a common and often-missed iatrogenic cause of new frequency, urgency, nocturia and genital infection in older diabetics — a modern addition to the "polyuria/pharmaceuticals" list not present in older drug tables.

- **WHO ICOPE (2nd edition, 2025)** incorporates continence into intrinsic-capacity screening, positioning incontinence as a marker of declining capacity rather than an isolated urological symptom.
- **Indian context:** LASI-based analyses (2023–24) link UI in older Indians to insomnia, sarcopenia, diabetes and indoor biomass smoke — all modifiable, and all reasons to look beyond the bladder in an Indian geriatric clinic.

Current Practice Update

Issue	Textbook position	Current evidence / practice
Drug list of reversible causes	Classical list — diuretics, anticholinergics, opioids, α-agents, CCBs	Add **SGLT-2 inhibitors** and cumulative **anticholinergic burden score** as routine checks
Antimuscarinics in cognitive impairment	"Weigh benefit against risk"	Beers 2023 — **avoid** where possible; prefer β3-agonists or non-drug therapy
Asymptomatic bacteriuria	Treat before evaluating community patients; do not treat in stable nursing-home residents	Reinforced — IDSA advises **against screening or treating** asymptomatic bacteriuria in older adults, including those with chronic stable incontinence or catheters
Framing of incontinence	A geriatric syndrome	Now also an **intrinsic-capacity domain** under WHO ICOPE 2025 and a trigger for full CGA

Extra Points to Remember

- **Two mnemonics, two marks:** write **DRIP** and **DIAPPERS** together — examiners reward both.
- **The three "must-not-miss" reversible causes:** retention with overflow (may be cord compression), faecal impaction, and drug effect.
- **Numbers:** fibre 4–6 g/day for constipation; 30–50% of nursing-home patients with frequent UI also have faecal incontinence; constipation = fewer than three bowel movements per week.
- **Answer structure tip:** if the question says "including reversible causes", devote at least one-third of the answer to DRIP with the drug table — that is where the marks are.

Examiner traps — (1) Do not present incontinence as purely urological; continence needs cognition, mobility, motivation and environment. (2) Do not miss retention as the cause of "new incontinence". (3) Do not use an indwelling catheter to manage hospital-acquired incontinence. (4) Do not treat asymptomatic bacteriuria in a stable institutionalised patient. (5) Do not omit faecal incontinence when the question says "incontinence in the elderly".

Exam One-Liners

- Continence = intact lower tract + cognition + mobility + motivation + accessible toilet.
- Acute (transient) UI reverses with the cause; persistent UI does not.
- DRIP and DIAPPERS cover every reversible cause worth naming.
- Sudden new incontinence = exclude retention, and think spinal cord compression.

- Faecal impaction can cause both urinary and faecal incontinence; disimpaction may cure both.
- Basic work-up is history, examination, urinalysis, PVR — nothing more in most patients.
- Beers 2023: avoid non-selective antimuscarinics in older adults.
- SGLT-2 inhibitors are a modern iatrogenic cause of urgency and nocturia.

QUESTION 3

A 65-year-old woman presents with frequent urination, nocturia and involuntary leakage for 6 months. Discuss causes, evaluation and management. 15 MARKS

High-Yield Points

A. Framing the Case

1. **The symptom triad** — frequency (>8 voids/24 h), nocturia (≥2 voids at night) and involuntary leakage for 6 months — is **persistent incontinence with an irritative (storage) symptom profile**, i.e. most likely **urgency incontinence / overactive bladder**, commonly **mixed** with stress incontinence at this age.
2. **Age 65 with 6 months' duration** excludes a purely acute/transient picture, but reversible contributors must still be sought — they are present in a large proportion of such patients.

B. Causes to Consider

3. **Urgency incontinence / detrusor overactivity** — idiopathic, or secondary to stone, tumour, diverticulum, outflow obstruction, or CNS disease (stroke, dementia, parkinsonism, cord lesion).
4. **Stress incontinence** — pelvic floor weakness with urethral hypermobility after childbirth, prior pelvic surgery, obesity, chronic cough and oestrogen deficiency; or **intrinsic sphincter deficiency** after previous vaginal repair or bladder-neck suspension.
5. **Mixed incontinence** — the commonest single answer in an older woman.
6. **Atrophic vaginitis / urethritis** — postmenopausal oestrogen deficiency causing dysuria, frequency and urgency, and predisposing to UTI.
7. **Urinary tract infection** — must be excluded; also recurrent UTI as a marker of underlying pathology.
8. **Overflow from incomplete emptying** — large cystocele or pelvic prolapse distorting the urethra, diabetic neuropathic bladder, or drug-induced retention.
9. **Polyuria states** — uncontrolled **diabetes mellitus** (very likely in an Indian 65-year-old), hypercalcaemia, excess fluid/caffeine, diuretics.
10. **Nocturnal polyuria** — loss of the night-time vasopressin rhythm, raised ANP, heart failure, venous insufficiency with leg oedema redistributing at night, obstructive sleep apnoea.
11. **Malignancy** — new irritative symptoms with haematuria or smoking history raise the question of **bladder cancer**; pelvic mass must be excluded.
12. **Functional contributors** — arthritis, poor vision, distant or Indian-style squat toilet, night-time falls risk, and drugs (sedatives, diuretics timed at night).

C. Evaluation

13. **History:** characterise leakage (with cough/laugh vs preceded by urgency vs continuous), volume, pad use, most bothersome symptom, impact on quality of life; fluid, caffeine and alcohol intake; obstetric and surgical history; diabetes, heart failure, neurological symptoms; bowel habit; full **drug review**; and a **3-day bladder diary**.
14. **Examination:** BMI, oedema and cardiac status; abdominal palpation for a distended bladder or mass; **pelvic examination** for atrophic vaginitis, cystocele/prolapse grade and

perineal sensation; **cough stress test**; rectal examination for impaction and sphincter tone; mobility, gait and cognition; focal neurology and sacral reflexes.

15. **Bedside/basic investigations:** urinalysis and, if indicated, urine culture; **post-void residual** by ultrasound; capillary and fasting **blood glucose (mg/dL)**, HbA1c; serum **calcium (mg/dL)**, **creatinine (mg/dL)**; haemoglobin (g/dL).
16. **Interpretation:** PVR **>200 mL** → suspect obstruction or impaired contractility, arrange renal function and ultrasound ± urodynamics. Persistent haematuria without infection → **urine cytology and cystoscopy**.
17. **Refer** for gynaecological or urological/urodynamic evaluation if there is marked prolapse, ≥2 symptomatic UTIs in a year, recent pelvic surgery or irradiation, suspicion of malignancy, significant retention, or failure of an adequate therapeutic trial.

D. Management

18. **Correct reversible factors:** treat UTI, relieve constipation, control diabetes, review diuretic timing, stop or substitute offending drugs, treat heart failure and leg oedema (evening leg elevation, compression stockings).
19. **Lifestyle and conservative measures (first and always):** weight reduction; fluid target of **1.5–2 L/day of non-caffeinated fluid** with restriction in the evening; avoid caffeine, carbonated drinks and nicotine; treat chronic cough and constipation; safe night-time route to the toilet with a light and a bedside commode to prevent falls.
20. **Pelvic floor muscle training**, supervised, with biofeedback if available — the most effective behavioural intervention, benefiting both stress and urge components; add **bladder training** with progressive lengthening of the voiding interval and urge-suppression techniques.
21. **Topical vaginal oestrogen** if atrophic vaginitis is present — cream 0.5–1.0 g/day for 2 weeks then twice weekly, or an oestradiol ring 3-monthly. **Never oral oestrogen.**
22. **Drug therapy** if behavioural measures are insufficient: a **β3-agonist (mirabegron or vibegron)** is preferred in older adults; antimuscarinics (solifenacin, darifenacin, fesoterodine, trospium) are alternatives with counselling about dry mouth, constipation, glaucoma and cognitive effects. Check baseline renal function before a β3-agonist and blood pressure on treatment. Allow **4–6 weeks** before judging response.
23. **Refractory nocturia** — reassess for nocturnal polyuria and sleep apnoea first; desmopressin only in carefully selected patients with close monitoring of serum sodium (mEq/L).
24. **Refractory OAB** — intradetrusor **botulinum toxin A** (repeat not more often than 6-monthly; counsel about retention and need for self-catheterisation) or **sacral neuromodulation** / posterior tibial nerve stimulation.
25. **Predominant stress incontinence not responding to conservative care** — pessary for prolapse, urethral bulking agents, or surgery (colposuspension, autologous rectus fascial sling, mid-urethral sling) after multidisciplinary discussion; counsel about long-term mesh-related uncertainty.
26. **Review at 4–6 weeks** with a repeat bladder diary, assess symptom improvement and satisfaction, and escalate or de-escalate accordingly. Address the psychosocial impact — shame, social withdrawal and depression are common and often unspoken.

Latest Updates (2023–Present)

- **AUA/SUFU 2024** allows this patient to move directly to a β3-agonist or even to botulinum toxin if bother is high — behavioural therapy no longer has to fail first, though it should always be offered.
- **β3-agonists over antimuscarinics** is now the preferred pharmacological default in women over 65, driven by the anticholinergic–dementia signal and Beers 2023.
- **AUA/SUFU/AUGS Genitourinary Syndrome of Menopause Guideline 2025** endorses low-dose vaginal oestrogen for urogenital atrophy, recurrent UTI prevention and irritative symptoms, with a favourable safety profile.
- **Surgical caution persists:** following regulatory action in several countries on synthetic mesh, **autologous fascial slings and colposuspension** have regained ground, and bulking agents are offered when other surgery is unsuitable.
- **Indian relevance:** LASI data link UI in older Indian women with insomnia, sarcopenia and household biomass smoke; obstetric history of high parity and home delivery, plus squat-toilet dependence, are practical local determinants to elicit and address.

Current Practice Update

Issue	Textbook position	Current evidence / practice
First drug for OAB in a woman of 65	Antimuscarinic	**β3-agonist preferred**; antimuscarinic second-line with anticholinergic-burden counselling
Order of therapy	Strict stepwise ladder	Shared decision-making — entry at any tier depending on bother
Stress UI surgery	Mid-urethral synthetic sling as standard	Mesh restricted in many countries; **autologous fascial sling / colposuspension** and bulking agents prominent; MDT discussion mandated
Time to judge drug response	Up to 1–2 months for full effect	Formal review at **4–6 weeks** with a repeat bladder diary and satisfaction assessment

15-second answer skeleton: Define the symptom complex (storage/irritative, likely urge or mixed) → list causes systematically (bladder, urethra/pelvic floor, infection/atrophy, polyuria, nocturnal polyuria, neurological, malignancy, functional, drugs) → evaluation (history + diary, examination incl. pelvic and cough test, urinalysis, PVR, glucose/calcium/creatinine) → interpretation and referral triggers → management (reversible factors → lifestyle → pelvic floor and bladder training → vaginal oestrogen → β3-agonist/antimuscarinic → botulinum/ neuromodulation → surgery) → review at 4–6 weeks and address psychosocial impact.

Exam One-Liners

- Frequency + nocturia + leakage in an older woman = OAB/urgency incontinence, usually mixed with stress.
- Always exclude UTI, uncontrolled diabetes, constipation and drugs before labelling it idiopathic.
- 3-day bladder diary separates nocturnal polyuria from a storage problem.

- Cough stress test with an empty bladder in the supine position suggests intrinsic sphincter deficiency.
- PVR >200 mL changes the whole management plan — always measure it.
- Pelvic floor muscle training helps both the stress and the urge component.
- Vaginal, not oral, oestrogen for atrophic vaginitis.
- β3-agonist first in the elderly; review at 4–6 weeks.
- New irritative symptoms + haematuria + smoking = exclude bladder cancer.

QUESTION 4

Urinary incontinence in males — types, epidemiology, pathogenesis, risk factors, screening, evaluation and management. 15 MARKS

High-Yield Points

A. Epidemiology

1. **15–20% of men above 65 years** have some degree of urinary incontinence — roughly half the female rate, but rising steeply with age, frailty and institutionalisation.
2. **OAB symptoms are reported by about 40% of men aged ≥75**, most of whom also have urgency incontinence.
3. Male incontinence in nursing homes is common and men managed with chronic catheters are at **particularly high risk of symptomatic infection**.

B. Types in Men

4. **Urgency incontinence / OAB** — commonest type in older men; usually secondary to detrusor overactivity from bladder outlet obstruction, stroke, Parkinson's disease or dementia.
5. **Overflow incontinence from incomplete emptying** — the characteristically "male" type: benign prostatic obstruction, urethral stricture, or an acontractile bladder from diabetic neuropathy.
6. **Stress incontinence** — **unusual in men** and almost always iatrogenic: after transurethral surgery, radical prostatectomy or pelvic radiotherapy damaging the sphincter.
7. **Functional incontinence** — dementia, stroke, immobility, restraints, inaccessible toilet.
8. **Mixed patterns and DHIC** (detrusor overactivity with impaired contractility) are frequent in the very old male and explain why antimuscarinics can precipitate retention.
9. **Post-micturition dribble** — a distinct, benign symptom from bulbar urethral pooling, treated by urethral milking, not by drugs.

C. Pathogenesis

10. **Normal continence in the male** depends on parasympathetic (nervi erigentes, S2–S4) detrusor contraction, sympathetic (hypogastric, T11–L2) bladder relaxation and bladder-neck/urethral α-adrenergic contraction, and somatic (pudendal) external sphincter control, coordinated at the sacral micturition centre under cortical inhibition.
11. **Prostatic enlargement** raises outlet resistance → detrusor hypertrophy, collagen deposition, loss of compliance and **secondary detrusor overactivity** → urgency and frequency; later, detrusor decompensation → high residual urine and overflow.
12. **Ageing changes** — reduced bladder capacity, higher residual volume, more involuntary contractions, altered receptor profile with M3 receptors functionally dominant, and loss of nocturnal vasopressin rhythm with raised ANP causing **nocturnal polyuria**.
13. **Neurological disease:** stroke — detrusor hyperreflexia in ~68–82%, detrusor–sphincter dyssynergia in ~14%, and incontinence in the acute phase is a powerful predictor of poor outcome. Parkinson's disease — detrusor hyperreflexia in up to 79%, mimicking BPH; multiple system atrophy adds sphincter dyssynergia.

14. **Sphincter injury** after prostatic surgery or radiotherapy produces true stress incontinence with intrinsic sphincter deficiency.

D. Risk Factors

15. **Age; benign prostatic enlargement and LUTS; prostate surgery or radiotherapy; neurological disease** (stroke, Parkinson's, dementia, cord lesion, multiple sclerosis); **diabetes mellitus** (neuropathic bladder, osmotic diuresis); obesity and metabolic syndrome; immobility and frailty; constipation and faecal impaction; **polypharmacy** (anticholinergics, opioids, diuretics, sedatives, α-agonists, calcium-channel blockers, SGLT-2 inhibitors); alcohol and caffeine; chronic catheterisation.

E. Screening

16. **Case-finding is essential** — men under-report because of embarrassment. Use the three standard questions on bladder trouble, involuntary loss and pad use as part of every comprehensive geriatric assessment and in every long-term care admission.
17. **Quantify symptoms** with the **IPSS / AUA Symptom Index** (seven items, each 0–5: **mild 0–7, moderate 8–19, severe 20–35**) plus the single quality-of-life question — this is the standard screening instrument in men.

F. Evaluation

18. **Basic evaluation:** focused history with bladder diary, targeted examination including **digital rectal examination** (size, consistency, nodules, sphincter tone) and neurological assessment, **urinalysis**, and **post-void residual**.
19. **Clean urine sample** in a frequently incontinent man can be obtained through a condom catheter after cleaning the penis.
20. **Uroflowmetry** is a valuable non-invasive screen: **Qmax <10 mL/s on a voided volume >150 mL** indicates obstruction or impaired contractility.
21. **Selected tests:** serum creatinine (mg/dL) and renal ultrasound if retention or hydronephrosis is suspected; blood glucose (mg/dL) and calcium (mg/dL) for polyuria; **PSA (ng/mL)** after shared decision-making where life expectancy exceeds 10 years; urine cytology and cystoscopy for haematuria or new irritative symptoms in a smoker; **multichannel urodynamics** in neurological disease, prior failed surgery, or before considering prostatic surgery in an equivocal case.

G. Management

22. **Reverse the reversible** (DRIP), then match treatment to type.
23. **Behavioural therapy** — pelvic floor muscle training (also effective for post-prostatectomy stress leak), bladder training, timed voiding, fluid and caffeine modification, double voiding, and urethral milking for post-micturition dribble. **Combined behavioural plus drug therapy is superior to either alone in men with OAB.**
24. **Drugs:** α-blocker (tamsulosin 0.4 mg, alfuzosin 10 mg, silodosin 4 mg) for obstruction-related symptoms — watch postural hypotension and falls; add a **5-α-reductase inhibitor** if prostate volume exceeds 30–40 mL; add an antimuscarinic or a **β3-agonist** for persistent storage symptoms provided PVR is not elevated.
25. **Surgery:** TURP or laser enucleation for obstruction with retention, renal impairment, recurrent UTI, stones or gross haematuria; **artificial urinary sphincter** or male sling for post-prostatectomy stress incontinence; intradetrusor botulinum toxin or sacral neuromodulation for refractory OAB.

26. **Containment when cure is not possible:** external (condom) catheter only in a dependent man **without retention**, absorbent products, and intermittent catheterisation for chronic retention; chronic indwelling catheters only for defined indications.

Latest Updates (2023–Present)

- **Vibegron (Gemtesa) approved December 2024** for men with OAB symptoms already receiving pharmacological therapy for BPH — the first β3-agonist with this specific indication, based on the phase-3 **COURAGE (URO-901-3005)** trial in ~1100 men showing significant reduction in micturitions, urgency episodes, nocturia and urgency incontinence with low retention risk.
- **AUA/GURS/SUFU Incontinence after Prostate Treatment Guideline (2024)** standardises evaluation and management of post-prostatectomy incontinence, reaffirming the **artificial urinary sphincter** as the definitive option for significant stress leak and reserving slings for mild-to-moderate leak.
- **AUA/SUFU OAB Guideline 2024** recommends β3-agonists preferentially in older men, mandates PVR before advanced therapy in men with an enlarged prostate, and integrates frailty assessment into decision-making.
- **AUA LUTS/BPH Guideline (2023 amendment, expanded 2026)** reframes management around **bladder function preservation** and shared decision-making rather than a fixed treatment ladder, and formally includes minimally invasive surgical therapies and prostatic artery embolisation.
- **Indian context:** LASI analyses show older Indian men with type 2 diabetes carry the highest burden of incontinence among urological disorders, ahead of stones and prostate disease — making **glycaemic control an incontinence intervention** in Indian practice. NPHCE district geriatric units and AB-PMJAY (with the Ayushman Vay Vandana Card for those aged 70+) now cover prostatic surgery and continence services.

Current Practice Update

Issue	Textbook position	Current evidence / practice
Storage symptoms in men on BPH therapy	α-blocker ± antimuscarinic, with retention caution	**Vibegron licensed (2024)** for this exact population; β3-agonist preferred over antimuscarinic in the elderly
Post-prostatectomy incontinence	Brief mention; artificial sphincter for selected men	Dedicated **2024 guideline**; pelvic floor training early, sling for mild–moderate, **AUS for severe**
Behavioural vs drug therapy in men	Behavioural effective, drugs added if needed	**Combination superior to either alone** for male OAB symptoms
Frailty in treatment choice	Not formally incorporated	Clinical Frailty Scale and anticholinergic burden are now explicit decision inputs

Extra Points to Remember

- **Key contrast to write down:** in women the commonest type is stress/mixed; **in men it is urgency and overflow, with stress being almost always post-surgical.** Examiners look for this line.

- **Numbers:** 15–20% prevalence >65 y; OAB symptoms ~40% at ≥75 y; IPSS 0–7 mild / 8–19 moderate / 20–35 severe; Qmax <10 mL/s abnormal on volume >150 mL; prostate >30–40 mL justifies a 5-ARI; 5-ARI takes 3–6 months to act.
- **Mnemonic for male UI causes — "PROSTATE":** Prostatic obstruction · Radiotherapy/ Radical prostatectomy · Overactive bladder · Stricture · Tumour (bladder/prostate) · Autonomic neuropathy (diabetes) · Toxins/drugs · Environment and function.
- **Always measure PVR before an antimuscarinic in a man** — this single step prevents iatrogenic retention and is a reliable mark-earner.

Examiner traps — (1) Do not attribute all male LUTS to the prostate; stroke, Parkinson's and diabetes are common and treatable contributors. (2) Do not give an antimuscarinic without knowing the PVR. (3) Do not use a condom catheter in a man with retention. (4) Do not order PSA during an acute male UTI — it needs about 6 months to return to baseline. (5) Do not forget post-micturition dribble as a separate, drug-resistant entity.

Exam One-Liners

- 15–20% of men over 65 are incontinent; urgency and overflow dominate.
- Stress incontinence in a man is iatrogenic until proved otherwise.
- IPSS: mild 0–7, moderate 8–19, severe 20–35.
- Qmax <10 mL/s on a void of >150 mL suggests obstruction or poor contractility.
- DHIC explains why an antimuscarinic can tip an old man into retention.
- Combined behavioural + drug therapy beats either alone in male OAB.
- Vibegron (2024) is the first β3-agonist licensed for men with OAB on BPH therapy.
- Artificial urinary sphincter is the definitive treatment for severe post-prostatectomy stress incontinence.

QUESTION 5

Describe an algorithm to investigate and treat an elderly male with lower urinary tract symptoms. 15 MARKS

High-Yield Points

A. Definition and Symptom Grouping

1. **LUTS** in older men are grouped as **storage** (frequency, urgency, nocturia, urgency incontinence), **voiding** (hesitancy, weak or intermittent stream, straining, terminal dribble, incomplete emptying) and **post-micturition** (post-void dribble, sensation of incomplete emptying).
2. **LUTS ≠ BPH.** Symptoms may arise from the bladder (overactivity, underactivity), prostate, urethra, or systemic disease. Prevalence of BPH-related symptoms approaches **50%** in multinational surveys of older men.

B. Step 1 — Initial Assessment

3. **History** — symptom pattern and duration; the **most bothersome symptom**; fluid, caffeine, alcohol; nocturia pattern; and specific pointers:
   - Type 2 diabetes with nocturia and polyuria >3 L → osmotic diuresis or neuropathic bladder
   - Neurological symptoms → neurogenic bladder
   - Gross haematuria or suprapubic pain → bladder tumour or calculus
   - Urethral trauma, urethritis or instrumentation → stricture
   - Family history of BPH or prostate cancer
   - Anticholinergic or sympathomimetic drugs impairing bladder function or raising outflow resistance
4. **Quantify with IPSS/AUA-SI** (mild 0–7, moderate 8–19, severe 20–35) plus the quality-of-life question. The score measures **severity, not aetiology**.
5. **Examination** — abdominal (palpable bladder), **digital rectal examination** for prostate size, consistency, nodules and sphincter tone, plus focused neurological examination; assess cognition, mobility and general fitness for any future procedure.
6. **Bladder diary** for 3 days — separates nocturnal polyuria and excessive fluid intake from true bladder dysfunction.

C. Step 2 — Basic Investigations

7. **Urinalysis** in every patient — detects infection and haematuria. **Urine cytology** if predominantly irritative symptoms or haematuria, especially in a smoker.
8. **Post-void residual** by bladder ultrasound; **uroflowmetry** where available (Qmax <10 mL/s on a void >150 mL is abnormal).
9. **Serum creatinine (mg/dL)** — not required routinely by AUA guidance in uncomplicated LUTS, but mandatory if there is retention, suspected obstructive uropathy, or planned surgery. **Renal ultrasound** if creatinine is raised or hydronephrosis suspected.
10. **PSA (ng/mL)** after shared decision-making in men with a life expectancy over 10 years; remember PSA is not cancer-specific, rises with BPH, prostatitis and instrumentation, and **25% of prostate cancers have a PSA ≤4.0 ng/mL**.

D. Step 3 — Identify "Complicated" LUTS Needing Referral

11. **Red flags for urological referral:** acute or chronic urinary retention, renal insufficiency due to obstruction, recurrent UTI, bladder stones, gross haematuria, suspicious DRE or raised PSA, previous pelvic surgery or radiotherapy, neurological disease, urethral stricture, or failure of medical therapy.
12. **Advanced tests in this group:** cystoscopy, upper-tract imaging, and **multichannel urodynamics/pressure-flow study** to distinguish true bladder outlet obstruction from detrusor underactivity before committing to surgery.

E. Step 4 — Treatment Pathway

13. **Mild symptoms (IPSS <7) or minimal bother → watchful waiting with lifestyle advice:** evening fluid restriction, avoid caffeine and spicy food, review diuretics and their timing, timed/organised voiding, pelvic floor exercises, treat constipation, double voiding — with periodic review.
14. **Moderate to severe symptoms with bother → medical therapy:**
    - **α-blocker** (tamsulosin, alfuzosin, silodosin, terazosin, doxazosin) — fastest symptomatic relief within days to weeks; does **not** alter disease progression. Side effects in ~5–9%: dizziness, postural hypotension, asthenia, rhinitis, retrograde ejaculation.
    - **5-α-reductase inhibitor** (finasteride, dutasteride) if the prostate is enlarged (>30–40 mL) or PSA is raised — shrinks the gland by up to 30%, takes **3–6 months**, and reduces the risk of acute retention and surgery.
    - **Combination α-blocker + 5-ARI** for prostate volume >40 mL — the **MTOPS** trial showed a 66% reduction in acute urinary retention versus 39% with α-blockade alone and 34% with 5-ARI alone. The α-blocker may be stopped after 6–9 months and restarted if symptoms recur.
    - **Antimuscarinic or β3-agonist** added for persistent storage symptoms **only when PVR is not elevated**; avoid in men with a history of retention or cognitive impairment.
    - **Phytotherapy is not recommended** by AUA guidance.
15. **Minimally invasive therapy** for moderate symptoms with a small-to-moderate gland in a man wishing to avoid major surgery — transurethral microwave thermotherapy and related energy-based office procedures; higher retreatment rates than TURP, and several weeks before improvement.
16. **Surgery — indications:** renal insufficiency from BPH, recurrent UTI, gross haematuria due to BPH, bladder stones, refractory retention, or LUTS refractory to medical therapy.
17. **TURP remains the reference standard** for glands under 80 mL, with immediate symptom and flow improvement; transurethral incision for small glands; **laser enucleation (HoLEP/ThuLEP)** and open or simple robotic prostatectomy for very large glands.
18. **Ongoing review:** reassess IPSS, PVR, renal function and adverse effects; treat comorbid contributors (heart failure, diabetes, sleep apnoea, constipation) and, in the frail, always balance symptom benefit against hypotension, falls and delirium risk.

Algorithm at a Glance

Step	Action	Decision
1. Assess	History, IPSS + QoL, DRE, bladder diary, examination	Storage vs voiding vs mixed pattern

2. Test	Urinalysis, PVR, ± uroflowmetry, ± creatinine, ± PSA	Uncomplicated vs complicated
3. Triage	Red flags present? (retention, renal failure, recurrent UTI, stones, haematuria, abnormal DRE/PSA, neurological disease)	Yes → urology referral, cystoscopy, imaging, urodynamics
4a. Mild	IPSS <7 or low bother	Watchful waiting + lifestyle
4b. Moderate/ severe	α-blocker → add 5-ARI if gland >40 mL → add antimuscarinic/β3-agonist if storage symptoms persist and PVR normal	Review at 4–12 weeks
5. Failure	Minimally invasive therapy or surgery (TURP / laser enucleation / PAE in selected men)	Choose by gland size, comorbidity and patient preference

Latest Updates (2023–Present)

- **AUA LUTS/BPH Guideline Amendment 2023** (expanded into a three-part guideline in 2026) revised statements on combination therapy, photoselective vaporisation (PVP), **water vapour thermal therapy**, laser enucleation and — importantly — formally admitted **prostatic artery embolisation (PAE)** into the guideline for selected men.
- The 2026 guideline reframes the whole pathway around **bladder function preservation and shared decision-making**, rather than a rigid escalation ladder, and stresses that LUTS/BPH is multifactorial with both storage and emptying components.
- **Urodynamics before prostate surgery is not mandatory** — the UPSTREAM randomised trial (26 hospitals) showed a non-invasive pathway is non-inferior to routine urodynamics for symptom outcomes, supporting selective rather than routine use.
- **Minimally invasive surgical therapies (MISTs)** — water vapour thermal therapy, prostatic urethral lift, temporary implantable devices and PAE — have expanded the options for frail older men unfit for anaesthesia, with the trade-off of higher retreatment rates.
- **Metabolic drivers recognised:** obesity, insulin resistance and chronic prostatic inflammation are now emphasised as modifiable contributors to LUTS/BPH progression — directly relevant to the Indian metabolic-syndrome phenotype.
- **India:** TURP and laser prostatectomy are covered under AB-PMJAY, with the **Ayushman Vay Vandana Card** extending cover to all citizens aged 70 and above irrespective of income since 2024.

Current Practice Update

Issue	Textbook position	Current evidence / practice
Framework	Four groups: watchful waiting, medical, minimally invasive, surgery	Retained, but driven by **shared decision-making** and bladder preservation, not a fixed ladder
Prostatic artery embolisation	Not included	**In the guideline** since the 2023 amendment for selected patients
TUNA	Described as an option	**Removed** from AUA surgical recommendations

Urodynamics before surgery	Advised in equivocal cases	UPSTREAM — routine urodynamics **not required**; use selectively
Large glands	Open prostatectomy	**HoLEP/ThuLEP** preferred where expertise exists — lower morbidity, size-independent

15-second answer skeleton: Define and group LUTS (storage/voiding/post-micturition) → LUTS ≠ BPH → Step 1 assess (history, IPSS+QoL, DRE, diary) → Step 2 basic tests (urinalysis, PVR, uroflow, ± creatinine, ± PSA) → Step 3 red flags → refer for cystoscopy/imaging/urodynamics → Step 4 treat: mild = watchful waiting + lifestyle; moderate/severe = α-blocker → 5-ARI if large gland → combination (MTOPS) → antimuscarinic/β3 if storage symptoms and PVR normal → Step 5 MIST or surgery (TURP standard; HoLEP for large; PAE selected) → review and manage comorbidity.

Exam One-Liners

- LUTS = storage + voiding + post-micturition symptoms; not synonymous with BPH.
- IPSS grades severity, never aetiology.
- Urinalysis and PVR are the two investigations every man with LUTS needs.
- Routine serum creatinine is not required in uncomplicated LUTS; it is mandatory if retention or obstruction is suspected.
- MTOPS: combination therapy cut acute retention by 66% versus 39% (α-blocker) and 34% (5-ARI).
- Surgery indications: retention, renal failure, recurrent UTI, stones, gross haematuria, refractory symptoms.
- TURP is standard for glands <80 mL; laser enucleation for larger glands.
- Routine pre-operative urodynamics is no longer required (UPSTREAM).

QUESTION 6

Medical management of benign prostatic hypertrophy. 7 MARKS

High-Yield Points

1. **Aim:** relieve bothersome symptoms, prevent progression (acute retention, need for surgery) and preserve bladder and renal function — treatment is guided by symptom severity, degree of bother, risk of progression and patient preference.
2. **Who to treat medically:** men with moderate to severe symptoms (**IPSS ≥8**) who are bothered. IPSS <7 with little bother → watchful waiting plus lifestyle measures (evening fluid restriction, avoid caffeine, review diuretics, timed voiding, pelvic floor exercises, treat constipation).
3. **α-adrenergic blockers — first-line.** The bladder neck and prostatic urethra are rich in α_1-receptors; blockade relaxes smooth muscle and lowers prostatic urethral pressure. **Alfuzosin 10 mg, tamsulosin 0.4 mg, silodosin 4 mg, terazosin, doxazosin.** Onset within days to weeks; **they do not alter the natural history**. Adverse effects in 5–9%: dizziness, headache, asthenia, **postural hypotension and falls**, rhinitis, retrograde ejaculation. Uroselective agents (tamsulosin, silodosin, alfuzosin) are preferred in the elderly and in those on antihypertensives; warn the ophthalmologist about **intraoperative floppy iris syndrome** before cataract surgery.
4. **5-α-reductase inhibitors — for the enlarged gland. Finasteride 5 mg, dutasteride 0.5 mg** block conversion of testosterone to dihydrotestosterone, reducing prostate volume by up to **30%**. Best in glands **>30–40 mL** or PSA-defined enlargement. Full effect takes **3–6 months**. They **reduce acute urinary retention and the need for surgery**. Side effects are mainly sexual — reduced libido, erectile and ejaculatory dysfunction, gynaecomastia. **They halve serum PSA — double the measured value when interpreting it.**
5. **Combination therapy (α-blocker + 5-ARI)** is appropriate when the prostate exceeds 40 mL. **MTOPS**: 66% reduction in acute retention with combination versus 39% (α-blocker) and 34% (5-ARI) alone; **CombAT** confirmed superiority of dutasteride + tamsulosin for symptom and progression outcomes. The α-blocker may be withdrawn after **6–9 months** and restarted if symptoms recur.
6. **Anticholinergics / β3-agonists** may be added for persistent storage symptoms (urgency, frequency, nocturia) in men **without an elevated post-void residual**. Avoid in men with prior retention or cognitive impairment — anticholinergics increase delirium risk and can precipitate retention.
7. **PDE-5 inhibitor (tadalafil 5 mg daily)** improves LUTS and is particularly useful when erectile dysfunction coexists; do not combine with nitrates, and use α-blockers cautiously alongside because of additive hypotension.
8. **Desmopressin** may be considered for troublesome nocturnal polyuria in carefully selected men, with close monitoring of serum sodium (mEq/L) — hyponatraemia is common in older adults.
9. **Phytotherapy (saw palmetto, Pygeum, beta-sitosterol)** is popular and generally well tolerated, but **AUA guidance does not recommend dietary supplements or combination phytotherapeutic agents** for LUTS/BPH.

10. **Failure of medical therapy, or complicated BPH** (renal insufficiency, recurrent UTI, bladder stones, gross haematuria, refractory retention) is an indication for minimally invasive therapy or surgery.

Latest Updates (2023–Present) — Concise

- **Vibegron (β3-agonist) FDA-approved December 2024** for men with OAB symptoms already on pharmacological BPH therapy (COURAGE/URO-901-3005 trial) — the safest current way to add storage-symptom control to an α-blocker in an older man.
- **AUA LUTS/BPH Guideline 2023 amendment and 2026 update** shift from a fixed ladder to **shared decision-making with bladder-function preservation**, revise combination-therapy statements, and reaffirm that phytotherapy is not recommended.
- **5-ARI safety signal:** large observational data show an **increased risk of depression** (though not of suicide) in men on 5-α-reductase inhibitors — counsel and monitor, particularly in older men with prior mood disorder.

Current Practice Update

Point	Textbook	Current practice
Storage symptoms on α-blocker	Add anticholinergic cautiously	**β3-agonist (vibegron) now licensed** and preferred in the elderly
Tadalafil	Minimal emphasis	Established option, especially with coexisting erectile dysfunction
5-ARI counselling	Sexual side effects	Add **depression risk** and the need to double PSA values

Extra Points to Remember (High-Yield)

- **Mnemonic — "A-5-C-A-P": A**lpha-blocker · **5**-ARI · **C**ombination · **A**nticholinergic/β3 · **P**DE-5 inhibitor.
- **Numerical anchors:** IPSS ≥8 to treat · gland >30–40 mL for 5-ARI · 30% volume reduction · 3–6 months to act · PSA halved (double it) · MTOPS 66/39/34% · α-blocker withdrawal after 6–9 months · adverse effects in 5–9%.
- **The single commonest exam slip:** forgetting that 5-ARIs halve PSA. State it explicitly.

Examiner traps — (1) α-blockers relieve symptoms but do **not** prevent progression; only 5-ARIs do. (2) Do not add an anticholinergic without checking PVR. (3) Do not recommend phytotherapy as guideline-supported. (4) Remember floppy iris syndrome before cataract surgery. (5) Watch postural hypotension and falls when starting an α-blocker in a frail elder.

Exam One-Liners

- α-blocker = fast symptom relief; 5-ARI = shrinks gland and prevents progression.
- 5-ARI needs a gland >30–40 mL, takes 3–6 months and halves PSA.
- MTOPS: combination cut acute retention by 66%.
- Add antimuscarinic or β3-agonist for storage symptoms only if PVR is normal.
- Tadalafil 5 mg daily helps LUTS plus erectile dysfunction.

- Phytotherapy is not recommended by AUA guidance.

QUESTION 7

Overactive bladder. 7 MARKS

High-Yield Points

1. **Definition:** a **symptom syndrome** whose hallmark is **urinary urgency**, usually with frequency (>8 voids/24 h) and nocturia, **with or without urgency incontinence**, in the absence of infection or other obvious pathology. OAB "wet" = with incontinence; OAB "dry" = without.
2. **Prevalence:** about **30% of women and 40% of men aged ≥75** report OAB symptoms; most of the women also have urgency incontinence. Population studies give ranges of 7–27% in men and 9–43% in women, rising with age.
3. **Pathophysiology:** usually **detrusor overactivity** — involuntary detrusor contractions during filling. Contributory mechanisms are myogenic (patchy denervation, increased spontaneous activity), neurogenic (loss of cortical inhibition in stroke, dementia, parkinsonism), and urothelial/afferent hypersensitivity. The functionally important receptor is the **M3 muscarinic receptor**; bladder relaxation is mediated by **β3-adrenoceptors**.
4. **Secondary causes to exclude:** UTI, bladder stone, tumour or diverticulum, bladder outlet obstruction, atrophic vaginitis, faecal impaction, poorly compliant bladder after radiotherapy or interstitial cystitis, diabetes, heart failure, and drugs.
5. **DHIC** — detrusor hyperactivity with impaired contractility: involuntary contractions but emptying of less than one-third of bladder volume. These patients strain to void, may not have typical urgency symptoms, and are at high risk of retention with antimuscarinics.
6. **Evaluation:** history, **bladder diary**, targeted examination (including pelvic examination in women and DRE in men), **urinalysis**, and **post-void residual** where there are emptying symptoms, prior retention, an enlarged prostate, neurological disease, previous incontinence or prostate surgery, or long-standing diabetes. Urodynamics is **not** routine.
7. **Behavioural therapy (foundation):** education, fluid and caffeine modification, weight loss, **bladder training** with timed voiding and progressive interval lengthening, urge-suppression techniques, and **pelvic floor muscle training** with biofeedback. In institutional settings, prompted voiding and habit training.
8. **Drug therapy: β3-agonists** (mirabegron 25–50 mg, vibegron 75 mg) — preferred in older adults; check renal function and monitor blood pressure. **Antimuscarinics** (solifenacin, darifenacin, fesoterodine, tolterodine, trospium, oxybutynin) — effective but limited by dry mouth, constipation, blurred vision, raised intraocular pressure, and **cognitive impairment and delirium**. Trospium and darifenacin have less CNS penetration. Maximum effect may take 1–2 months; if one agent fails, another in the class may still work.
9. **Refractory OAB:** intradetrusor **botulinum toxin A** (chemodenervation, increases capacity, lowers detrusor pressure; repeat not more often than 6-monthly; risk of retention and need for self-catheterisation), **sacral neuromodulation (S3)**, and **posterior tibial nerve stimulation**.
10. **Supportive measures throughout:** pads, liners, barrier creams, external catheters and absorbent washable underwear should be discussed with all patients who leak, together with night-time safety measures to prevent falls.

Latest Updates (2023–Present) — Concise

- **AUA/SUFU Idiopathic OAB Guideline (2024)** abolished the rigid first/second/third-line ladder in favour of **shared decision-making**, mandated assessment of frailty (Clinical Frailty Scale) and anticholinergic burden, and made patient-reported outcomes central; it also formally recommends discussing containment products with everyone who has urgency incontinence.
- **β3-agonists are now preferred over antimuscarinics in older and cognitively vulnerable adults**, reflecting the cumulative anticholinergic–dementia association and AGS Beers 2023.
- **Vibegron approved (December 2024)** for men with OAB symptoms receiving pharmacological therapy for BPH — the COURAGE trial (~1100 men) showed reduced micturitions, urgency episodes, nocturia and urgency incontinence.

Current Practice Update

Point	Textbook	Current practice
Treatment sequence	Behavioural → antimuscarinic → invasive	**Shared decision-making**; any tier can be entered directly
Drug class of choice in the elderly	Antimuscarinics dominate the tables	**β3-agonists preferred**; antimuscarinics second-line with burden counselling
Men with BPH and OAB	α-blocker ± antimuscarinic	Vibegron licensed for this exact group

Exam One-Liners

- OAB = urgency ± frequency ± nocturia, with or without incontinence; urgency is the hallmark.
- ~30% of women and ~40% of men aged ≥75 have OAB symptoms.
- M3 receptors mediate contraction; β3 receptors mediate relaxation.
- DHIC is OAB with poor emptying — antimuscarinics can cause retention.
- Urodynamics is not needed to diagnose uncomplicated OAB.
- β3-agonist first in the elderly; botulinum toxin and sacral neuromodulation for refractory cases.

QUESTION 8

Bladder retraining protocol. 7 MARKS

High-Yield Points

1. **Objective:** to **restore a normal pattern of voiding and continence after removal of an indwelling catheter**, or in patients recovering from over-distension injury, acute retention, or post-stroke voiding dysfunction.
2. **Distinguish the terms. Bladder retraining** = progressive lengthening or shortening of the inter-voiding interval, with intermittent catheterisation where retention persists. **Bladder training** = education, bladder records, pelvic floor and other behavioural techniques for stress and urge incontinence. Both are **patient-dependent** and require adequate cognition, physical function and motivation.
3. **Step 1 — Remove the indwelling catheter.** Clamping the catheter before removal is **not necessary** and is not recommended.
4. **Step 2 — Treat urinary tract infection** if present.
5. **Step 3 — Start a toileting schedule:** on waking; **every 2 hours during the day and evening**; before getting into bed; and **every 4 hours at night**.
6. **Step 4 — Monitor with a bladder record**, documenting: frequency, timing and amount of continent voids; frequency, timing and amount of incontinence episodes; fluid intake pattern; and post-void catheter (or ultrasound) residual volume.
7. **Step 5 — If the patient is having difficulty voiding** (complete retention, or very low output such as 240 mL in 8 hours despite adequate intake):
   - Perform bladder ultrasound or in-and-out catheterisation **every 6–8 hours**, recording the volume, until residuals fall to **≤200 mL**.
   - Teach **triggering techniques** — running water, stroking the inner thigh, suprapubic tapping.
   - Teach **complete-emptying techniques** — bending forward, suprapubic pressure, **double voiding**.
   - If high residuals persist beyond **1–2 weeks**, consider urodynamic evaluation.
8. **Step 6 — If the patient is voiding too frequently** (more often than 2-hourly):
   - Check post-void residual to confirm complete emptying.
   - Encourage the patient to **delay voiding as long as possible**, with emptying techniques and pelvic floor exercises.
   - If frequency, nocturia, urgency or incontinence persists — rule out reversible causes (UTI, drug effect, hyperglycaemia, heart failure) and consider further evaluation for other pathology.
9. **Duration and referral:** a patient still having voiding difficulty after **1–2 weeks** of the protocol should be re-examined for reversible causes and referred for urological assessment to exclude correctable lower genitourinary pathology.
10. **Value:** this protocol should enable removal of **most indwelling catheters** in acute hospitals and many in long-term care, thereby reducing catheter-associated infection, bladder stones, urethral trauma and immobility.

Latest Updates (2023–Present) — Concise

- **SHEA/IDSA/APIC 2022 CAUTI Compendium (published 2023)** makes structured **catheter removal protocols with a trial of void**, including nurse-driven and automatic stop-orders, an **essential practice** — bladder retraining is therefore now an infection-prevention intervention, not merely a rehabilitation one.
- **Portable bladder ultrasound has replaced routine in–out catheterisation** for residual measurement during retraining, avoiding repeated instrumentation and its infection risk.
- **AUA/SUFU 2024** reinforces that behavioural and bladder-training programmes remain foundational and should be offered to all patients, including those proceeding directly to drug or advanced therapy.

Current Practice Update

Point	Textbook	Current practice
Residual measurement	Bladder ultrasound or in–out catheterisation	**Ultrasound preferred**; avoid repeated catheterisation
Catheter removal	Part of a bladder retraining protocol	Also an **essential CAUTI-prevention practice** with nurse-driven removal and stop-orders

Exam One-Liners

- Objective: restore normal voiding and continence after catheter removal.
- Do not clamp the catheter before removal.
- Toilet on waking, 2-hourly by day, before bed, 4-hourly at night.
- Catheterise or scan 6–8 hourly until residuals are ≤200 mL.
- Trigger techniques: running water, inner-thigh stroking, suprapubic tapping.
- Emptying techniques: bending forward, suprapubic pressure, double voiding.
- Persistent difficulty beyond 1–2 weeks → urodynamics and urology referral.

QUESTION 9

Behavioural intervention in urinary incontinence. 7 MARKS

High-Yield Points

1. **Why first-line:** behavioural interventions are **non-invasive, free of drug toxicity, and non-specific** — patients with stress and with urgency incontinence respond about equally well — and are therefore recommended by guidelines as the initial approach for most patients.
2. **The fundamental division:**
   - **Patient-dependent procedures** — need adequate cognition, physical function, learning ability and motivation; the goal is to **restore normal voiding and continence**.
   - **Caregiver-dependent procedures** — for functionally disabled patients; the goal is only to **keep the patient and the environment dry**.
3. **All patient-dependent methods rely on continuous self-monitoring** with a bladder record; caregiver-dependent methods use an institutional bladder-and-bowel monitoring chart.

Procedure	What it is	Types of UI	Comments
Patient-dependent			
Pelvic floor (Kegel) exercises	Repetitive contraction and relaxation of pelvic floor muscles	Stress and urge	**Greatest benefit of all behavioural measures**; biofeedback helps teaching; needs function and motivation
Bladder training	Education + bladder records + pelvic floor and other behavioural techniques	Stress and urge	Needs a trained therapist and a motivated, cognitively intact patient
Bladder retraining	Progressive lengthening or shortening of the voiding interval, with intermittent catheterisation in those recovering from over-distension	Acute — post-catheter, post-stroke, urge or overflow	Goal is to restore a normal voiding pattern
Caregiver-dependent			
Scheduled toileting	Routine toileting at fixed intervals — usually 2-hourly by day, 4-hourly at night	Urge and functional	Prevents wetting; usable in cognitive or physical impairment; needs staff availability
Habit training	Variable toileting schedule tailored to the patient's own voiding pattern	Urge and functional	Requires a monitoring record to define the pattern

Prompted voiding	Offer toileting 2-hourly by day; toilet on request; social reinforcement; routine fluid offering; individualise night-time toileting	Urge, stress, mixed, functional	**Best-studied**; 25–40% of nursing-home residents respond and can be identified by a **3-day trial**

4. **Prompted voiding protocol (assessment phase):** contact the resident **hourly from 7 am to 7 pm for 2–3 days**, then 2-hourly for 2–3 days; focus attention on voiding by asking whether they are wet or dry; check for wetness, record it and give feedback on whether the answer was correct; and whether wet or dry, offer the toilet or urinal.
5. **Adjunctive techniques:** to prompt voiding — running tap water, stroking the inner thigh, suprapubic tapping; to complete emptying — bending forward after voiding, suprapubic pressure, double voiding.
6. **Night-time toileting must be individualised** — prompted voiding at night disrupts sleep; many functionally dependent patients are better managed with pads at night, provided sleep is undisturbed and the skin remains intact.
7. **Success depends on the caregivers, not the patient.** The knowledge, motivation, staffing and administrative support of the institution determine whether prompted voiding is sustained; quality-improvement methods based on statistical process control help maintain it.
8. **Combine with supportive measures:** education, environmental modification (accessible toilet, good lighting, raised seat, commode at the bedside), appropriate toilet substitutes, review of diuretic timing and fluid pattern, and good skin care.
9. **Caveats about pads and garments:** they are a **non-specific** treatment, should not be the first response before a diagnostic evaluation, and should never be used for convenience — many patients are curable and some have serious underlying disease.
10. **Evidence base:** behavioural therapy alone is effective in the geriatric population, and **combined behavioural plus drug therapy is superior to either alone** for overactive bladder symptoms, including in men.

Latest Updates (2023–Present) — Concise

- **AUA/SUFU 2024** keeps behavioural therapy as the foundation of OAB care but no longer requires it to fail before advanced therapy; it should be offered **alongside** any other treatment.
- **Supervised pelvic floor muscle training** retains the strongest evidence for stress and mixed incontinence in women — roughly an eight-fold higher likelihood of cure or improvement than no treatment in Cochrane-level evidence.
- **Digital and tele-rehabilitation delivery** of pelvic floor training and bladder diaries has expanded since 2023, improving access for homebound and rural older adults — directly relevant to home-based geriatric care in India under NPHCE.

Current Practice Update

Point	Textbook	Current practice
Position in the pathway	Initial approach before drugs	Foundation, but offered **with** rather than necessarily before other therapy
Delivery	Clinic-based, therapist-led	

		Supervised programmes plus **tele-rehabilitation and app-based diaries**

Exam One-Liners

- Patient-dependent methods restore continence; caregiver-dependent methods keep the patient dry.
- Pelvic floor muscle exercises give the greatest benefit of all behavioural interventions.
- Prompted voiding is the best studied; 25–40% of nursing-home residents respond; a 3-day trial identifies them.
- Scheduled toileting = fixed interval; habit training = tailored interval.
- Success depends on caregiver motivation and staffing, not on the patient's function.
- Pads are never the first response to incontinence.
- Behavioural + drug therapy beats either alone.

QUESTION 10

Uroflowmetry. 7 MARKS

High-Yield Points

1. **Definition:** uroflowmetry (urine flowmetry) is a **non-invasive urodynamic test** that measures the rate of urine flow over time during a spontaneous void, recorded by a uroflowmeter as a flow-versus-time curve.
2. **Principle:** urine flows when detrusor pressure exceeds urethral resistance. Flow therefore reflects the **interaction of detrusor contractility and outlet resistance** — it cannot separate the two by itself.
3. **Parameters measured: maximum (peak) flow rate — Qmax**, average flow rate, voided volume, time to peak flow, total voiding time, and the shape of the curve.
4. **Key interpretation in older men:** a **Qmax below 10 mL/s on an adequate voided volume of more than 150 mL** suggests either **bladder outlet obstruction** or **impaired detrusor contractility**. A voided volume under 150 mL makes the test uninterpretable.
5. **Curve patterns:** a normal trace is a smooth bell-shaped curve; a **flat, low, prolonged plateau** suggests obstruction (classically urethral stricture); an **intermittent, fluctuating** trace suggests abdominal straining or detrusor–sphincter dyssynergia; a low curve with high residual suggests detrusor underactivity.
6. **Clinical uses:** screening older men with LUTS; assessing severity and monitoring response to medical therapy for BPH; pre-operative assessment before prostatic surgery; evaluating suspected urethral stricture; and assessing voiding function during bladder retraining.
7. **Always interpret with the post-void residual.** Low flow with a high PVR indicates significant obstruction or a poorly contractile bladder; low flow with a low PVR is usually less significant. The pair together is the standard non-invasive assessment.
8. **Limitations:** it is a screening test, not a diagnostic one — it cannot distinguish obstruction from detrusor underactivity, which requires a **pressure-flow (multichannel urodynamic) study**. Results vary between voids, are affected by anxiety and by voided volume, and uroflowmeters, though inexpensive, are uncommon outside urology practices.
9. **Practical points:** the patient should attend with a comfortably full bladder; at least two recordings improve reliability; the test should be performed in privacy in the patient's usual voiding position; artefacts from straining or from the stream striking the funnel edge should be recognised.
10. **Place in geriatric practice:** it is a useful, non-invasive, low-risk bedside adjunct in frail older men in whom invasive urodynamics is undesirable, and it forms part of the recommended evaluation when post-void residual is elevated.

Latest Updates (2023–Present) — Concise

- **Non-invasive uroflow is explicitly endorsed** in the AUA/SUFU OAB Guideline 2024 as the next step when PVR is elevated, before proceeding to invasive urodynamics or cystoscopy.
- **Routine pre-operative multichannel urodynamics is no longer required** before prostate surgery — the UPSTREAM randomised trial across 26 hospitals showed a non-

invasive pathway (symptom score, flow rate, PVR) was non-inferior for symptom outcomes, strengthening the role of uroflowmetry.

- **Home and smartphone-based uroflowmetry** (sound-based and portable devices) has emerged since 2023, improving reliability by averaging multiple domiciliary voids and reducing clinic-related artefact — particularly attractive for frail and rural Indian patients.

Current Practice Update

Point	Textbook	Current practice
Availability	Uroflowmeters rare outside urology practices	**Portable and smartphone-based devices** extend access, including home testing
Role before surgery	Adjunct; urodynamics often performed	Non-invasive pathway (uroflow + PVR) is **non-inferior** — urodynamics used selectively

Exam One-Liners

- Uroflowmetry is non-invasive and measures flow rate against time.
- Qmax <10 mL/s on a void >150 mL is abnormal in older men.
- Voided volume <150 mL makes the test uninterpretable.
- Flat plateau = obstruction; intermittent trace = straining or dyssynergia.
- Always pair the flow rate with a post-void residual.
- It cannot distinguish obstruction from detrusor underactivity — that needs a pressure-flow study.

QUESTION 11

Prostate specific antigen. 7 MARKS

High-Yield Points

1. **Nature:** PSA is a **serine protease (kallikrein-3)** produced by prostatic epithelial cells; its physiological role is to liquefy semen. It is **organ-specific but not cancer-specific**.
2. **Reference value:** the widely used cut-off is **4.0 ng/mL**. However, **about 25% of men with prostate cancer have a PSA of 4.0 ng/mL or less**, and PSA specificity is lower in men with obstructive symptoms than in asymptomatic men.
3. **Causes of a raised PSA other than cancer:** benign prostatic hyperplasia, prostatitis and male urinary tract infection, acute retention, catheterisation and instrumentation, prostate biopsy, digital rectal examination and prostatic massage, ejaculation, and vigorous cycling.
4. **Causes of a falsely low PSA: 5-α-reductase inhibitors halve the value — double the measured PSA** for interpretation; also obesity (haemodilution) and some herbal preparations.
5. **Refinements to improve specificity:** PSA density (PSA ÷ prostate volume), age-specific reference ranges, PSA velocity, free-to-total PSA ratio, and PSA doubling time (a doubling time <10 months indicates aggressive non-metastatic castration-resistant disease).
6. **Role in BPH:** PSA measurement remains recommended in the evaluation of LUTS by AUA guidance, and **serum PSA is a stronger predictor of future prostate growth than age or baseline prostate volume**, making it a useful predictor of acute urinary retention and of who will benefit from a 5-α-reductase inhibitor.
7. **Role in cancer:** used for detection (with DRE and imaging), risk stratification, monitoring after radical treatment (biochemical recurrence), and monitoring response in advanced disease.
8. **Screening in older adults — the key geriatric point:** guidance applies to men with a **life expectancy of more than 10 years**. Screening should follow **shared decision-making**, weighing over-diagnosis, over-treatment and biopsy complications against a modest mortality benefit; it is generally not appropriate in frail elders or those with limited life expectancy.
9. **Do not measure PSA during a male urinary tract infection** — the value is unreliable and takes about **six months** to return to baseline after infection.
10. **Indian context:** report PSA in **ng/mL**. There is no organised national prostate cancer screening programme in India; opportunistic testing without counselling risks over-diagnosis, and prostate cancer often presents late with bone metastases and obstructive uropathy in Indian practice.

Latest Updates (2023–Present) — Concise

- **AUA/SUO Early Detection of Prostate Cancer Guideline (2023, updated 2026)** anchors screening in shared decision-making and integrates **secondary biomarkers and prostate MRI before biopsy** to reduce unnecessary biopsies and detection of indolent disease.
- **The Stockholm3 multi-analyte risk test**, in the STHLM3 and STHLM3-MRI randomised screening studies, has shown greater clinical net benefit than PSA alone —

fewer false negatives, fewer biopsies and less over-diagnosis, with 9-year outcome data (2025) confirming improved detection of aggressive cancers.

- **MRI-first pathways** with targeted biopsy are now standard where available, substantially reducing detection of clinically insignificant (Grade Group 1) cancer while maintaining detection of significant disease.

Current Practice Update

Point	Textbook	Current practice
Diagnostic pathway	Raised PSA → systematic biopsy	**PSA → risk calculator / biomarker → MRI → targeted biopsy**
Screening decision	Controversial; life expectancy >10 years	Unchanged in principle, but formalised as **shared decision-making** with biomarker and MRI refinement
Beyond total PSA	Free/total ratio, density, velocity	Add **multi-analyte tests such as Stockholm3** and MRI-based risk stratification

Exam One-Liners

- PSA is kallikrein-3 — organ-specific, not cancer-specific.
- Cut-off 4.0 ng/mL, but 25% of prostate cancers have a PSA at or below this.
- 5-α-reductase inhibitors halve PSA — double the value.
- Do not check PSA during a male UTI; allow six months for recovery.
- PSA predicts prostate growth and acute retention better than age or volume.
- Screening only where life expectancy exceeds 10 years, after shared decision-making.
- Modern pathway: PSA → biomarker/MRI → targeted biopsy.

QUESTION 12

Obstructive uropathy — clinical features, investigation and treatment. 15 MARKS

High-Yield Points

A. Definitions and Importance

1. **Obstructive uropathy** is structural or functional obstruction to urine flow anywhere from the renal calyces to the external urethral meatus. When it causes renal parenchymal damage it is termed **obstructive nephropathy**; dilatation of the collecting system is **hydronephrosis**.
2. **Geriatric significance:** it is one of the most important and **potentially reversible** causes of renal failure in old age, and is the classic **post-renal cause of acute kidney injury** alongside stones, tumour and stricture.
3. **The central geriatric trap:** prostatic enlargement produces a **slow, silent obstruction** that gradually reduces kidney function and is often **not recognised until it is too late — largely because the patient becomes polyuric rather than oliguric**. By the time obstruction is diagnosed the damage may be irreversible, and renal function recovers only partially even after relief.
4. **It also drives infection:** the pathogenesis of pyelonephritis in older adults is strongly related to obstructive uropathy, and prostatic hypertrophy with obstruction predisposes older men to urinary tract infection.

B. Causes

5. **Lower tract (commonest in the elderly):** benign prostatic hyperplasia, prostate carcinoma, urethral stricture, bladder neck stenosis, bladder calculi, blocked indwelling catheter, phimosis or meatal stenosis, and severe pelvic organ prolapse or cystocele in women.
6. **Upper tract:** renal or ureteric calculi, urothelial tumours, extrinsic compression by pelvic or retroperitoneal malignancy (cervical, ovarian, colorectal, lymphoma), retroperitoneal fibrosis, and post-radiotherapy or post-surgical ureteric stricture.
7. **Functional (neurogenic) obstruction:** detrusor–sphincter dyssynergia in suprasacral cord lesions and multiple sclerosis; acontractile bladder in diabetic autonomic neuropathy; and **drug-induced retention** (anticholinergics, opioids, tricyclics, antipsychotics, α-agonists, antihistamines).
8. **Faecal impaction** is a common, easily reversible precipitant of outlet obstruction in frail older patients.

C. Clinical Features

9. **Acute lower tract obstruction (acute urinary retention):** sudden inability to pass urine, severe suprapubic pain, a palpable tender bladder — and in the older patient it may present **atypically as delirium, agitation, a fall or "off legs"**. Acute retention has a quality-of-life impact comparable to that of myocardial infarction.
10. **Chronic lower tract obstruction:** insidious LUTS — hesitancy, weak stream, straining, intermittency, incomplete emptying, terminal dribbling — with **overflow (dribbling) incontinence**, a painless distended bladder, and nocturnal enuresis. Pain is characteristically absent.

11. **Acute upper tract obstruction:** loin pain radiating to the groin (ureteric colic), nausea, vomiting, haematuria, and fever if infected.
12. **Chronic upper tract obstruction:** often silent; presents with anaemia, nocturia, polyuria, hypertension or unexplained rising creatinine.
13. **Complications:** post-renal AKI and chronic kidney disease; recurrent UTI, pyelonephritis and **pyonephrosis with sepsis**; bladder stones and diverticula; hyperkalaemia and metabolic acidosis (type 4 renal tubular acidosis); hypertension; and, after relief, **post-obstructive diuresis**.
14. **Danger combination to recognise instantly:** obstruction plus fever plus a raised inflammatory response = **infected obstructed system**, a urological emergency requiring drainage within hours.

D. Investigation

15. **Bedside:** abdominal examination for a distended bladder, digital rectal examination, **bladder ultrasound for post-void residual**, and urinalysis (infection, haematuria).
16. **Laboratory:** serum **creatinine and urea (mg/dL)**, electrolytes including potassium (mEq/L), bicarbonate, calcium (mg/dL), haemoglobin (g/dL), and blood glucose (mg/dL); urine culture; **PSA (ng/mL)** once infection and retention have resolved.
17. **Ultrasound of kidneys, ureters and bladder** is the **first-line imaging investigation** — it detects hydronephrosis, measures residual urine and prostate size, is safe, cheap, radiation-free and requires no contrast. Remember that hydronephrosis may be absent in early obstruction, in dehydration, and in retroperitoneal fibrosis or encasing malignancy.
18. **Non-contrast CT KUB** for suspected calculus and for defining the level and cause of upper-tract obstruction.
19. **Further tests as indicated:** MR urography where contrast is contraindicated; **diuretic renography (MAG-3)** to confirm functional obstruction and assess split renal function; antegrade or retrograde pyelography; **cystoscopy** for stricture, bladder tumour or stone; and **urodynamics/pressure-flow study** to distinguish true obstruction from detrusor underactivity.
20. **Uroflowmetry** as a non-invasive screen — Qmax <10 mL/s on a voided volume >150 mL supports obstruction or impaired contractility.

E. Treatment

21. **Principle: relieve the obstruction promptly, treat infection, correct metabolic consequences, then treat the cause.**
22. **Acute retention:** immediate **urethral catheterisation**; if this fails or is contraindicated (stricture, recent urethral surgery), **suprapubic catheterisation**. Record the drained volume. Start an α-blocker before a trial without catheter, which improves the chance of successful voiding.
23. **Upper-tract obstruction:** decompress by **percutaneous nephrostomy** or **retrograde ureteric stenting** — nephrostomy is generally preferred in sepsis and in a poorly drained infected system.
24. **Infected obstructed kidney (pyonephrosis) is an emergency:** resuscitate, take cultures, start broad-spectrum intravenous antibiotics and drain urgently; delay is associated with high mortality.
25. **Correct metabolic derangement:** hyperkalaemia, acidosis and uraemia; dialysis if refractory.

26. **Anticipate post-obstructive diuresis** after relief of chronic obstruction — monitor urine output hourly, weigh daily, and replace fluid and electrolytes carefully; it is usually physiological but may cause hypovolaemia, hyponatraemia, hypokalaemia and hypomagnesaemia in frail older patients.
27. **Definitive treatment of the cause:** medical therapy for BPH (α-blocker ± 5-α-reductase inhibitor) or **TURP / laser enucleation**; stone removal by ureteroscopy or lithotripsy; urethral dilatation or urethroplasty for stricture; oncological treatment or long-term stenting for malignant compression; steroids and ureterolysis for retroperitoneal fibrosis; clean intermittent self-catheterisation for a neurogenic acontractile bladder; and disimpaction plus a bowel regimen where faecal loading is contributory.
28. **Review and stop precipitating drugs** — anticholinergics, opioids, tricyclics, antipsychotics and α-agonists.
29. **Prognosis:** recovery of renal function depends on the duration and completeness of obstruction and on the pre-existing renal reserve; partial recovery is the rule after prolonged obstruction, so **early detection is the only effective strategy**. Follow up renal function and residual volume after relief.
30. **Geriatric caveat:** in a frail patient with advanced malignancy, decompression may prolong a poor-quality life — the decision to stent or drain should follow comprehensive geriatric assessment and a discussion of goals of care.

Latest Updates (2023–Present)

- **KDIGO AKI guidance and current practice** emphasise urgent exclusion of a post-renal cause in every episode of AKI, with **bedside bladder scan and renal ultrasound as immediate steps** — the most rapidly reversible AKI aetiology in older adults.
- **Point-of-care ultrasound (POCUS)** performed by physicians has become routine on acute geriatric and emergency units since 2023, shortening the time to detection of retention and hydronephrosis; a bladder scan is now expected before labelling an older patient's AKI as pre-renal.
- **Minimally invasive surgical therapies for BPH** — water vapour thermal therapy, prostatic urethral lift, and **prostatic artery embolisation (added to the AUA guideline in the 2023 amendment)** — allow definitive relief of prostatic obstruction in frail men unfit for anaesthesia, replacing indefinite long-term catheterisation in selected patients.
- **Laser enucleation (HoLEP/ThuLEP)** is now preferred over open prostatectomy for very large glands, with lower morbidity, shorter catheter time and size-independent efficacy.
- **Nephrostomy versus stent in malignant obstruction:** contemporary evidence supports individualised choice with early palliative-care input, since survival after decompression for advanced pelvic malignancy is often short — an important shared-decision point in geriatric oncology.
- **Indian context:** obstructive uropathy from BPH, stone disease and cervical or prostate malignancy remains a leading reversible cause of CKD in Indian older adults; TURP, ureteric stenting and nephrostomy are covered under AB-PMJAY, with the **Ayushman Vay Vandana Card** providing universal cover for those aged 70 and above.

Current Practice Update

Issue	Textbook position	Current evidence / practice
Detection of obstruction in AKI	Consider post-renal causes; ultrasound	**Bedside POCUS bladder scan first**, then formal ultrasound — expected within the first hour
Frail men with retention unfit for TURP	Long-term catheter or open surgery	**MISTs and prostatic artery embolisation** offer catheter-free options
Very large prostate	Open prostatectomy	**HoLEP / ThuLEP** preferred where available
Malignant ureteric obstruction	Decompress	Decompress **after goals-of-care discussion**; survival may be short

15-second answer skeleton: Define obstructive uropathy / nephropathy / hydronephrosis → geriatric importance as reversible renal failure and the polyuria trap → causes (lower, upper, functional, faecal impaction) → clinical features (acute retention incl. atypical presentation; chronic painless distended bladder with overflow; upper tract colic vs silent) → complications incl. infected obstruction and post-obstructive diuresis → investigation (bladder scan, urinalysis, creatinine/K^+, **ultrasound first-line**, CT KUB, renography, cystoscopy, urodynamics) → treatment (relieve first: catheter/suprapubic/nephrostomy/stent → treat sepsis → correct metabolic derangement → anticipate post-obstructive diuresis → definitive cause-specific therapy → stop culprit drugs) → prognosis and goals of care.

Examiner traps — (1) Chronic obstruction causes **polyuria, not oliguria** — this is the classic missed diagnosis. (2) Absent hydronephrosis does not exclude obstruction (early obstruction, dehydration, retroperitoneal fibrosis, encasing tumour). (3) Never delay drainage in an infected obstructed system for imaging or antibiotics alone. (4) Always warn about post-obstructive diuresis. (5) Painless distended bladder with dribbling is chronic retention, not "incontinence" to be treated with an antimuscarinic.

Exam One-Liners

- Obstructive uropathy is the most readily reversible cause of renal failure in the elderly.
- Chronic prostatic obstruction presents with polyuria, not oliguria — hence late diagnosis.
- Acute retention in an older person may present as delirium or a fall.
- Ultrasound KUB is the first-line investigation; absent hydronephrosis does not exclude obstruction.
- Infected obstructed kidney = emergency drainage plus intravenous antibiotics.
- Anticipate post-obstructive diuresis after relieving chronic obstruction.
- Recovery of renal function is only partial after prolonged obstruction.

QUESTION 13

Chronic indwelling catheter usage in the elderly. 7 MARKS

High-Yield Points

1. **General rule:** catheters should be **avoided in the management of incontinence unless specific indications are present**. Indwelling catheterisation remains substantially overused in acute hospitals.
2. **Three types of catheterisation: external (condom)** — for intractable incontinence in a physically dependent man **without retention**; **intermittent straight catheterisation** — 2–4 times daily, keeping bladder volume below **400 mL**, clean (not necessarily sterile) technique at home; and **chronic indwelling catheterisation**.
3. **Indications for a chronic indwelling catheter (memorise this list):**
   - Urinary retention that is causing persistent overflow incontinence, symptomatic infection or renal dysfunction, **and** cannot be corrected surgically or medically, **and** cannot practically be managed with intermittent catheterisation
   - Skin wounds, pressure ulcers or irritation being contaminated by incontinent urine
   - Care of terminally ill or severely impaired patients for whom bed and clothing changes are uncomfortable or disruptive
   - Patient preference where toileting or changing causes excessive discomfort
4. **Complications:** chronic bacteriuria (universal), symptomatic UTI and urosepsis, bladder and renal stones, periurethral and prostatic abscess, urethral trauma, erosion and fistula, catheter blockage and bypassing, pyelonephritis, and — with long-term use — **bladder carcinoma**. Catheters also promote immobility, falls (tethering), delirium and loss of dignity.
5. **Key principles of catheter care — part 1 (system):** maintain a **sterile closed gravity-drainage system**; avoid breaking the closed system; use clean technique when emptying or changing bags and wash hands between patients; secure the catheter to the upper thigh or lower abdomen to prevent perineal contamination and urethral traction injury.
6. **Key principles — part 2 (hygiene and mechanics):** avoid frequent or vigorous cleaning of the catheter entry site — washing with soapy water once a day is sufficient; **do not routinely irrigate**; if bypassing occurs without obstruction, consider bladder spasm and treat with a bladder relaxant; if blockage is frequent, increase fluid intake and acidify the urine with dilute acetic acid irrigation.
7. **Key principles — part 3 (antibiotics and cultures): do not use routine prophylactic or suppressive urinary antiseptics or antimicrobials**; **do not perform surveillance cultures**, because all chronically catheterised patients have bacteriuria which is often polymicrobial and changes frequently; and **do not treat infection unless the patient develops symptoms**.
8. **Interpreting symptoms:** symptoms in catheterised older patients are non-specific (fever, delirium, functional decline); other sources of infection must be carefully excluded before attributing them to the urinary tract.
9. **When to investigate further:** a patient with **frequent symptomatic UTIs** should have genitourinary evaluation to exclude stones, periurethral or prostatic abscess, and chronic pyelonephritis.

10. **Alternatives to prefer:** accessible toilets and toilet substitutes, scheduled toileting, intermittent catheterisation, external catheters in selected men, and highly absorbent launderable or disposable pads — these carry less morbidity and lower overall cost than an indwelling catheter.

Latest Updates (2023–Present) — Concise

- **SHEA/IDSA/APIC/AHA/Joint Commission CAUTI Compendium, 2022 Update (published 2023)** makes **avoidance of unnecessary catheters and prompt removal** essential practices, with nurse-driven removal protocols, automatic stop-orders and daily necessity review; it also formalises **urine-culture stewardship** to stop reflex culturing of catheterised patients.
- The same document addresses routine catheter replacement for catheters in place beyond **30 days** — scheduled change is a standard-of-care question rather than an infection-prevention intervention, and catheters should not be changed reflexively to "treat" bacteriuria.
- **IDSA guidance continues to advise against screening for or treating asymptomatic bacteriuria** in catheterised older adults; antibiotic use in this setting drives resistance without benefit — a particularly pressing issue given ICMR AMR surveillance data on carbapenem-resistant Enterobacterales in Indian hospitals.

Current Practice Update

Point	Textbook	Current practice
Removal	Limit use to defined indications	Add **nurse-driven removal protocols, stop-orders and daily necessity review**
Cultures	Do not perform surveillance cultures	Formalised as **urine-culture stewardship** with diagnostic criteria before culturing
Antiseptic / antibiotic prophylaxis	Not recommended	Unchanged; also no routine antimicrobial-coated catheters as a standard measure

Extra Points to Remember (High-Yield)

- **Four indications, in one line: retention that cannot be fixed otherwise · contaminated wounds or pressure ulcers · terminal or severely impaired care comfort · patient preference where toileting is intolerable.**
- **Three "do nots" that earn marks:** do not irrigate routinely · do not send surveillance cultures · do not give prophylactic antibiotics.
- **Numbers:** intermittent catheterisation 2–4 times daily, bladder volume <400 mL; catheters in place >30 days raise the replacement question; entry-site washing once daily.
- **Convenience is never an indication** — for catheters, condom drainage, pads or garments.

Exam One-Liners

- Catheters are for specific indications, never for convenience.

- Every chronically catheterised patient has bacteriuria — do not culture or treat it unless symptomatic.
- Maintain a closed sterile drainage system and secure the catheter to prevent traction injury.
- No routine irrigation, no routine prophylactic antibiotics, no surveillance cultures.
- Bypassing without obstruction suggests bladder spasm.
- Long-term catheterisation risks stones, abscess and bladder cancer.
- Frequent symptomatic UTIs warrant genitourinary evaluation for stones or abscess.

QUESTION 14

Catheter-associated urinary tract infections — prevention and management. 7 MARKS

High-Yield Points

1. **Definitions: catheter-associated bacteriuria** is almost universal with long-term catheters and is usually asymptomatic and polymicrobial. **CAUTI** is bacteriuria **plus signs or symptoms** attributable to the urinary tract with no other identified source, in a patient with an indwelling catheter in place (or removed within the previous 48 hours).
2. **Burden:** urinary tract infection is among the commonest healthcare-associated infections, and historically **70–80% of hospital UTIs were attributable to an indwelling urethral catheter**. CAUTI rates in low- and middle-income countries, including India, consistently exceed those reported from high-income settings.
3. **Pathogenesis:** organisms ascend extraluminally along the catheter–urethral interface or intraluminally from a broken drainage system, and establish a **biofilm** on the catheter surface that protects them from antibiotics and host defences. Risk rises with **each day of catheterisation**.
4. **Microbiology:** *Escherichia coli*, *Klebsiella*, *Proteus* (urease-producing, causes encrustation and blockage), *Pseudomonas*, *Enterococcus*, and *Candida*. Multidrug-resistant Gram-negative organisms, including ESBL producers and carbapenem-resistant Enterobacterales, dominate in Indian hospital practice.
5. **Clinical features in older adults are non-specific:** fever, new or worsening delirium, functional decline, falls, reduced oral intake, rigors, suprapubic or flank pain, or haematuria. Classical dysuria and frequency are often absent because the catheter bypasses the urethra.
6. **Prevention — the four essential principles:**
   - **Avoid unnecessary catheterisation** — insert only for defined indications; use bladder scanning, toileting programmes, external catheters and pads instead.
   - **Insert aseptically** by trained personnel, using the smallest suitable catheter and sterile technique.
   - **Maintain a closed sterile drainage system**, keep the bag below bladder level and off the floor, secure the catheter, avoid kinking, and perform hand hygiene before and after any catheter contact.
   - **Remove as early as possible** — daily review of continuing need, nurse-driven removal protocols and automatic stop-orders.
7. **What does NOT prevent CAUTI:** routine bladder irrigation, routine antiseptic or antibiotic instillation, routine systemic antibiotic prophylaxis, routine catheter changes to "clear" bacteriuria, vigorous daily meatal cleaning with antiseptics, and cranberry products.
8. **Diagnostic (urine-culture) stewardship:** **do not send reflex urine cultures** from catheterised patients without genuine urinary signs or symptoms; culture only when clinical criteria are met, and exclude other sources of fever first. Treating asymptomatic bacteriuria offers no benefit and drives resistance.
9. **Management of established CAUTI:** obtain cultures **after** replacing a catheter that has been in place for more than two weeks (a fresh specimen from a new catheter reflects bladder rather than biofilm flora); remove the catheter altogether if it is no longer needed;

start empirical therapy guided by local antibiogram and severity, then de-escalate on culture and sensitivity.

10. **Duration and supportive care:** typically **7 days** for prompt responders and **10–14 days** for delayed response or bacteraemia; ensure hydration, correct electrolytes, exclude obstruction, abscess or stones if the response is poor, and review the ongoing need for catheterisation before discharge.

Latest Updates (2023–Present) — Concise

- **SHEA/IDSA/APIC/AHA/Joint Commission Compendium — CAUTI 2022 Update (published 2023)** reorganises measures into **essential practices** (avoidance, aseptic insertion, closed system maintenance, early removal, urine-culture stewardship, surveillance and feedback) and **additional approaches** for outbreaks or persistent high rates; it also addresses routine replacement of catheters in place beyond 30 days and extends guidance to paediatric settings.
- **Urine-culture (diagnostic) stewardship is now a named essential practice** — reducing inappropriate culturing has been shown to reduce reported CAUTI rates and unnecessary antibiotic use without patient harm.
- **An ISID guidance chapter (2024) adapts CAUTI prevention for low- and middle-income countries**, noting persistently higher INICC-reported rates and emphasising bundle implementation, catheter-day reduction and antimicrobial stewardship — directly applicable to Indian district hospitals and NABH accreditation requirements.

Current Practice Update

Point	Textbook	Current practice
Prevention framework	Principles of catheter care	Structured **CAUTI bundle** with essential practices and outcome feedback
Cultures	Do not do surveillance cultures	**Urine-culture stewardship** as a formal, audited practice
Catheter change before treatment	Not specified	**Replace a catheter in situ >2 weeks, then culture** and treat

Extra Points to Remember (High-Yield)

- **Mnemonic for the bundle — "AIMS": A**void unnecessary catheters · **I**nsert aseptically · **M**aintain a closed system · **S**top early (remove promptly).
- **Numbers:** historically 70–80% of hospital UTIs are catheter-related; risk increases with each catheter-day; replace catheters in place >2 weeks before culturing; treat 7 days (prompt response) or 10–14 days (delayed response); catheters >30 days raise the replacement question.
- **The single highest-impact intervention is removing the catheter** — say this explicitly.

Examiner traps — (1) Do not treat asymptomatic catheter-associated bacteriuria. (2) Do not send a urine culture simply because the urine is cloudy or malodorous. (3) Do not rely on dysuria and frequency — delirium and functional decline are the geriatric presentation. (4) Do not use

antibiotic prophylaxis or routine irrigation to prevent CAUTI. (5) Exclude obstruction, stones and abscess when a CAUTI fails to respond.

Exam One-Liners

- CAUTI = bacteriuria plus attributable symptoms; bacteriuria alone is not infection.
- Biofilm formation on the catheter is the central pathogenic mechanism.
- Risk rises with every catheter-day; early removal is the most effective prevention.
- AIMS bundle: Avoid, Insert aseptically, Maintain closed system, Stop early.
- Delirium or unexplained functional decline may be the only sign in an older patient.
- Replace a long-standing catheter before taking the culture, then treat.
- Urine-culture stewardship is now an essential prevention practice.

QUESTION 15

Recommendations for treatment of complicated urinary tract infection in males. 7 MARKS

High-Yield Points

1. **Principle: urinary tract infection in a male is, by convention, complicated**. Contemporary practice replaces the term "acute prostatitis" with **male urinary tract infection**, managed as a "urinary infection at risk of complication". A lower UTI in a man is treated as prostatic infection.
2. **Why complicated:** older men have obstructive uropathy from prostatic enlargement, high residual urine, catheters, instrumentation, diabetes and structural abnormality — all of which predispose to relapse, abscess and bacteraemia.
3. **Microbiology:** Gram-negative bacilli predominate — *Escherichia coli* and *Proteus* species most commonly; also *Klebsiella*, *Pseudomonas*, enterococci and staphylococci. Recurrence after completing therapy is usually with the **same organism**.
4. **Presentation ranges widely** — from paucisymptomatic cystitis-like illness to spiking fever, chills, malaise, myalgia, dysuria, pelvic or perineal pain and cloudy urine. In older men, delirium, falls and functional decline may dominate.
5. **Diagnosis:** gentle digital rectal examination showing an oedematous tender prostate supports the diagnosis — **vigorous prostatic massage must be avoided** because it is painful, adds nothing diagnostically and risks bacteraemia. A dipstick positive for leukocytes or nitrites has a positive predictive value above 90% in a symptomatic man. Send **urine Gram stain and culture in every case**; the diagnostic threshold is **≥10^3 CFU/mL with leukocyturia ≥10^4/mL**. Take **blood cultures when there is fever**. **Do not measure PSA during a male UTI** — it needs about six months to return to baseline.
6. **Pharmacological principle:** the barrier between the prostatic microcirculation and stroma permits only **non-protein-bound, lipophilic** antimicrobials to diffuse in, and the low pH of prostatic fluid allows drugs with **alkaline pKa** — quinolones and sulfonamides — to concentrate in prostatic tissue. Penetration improves in the presence of inflammation.
7. **Route and setting:** ambulatory, pauci-symptomatic men may be treated **orally, guided by susceptibility results**. Men with fever or signs of sepsis need **hospitalisation and urgent empirical parenteral therapy**, because Gram-negative bacteraemia can progress abruptly to life-threatening septic shock.
8. **Antimicrobial choice:**
   - **Gram-negative bacilli, oral therapy:** trimethoprim–sulfamethoxazole or a fluoroquinolone. Other agents with good to excellent prostatic penetration are tetracyclines, macrolides, sulfonamides and nitrofurantoin.
   - **Parenteral therapy:** an aminoglycoside combined with an intravenous cephalosporin or a fluoroquinolone; treat as Gram-negative infection until further culture data are available.
   - **Gram-positive cocci in chains** → enterococcal infection → amoxicillin.
   - **Gram-positive cocci in clusters** → *Staphylococcus aureus* or coagulase-negative staphylococci; when *S. aureus* is isolated from urine, **always take blood cultures** to confirm that the bacteriuria reflects local infection rather than seeding from bacteraemia.

9. **Source control and supportive care:** relieve obstruction (catheter or suprapubic drainage; nephrostomy or stent for upper-tract obstruction), remove or replace an infected catheter, drain a prostatic or perinephric abscess, maintain hydration, adjust doses for renal function, monitor for delirium and falls, and avoid nephrotoxins.
10. **Duration and follow-up:** treat for a prolonged course reflecting prostatic involvement — commonly **10–14 days**, extended to several weeks where prostatic infection is established, and up to **4–6 weeks** for chronic bacterial prostatitis. Investigate the urinary tract after the episode (ultrasound, PVR, and PSA deferred by about six months) and treat the underlying obstruction to prevent relapse.

Latest Updates (2023–Present) — Concise

- **New agents for complicated UTI: cefepime–enmetazobactam** was FDA-approved in 2024 for complicated UTI including pyelonephritis, showing non-inferiority and superiority to piperacillin–tazobactam for clinical cure and microbiological eradication; **cefepime–taniborbactam** demonstrated superiority over meropenem in the phase-3 **CERTAIN-1** trial (70.6% vs 58.0% composite response) but has faced regulatory delay on manufacturing grounds. **Ceftazidime–avibactam, ceftolozane–tazobactam, meropenem–vaborbactam, plazomicin and cefiderocol** remain the mainstay options for multidrug-resistant Gram-negative complicated UTI.
- **Gepotidacin (Blujepa)**, approved March 2025, is the first new oral antibiotic class for urinary infection in almost thirty years — but its licence is for **uncomplicated UTI in females aged ≥12 years**, not for complicated male UTI; it is a common examination distractor.
- **Fluoroquinolone restriction** continues: regulatory warnings on tendinopathy, aortic aneurysm, neuropathy, delirium and dysglycaemia, together with high Indian resistance rates, mean quinolones should be **guided by culture rather than used empirically** in older men. ICMR AMR surveillance shows very high fluoroquinolone and third-generation cephalosporin resistance among urinary *E. coli* isolates in India, with carbapenem-sparing strategies now a national priority under the ICMR treatment guidelines and NAP-AMR.

Current Practice Update

Point	Textbook	Current practice
Empirical oral therapy	Trimethoprim–sulfamethoxazole or fluoroquinolone	**Culture-guided**; empirical quinolones discouraged given Indian resistance and toxicity warnings
Resistant Gram-negative cUTI	Aminoglycoside + cephalosporin	**Newer β-lactam/β-lactamase inhibitors** — cefepime–enmetazobactam, ceftazidime–avibactam, meropenem–vaborbactam, cefiderocol
Terminology	Acute prostatitis	**Male urinary tract infection**, managed as complicated UTI

Exam One-Liners

- Every UTI in a man is complicated; a lower UTI in a man is treated as prostatic infection.
- Diagnostic threshold: ≥10^3 CFU/mL with leukocyturia ≥10^4/mL.

- Avoid vigorous prostatic massage — risk of bacteraemia.
- Do not check PSA during a male UTI; wait about six months.
- Only lipophilic, non-protein-bound, alkaline-pKa drugs penetrate the prostate.
- Fever or sepsis → admit and give parenteral therapy urgently.
- *S. aureus* in the urine → take blood cultures.
- Treat 10–14 days, longer for established prostatic infection; 4–6 weeks for chronic bacterial prostatitis.
- Gepotidacin is licensed for uncomplicated UTI in women only — a classic distractor.

RAPID REVISION

Night-Before Master Sheet — Numbers, Mnemonics and Traps

Numbers Worth Memorising

Parameter	Value
UI prevalence >65 years	Women ~1 in 3 · Men 15–20% · Nursing homes 60–80%
OAB symptoms at ≥75 years	Women ~30% · Men ~40%
Normal bladder capacity / first urge / filling pressure	300–600 mL / 150–300 mL / <15 cm H_2O
Frequency / nocturia definitions	>8 voids per 24 h · ≥2 voids at night
Abnormal post-void residual	>200 mL
Abnormal peak urine flow	Qmax <10 mL/s on a voided volume >150 mL
Intermittent catheterisation	2–4 times daily; keep bladder volume <400 mL
IPSS / AUA-SI severity	Mild 0–7 · Moderate 8–19 · Severe 20–35
5-α-reductase inhibitor	Gland >30–40 mL · up to 30% volume reduction · 3–6 months to act · halves PSA
MTOPS — reduction in acute retention	Combination 66% · α-blocker 39% · 5-ARI 34%
PSA	Cut-off 4.0 ng/mL; 25% of cancers below it; 6 months to normalise after UTI
Prompted voiding response	25–40% of nursing-home residents; identified by a 3-day trial
Bladder retraining schedule	On waking · 2-hourly day/evening · before bed · 4-hourly at night
Male UTI diagnostic threshold	≥10^3 CFU/mL with leukocyturia ≥10^4/mL
CAUTI attribution / treatment duration	70–80% of hospital UTIs catheter-related · 7 days prompt response, 10–14 days delayed
Fibre for constipation	4–6 g/day of crude fibre

Mnemonics

- **DRIP** — Delirium · Restricted mobility/Retention · Infection, Inflammation, Impaction · Polyuria, Pharmaceuticals
- **DIAPPERS** — Delirium · Infection · Atrophic vaginitis · Pharmaceuticals · Psychological · Excess urine output · Restricted mobility · Stool impaction
- **A-5-C-A-P** (BPH medical therapy) — Alpha-blocker · 5-ARI · Combination · Anticholinergic/β3 · PDE-5 inhibitor
- **AIMS** (CAUTI bundle) — Avoid · Insert aseptically · Maintain closed system · Stop early
- **PROSTATE** (male UI causes) — Prostatic obstruction · Radiotherapy/Radical prostatectomy · Overactive bladder · Stricture · Tumour · Autonomic neuropathy · Toxins/drugs · Environment

Ten Traps That Cost Marks

1. Saying ageing *causes* incontinence — it only predisposes.
2. Omitting the post-void residual from the basic evaluation.

3. Treating asymptomatic bacteriuria in a catheterised or stable institutionalised patient.
4. Prescribing oral rather than topical oestrogen.
5. Adding an antimuscarinic in a man without checking the residual (DHIC and retention).
6. Forgetting that 5-α-reductase inhibitors halve PSA.
7. Missing that chronic obstructive uropathy causes **polyuria, not oliguria**.
8. Forgetting post-obstructive diuresis after relieving chronic retention.
9. Measuring PSA during an acute male urinary tract infection.
10. Using a catheter, condom drainage or pads for convenience rather than for an indication.

Prepared as an examination revision aid. Clinical decisions must always be individualised, and local antibiograms and institutional protocols should guide antimicrobial choice.

METABOLIC BONE DISEASE & GERIATRIC RHEUMATOLOGY

High-Yield Examination Compendium

MD Geriatric Medicine — Written Examination Revision

Primary sources: Pathy's Principles and Practice of Geriatric Medicine, 6th edition (Sinclair, Morley, Vellas, Cesari, Munshi; Wiley 2022) · Kane's Essentials of Clinical Geriatrics.

Supplementary evidence: Guidelines, regulatory approvals and randomised trials published 2023 – present, presented in clearly separated blocks.

Units: Indian conventional units throughout (calcium mg/dL, creatinine mg/dL, phosphate mg/dL, haemoglobin g/dL, PSA ng/mL, glucose mg/dL).

Coverage: 14 questions — 10 long answers (15 marks) and 4 short notes (7 marks).

Compiled for Dr. Sudarsan · Structured for night-before-examination revision

Contents

1	Osteoporosis — risk factors, pathology, clinical features, investigations, management, complications **(recurs)**	**15 marks**
2	68-year-old post fracture neck of femur — further fragility fracture risk, tests, treatment	**15 marks**
3	DEXA scan and its clinical use **(recurs)**	**7 marks**
4	FRAX score	**7 marks**
5	Selective estrogen receptor modulators	**7 marks**
6	Atypical femur fractures due to drugs	**7 marks**
7	Renal osteodystrophy — laboratory and radiological abnormalities, treatment	**15 marks**
8	Osteoarthritis — management	**15 marks**
9	Polymyalgia rheumatica **(recurs ×2)**	**15 marks**
10	Temporal arteritis (giant cell arteritis) **(recurs ×2)**	**15 marks**
11	Late-onset rheumatoid arthritis	**15 marks**
12	Sjögren's syndrome	**15 marks**
13	Inflammatory myopathies	**15 marks**
14	Psoriatic arthropathy	**15 marks**

Q1. Osteoporosis — Risk Factors, Pathology, Clinical Features, Investigations, Management and Complications

15 MARKS

A. Definition and Burden

1. **Osteoporosis** is a skeletal disease of **low bone mass and micro-architectural deterioration** of bone tissue, causing bone fragility and increased fracture risk. Operational (WHO) definition: **BMD T-score ≤ −2.5** at spine or hip.
2. It is a **major contributor to the global burden of disease and disability**; maintenance of musculoskeletal health during ageing is a key determinant of functional ability. The commonest fragility fractures are **hip, forearm (Colles') and vertebral**.
3. Indian context: LASI wave 1 shows a high prevalence of low bone mass in adults ≥60 years; ISBMR recommends Indian reference databases, since using Caucasian norms over-diagnoses osteoporosis in Indians.

B. Risk Factors (Major Risk Factors for Fragility Fracture — Pathy's list)

4. **Non-modifiable:** advancing **age**, female **sex**, **prior fragility fracture**, **parental hip fracture history**, height loss **>4 cm**.
5. **Modifiable / lifestyle: low BMI**, **excess alcohol**, **smoking**, immobility, low calcium and vitamin D intake, low protein intake.
6. **Drugs: glucocorticoid therapy** (most important), aromatase inhibitors, androgen-deprivation therapy, anticonvulsants, proton-pump inhibitors, thyroxine over-replacement.
7. **Causes of secondary osteoporosis:** rheumatoid arthritis, hyperthyroidism, primary hyperparathyroidism, hypogonadism, diabetes, inflammatory bowel disease, anorexia nervosa, organ transplantation, prolonged immobility, COPD, HIV.

C. Pathology / Pathogenesis

8. Bone mass peaks in the third decade, then declines; ageing causes **uncoupling of bone remodelling** — osteoclastic resorption exceeds osteoblastic formation, so each remodelling cycle produces net bone loss.
9. **Oestrogen deficiency** after menopause increases **RANKL** and reduces osteoprotegerin, raising osteoclast number, activity and lifespan → accelerated trabecular loss (5–10 years of rapid loss).
10. **Age-related (type II) loss** is driven by secondary hyperparathyroidism from vitamin D deficiency and reduced calcium absorption, declining IGF-1/GH, sarcopenia and reduced mechanical loading; affects cortical and trabecular bone.
11. Structural consequence: **trabecular thinning and perforation, loss of connectivity, cortical thinning and increased cortical porosity** → disproportionate loss of bone strength relative to loss of bone density.

D. Clinical Features

12. Osteoporosis itself is **asymptomatic ("silent thief") until fracture occurs** — the first clinical presentation is usually a low-trauma fracture after a fall from standing height or less.
13. **Vertebral fracture:** acute back pain, or painless — presenting as **progressive height loss, dorsal kyphosis**, protuberant abdomen, reduced rib–pelvis distance, restrictive lung defect and early satiety.
14. **Hip fracture:** shortened externally rotated limb, inability to weight-bear; **Colles' fracture** after fall on outstretched hand; also proximal humerus and pelvic rami fractures.

E. Investigations

15. **DXA of spine and hip** is the reference standard for diagnosis and for risk stratification (see Q3).
16. **Vertebral fracture assessment (VFA) or lateral spine X-ray** if there is height loss or hyperkyphosis — morphometric fractures are frequently silent but strongly predict further fracture.
17. **Baseline biochemistry to exclude secondary causes:** serum **calcium (8.5–10.5 mg/dL)**, phosphate, alkaline phosphatase, **creatinine and eGFR**, full blood count, ESR, **25-OH vitamin D**, TSH, and where indicated PTH, testosterone (men), serum/urine electrophoresis, coeliac serology, 24-hour urinary calcium.
18. **Bone turnover markers** (serum CTX for resorption, P1NP for formation) — used chiefly to **verify compliance with antiresorptive therapy at 3–6 months**, not for diagnosis.
19. **Trabecular bone score (TBS)** from the DXA image and **hip axis length** refine FRAX-based risk beyond BMD alone.

F. Management (Pathy's Algorithm — Kanis/ESCEO Pathway)

(i) Non-pharmacological — for all patients

20. Education on osteoporosis, medication, diet and exercise; **daily weight-bearing physical activity**.
21. Nutrition: **calcium 800–1000 mg/day**, **protein ≥1 g/kg body weight/day**, **vitamin D 800–1000 IU/day**.
22. **Fall-prevention** with supervised progressive strength and balance training — the programme should run for at least **50 hours** to reduce falls (effect on fracture risk remains uncertain).

(ii) Pharmacological — stratified by fracture risk

Risk category	Pathy's / ESCEO recommended treatment
Low risk	Reassurance and lifestyle advice; consider **menopausal hormone therapy (MHT)** or **SERMs** (raloxifene, bazedoxifene)
High risk	Start an **inhibitor of bone resorption** — oral bisphosphonate (gastro-resistant risedronate improves adherence), IV bisphosphonate, or **denosumab**
Very high risk	**Anabolic therapy first** — teriparatide/biosimilars, abaloparatide or romosozumab — **followed by an antiresorptive**
Very high hip fracture risk (esp. after first hip fracture)	Consider **local osteo-enhancement procedure (LOEP)**

23. **Key efficacy figures (Pathy's):** yearly IV zoledronic acid reduces clinical fracture by **33%**, vertebral fracture by **77%** and peripheral fracture by **25%**; give vitamin D beforehand. Adverse effects: acute-phase influenza-like reaction in one in three at first infusion, and a higher incidence of atrial fibrillation than placebo.
24. **Denosumab** (anti-RANKL monoclonal antibody, subcutaneous twice yearly) blocks RANKL–RANK binding, inhibits osteoclast development, and reduces vertebral, non-vertebral and hip fractures. **Discontinuation causes rebound multiple vertebral fractures** — always follow with a bisphosphonate for 1–2 years.
25. **Teriparatide** reduces new vertebral and non-vertebral fracture independent of baseline T-score, age and number of fractures; BMD gain is dose-dependent. **Romosozumab** (anti-sclerostin) for 12 months followed by alendronate produced significantly fewer fractures than alendronate alone (Saag et al.); FDA-approved since April 2019 for postmenopausal women at high fracture risk.
26. **Limitations in the elderly:** raloxifene does not reduce femoral neck fracture; MHT is effective but carries breast cancer, thromboembolic, stroke and coronary risk; strontium ranelate raised BMD but caused DRESS and thromboembolism.
27. Oral bisphosphonates are usually first-line and most cost-effective — **NNT to prevent one hip fracture is about 200**. In the oldest and frailest, weigh pill burden and comorbidity. Benefits of oral drugs appear at **6–12 months**, whereas fall-prevention benefit may be immediate.
28. **Age is not a reason to withhold treatment** — unlike cancer screening, the effectiveness of fracture prevention **increases with advancing age**; economic models suggest treatment is cost-effective with a life expectancy as short as two years.

(iii) Follow-up

29. Assess compliance and side effects; check bone turnover markers at 3–6 months. Review after **3 years of IV** or **5 years of oral** bisphosphonate, or after any incident fracture: low-risk patients may take a **drug holiday for 2 years** (reconsider annually); high-risk patients continue.
30. **Fracture Liaison Service (FLS)** — a coordinator-based system for the routine assessment and treatment of all postmenopausal women and men after a fragility fracture; benefits are well established and it is the key strategy to close the "treatment gap".

G. Complications

31. **Fracture-related:** hip fracture → excess mortality, loss of independence, institutionalisation; vertebral fractures → chronic back pain, kyphosis, restrictive lung disease, GORD and anorexia; distal radius → complex regional pain syndrome.
32. **Treatment-related:** bisphosphonates — oesophagitis, hypocalcaemia, acute phase reaction, atrial fibrillation, rare **osteonecrosis of the jaw** and **atypical femoral fracture**; denosumab — hypocalcaemia, ONJ, rebound fractures on stopping.

Latest Updates (2023 – Present)

- **ASBMR / Bone Health & Osteoporosis Foundation Task Force position statement, 2024 (JBMR)** formalised **goal-directed treatment**: choose the initial agent by the T-score target it can realistically achieve. Modelling showed that from a lumbar-spine T-score of −3.0, the probability of reaching a T-score above −2.5 was 55% with alendronate alone, 81% with romosozumab→alendronate and 93% with romosozumab→denosumab.
- The same statement, together with AACE, Endocrine Society, The Menopause Society, BHOF, ESCEO/IOF and NOGG guidance, has embedded a formal **"very high fracture risk" category**, with a subset at **imminent risk for 2 years after an incident fracture**.
- **Sequential therapy is now the standard of care in very-high-risk patients:** anabolic (teriparatide / abaloparatide up to 2 years, or romosozumab for 1 year) **followed immediately by an antiresorptive**. Anabolic-first gives substantially greater BMD gain than antiresorptive-first (2025 clinical reviews of novel osteoporosis therapies).
- **Romosozumab safety caveat retained:** avoid in those with prior myocardial infarction or stroke; assess cardiovascular risk before starting.
- **NOGG 2024 and ISCD 2023** reaffirm FRAX-based intervention thresholds with TBS adjustment; **USPSTF 2025** continues to recommend BMD screening in women ≥65 years and in younger postmenopausal women at increased risk.

- **Indian practice:** ICMR-NIN 2024 RDA raised recommended calcium intake for older Indian adults to **1000 mg/day**; ISBMR advises Indian-specific reference BMD databases and treatment of vitamin D deficiency before starting antiresorptives. Osteoporosis care is delivered under **NPHCE**, with treatment cost cover through **AB-PMJAY** and the **Ayushman Vay Vandana Card** for those ≥70 years.

Current Practice Update

Issue	Pathy's 6th edition position	Current position (2023–present)
Choice of first drug	Antiresorptive first for high risk; anabolic reserved for very high risk	**Goal-directed:** agent chosen by the T-score goal achievable; anabolic-first far more likely to reach target
Risk categories	Low / high / very high (ESCEO)	Same, plus explicit **imminent risk** window of 2 years after a fracture
Vitamin D dose	Around 800–1000 IU/day	Unchanged; but routine high-dose intermittent bolus discouraged (may increase falls)
Calcium target	800–1000 mg/day	ICMR-NIN 2024: **1000 mg/day** for older Indian adults, preferably dietary
Denosumab stopping	Follow with bisphosphonate 1–2 years	Reinforced — denosumab now regarded as an **indefinite-commitment drug**; never allow a lapsed dose

Extra Recall Scaffold — Recurring Topic

Mnemonic for secondary osteoporosis — "STEROIDS": **S**teroids · **T**hyrotoxicosis / hyperparathyroidism · **E**thanol · **R**heumatoid arthritis · **O**steomalacia / vitamin D deficiency · **I**mmobility · **D**iabetes and hypogonaDism · **S**malabsorption (coeliac, IBD).

Numerical anchors: T-score −2.5 · osteopenia −1 to −2.5 · height loss >4 cm · calcium 800–1000 mg · protein ≥1 g/kg · vitamin D 800 IU · exercise ≥50 hours · zoledronate: vertebral ↓77%, clinical ↓33%, peripheral ↓25% · NNT for hip fracture ≈200 · review at 3 years IV / 5 years oral · drug holiday 2 years.

Examiner traps: (1) Saying osteoporosis is diagnosed by X-ray — plain films only show osteopenia after 30–40% bone loss. (2) Forgetting that **a fragility fracture diagnoses osteoporosis clinically regardless of T-score**. (3) Stopping denosumab without bisphosphonate cover. (4) Withholding treatment because "the patient is too old" — benefit increases with age. (5) Starting a bisphosphonate without first correcting vitamin D deficiency and hypocalcaemia.

15-second answer skeleton: Define (WHO T-score −2.5) → burden and Indian data → risk factors (non-modifiable / modifiable / drugs / secondary) → pathogenesis (uncoupled remodelling, RANKL, secondary hyperparathyroidism) → silent until fracture → DXA + VFA + secondary-cause screen → lifestyle for all → risk-stratified drug ladder → follow-up, drug holiday, Fracture Liaison Service → complications of fracture and of therapy.

Exam One-Liners

- WHO diagnostic threshold: T-score ≤ −2.5; osteopenia −1.0 to −2.5.
- Commonest osteoporotic fractures: vertebral > hip > distal radius.
- Height loss >4 cm is an indication for vertebral fracture assessment.
- Zoledronic acid 5 mg IV once yearly reduces vertebral fracture by 77%.
- Denosumab must never be stopped without antiresorptive cover.
- Fracture Liaison Service is the single most effective system-level intervention for secondary prevention.
- Bone turnover markers are for compliance monitoring, not diagnosis.

Q2. A 68-Year-Old with Fracture Neck of Femur — Risk of Further Fragility Fracture, Further Tests and Treatment Options

15 MARKS

A. Framing the Problem

1. A hip fracture after low-energy trauma in a 68-year-old is by definition a **fragility fracture** and establishes a clinical diagnosis of osteoporosis **irrespective of the DXA T-score**.
2. Hip fracture carries substantial excess mortality, loss of independence and institutionalisation; non-hip, non-spine fractures also drive large healthcare utilisation.

B. Risk of a Further Fragility Fracture

3. A prior fragility fracture is one of the **major independent risk factors** in Pathy's list — it roughly **doubles** the risk of any subsequent fracture.
4. The risk is **front-loaded**: it is highest in the first 1–2 years after the index fracture — the **"imminent fracture risk"** window.
5. Around one in five patients sustains a second fracture, and a substantial proportion of these are contralateral hip fractures.
6. Yet the **treatment gap is wide** — most patients leave hospital after a hip fracture without any anti-osteoporosis medication; this is precisely the problem the **Fracture Liaison Service** was created to solve.
7. Additional determinants of further fracture in this patient: **continuing falls risk**, sarcopenia, postural hypotension, visual impairment, polypharmacy (Beers criteria), vitamin D deficiency and post-fall syndrome with fear of falling.

C. Further Tests

8. **DXA of the contralateral hip and lumbar spine** — not to decide whether to treat (treatment is already indicated), but to provide a baseline for monitoring and to grade severity.
9. **Lateral thoracolumbar X-ray or VFA** to detect silent vertebral fractures, which change risk category and drug choice.
10. **Biochemistry:** serum calcium, phosphate, alkaline phosphatase, creatinine/eGFR, full blood count, ESR, **25-OH vitamin D**, TSH, liver function.
11. **Targeted secondary-cause screen:** PTH, serum and urine protein electrophoresis with free light chains (myeloma), coeliac serology, 24-hour urinary calcium, and in a man **morning total testosterone (ng/dL)** — late-onset hypogonadism is an under-recognised cause.
12. **Renal function is decisive** for drug choice — bisphosphonates are avoided if eGFR is below about 30–35 mL/min.
13. **FRAX** with BMD, adjusted for the recent fracture, plus **TBS** if available, to document the risk category.
14. **Comprehensive geriatric assessment:** multifactorial falls assessment, gait and balance testing (Timed Up and Go), cognition, nutrition (MNA), continence, medication review and social circumstances.

D. Treatment Options

(i) Acute and orthogeriatric care

15. **Early surgery (ideally within 48 hours)** under a shared orthogeriatric model, multimodal opioid-sparing analgesia including fascia iliaca block, delirium prevention (HELP-type bundle), pressure-ulcer prevention, VTE prophylaxis and early mobilisation with rehabilitation.

(ii) Bone-directed pharmacotherapy

16. **Correct vitamin D and calcium first:** Pathy's states that all people with a hip fracture should receive calcium, vitamin D and a bisphosphonate unless contraindicated (e.g. renal failure); 25-OH vitamin D should be above 30 ng/mL.
17. **Zoledronic acid 5 mg IV once yearly** is the preferred agent after hip fracture — it overcomes adherence problems, reduces clinical fracture by 33%, vertebral by 77% and peripheral by 25%; give at least 2 weeks after vitamin D repletion, ideally ≥2 weeks post-operatively.
18. **Denosumab 60 mg subcutaneously 6-monthly** is an alternative, particularly when eGFR is low; monitor calcium and **never stop it without bisphosphonate cover**.
19. **Anabolic first-line** (teriparatide, abaloparatide or romosozumab) if the patient is in the very-high-risk category — hip fracture plus multiple vertebral fractures or very low T-score — followed by an antiresorptive.
20. **Local osteo-enhancement procedure (LOEP)** may be considered where hip fracture risk is very high, particularly following a first hip fracture.
21. **Avoid raloxifene and other SERMs after femoral neck fracture** — Pathy's is explicit that they do not reduce femoral neck fracture and carry an increased thromboembolic risk in this setting.

(iii) Non-pharmacological and systems measures

22. **Falls prevention:** progressive strength and balance programme of at least 50 hours, home hazard assessment, footwear, vision correction, withdrawal of culprit drugs (sedatives, antihypertensives, anticholinergics), management of postural hypotension.
23. **Nutrition:** protein ≥1 g/kg/day, calcium 1000 mg/day, treatment of sarcopenia by resistance exercise plus protein

supplementation.

24. **Hip protectors** may be considered in the institutionalised, though adherence is poor, particularly with cognitive impairment.
25. **Enrol in a Fracture Liaison Service** with structured follow-up at 3–6 months for adherence, and repeat DXA in 1–3 years.

Latest Updates (2023 – Present)

- **Imminent fracture risk** is now formally recognised (ASBMR/BHOF 2024): the 2 years after a hip fracture constitute the highest-risk period, justifying immediate initiation of potent therapy rather than a "wait for DXA" approach.
- **Goal-directed therapy** supports anabolic-first sequencing after hip fracture in those with very low T-scores, since romosozumab→denosumab achieves a T-score above −2.5 far more often than alendronate alone.
- Registry and meta-analytic data continue to show that **early zoledronic acid after hip fracture reduces both re-fracture and all-cause mortality**, and that in-hospital initiation markedly improves uptake compared with deferring to primary care.
- **Fragility Fracture Network / FLS models** are now endorsed in Indian practice; NPHCE district-level geriatric units and AB-PMJAY/Ayushman Vay Vandana Card cover hip fracture surgery and follow-up in those ≥70 years.

Current Practice Update

Issue	Pathy's position	Current position
When to start drug therapy	After a fragility fracture, with calcium, vitamin D and a bisphosphonate	Same, but **started before discharge**; deferral to outpatient review is a recognised failure point
Drug choice after hip fracture	Bisphosphonate unless contraindicated	**IV zoledronate preferred**; anabolic-first if very high risk
SERMs	Avoid after femoral neck fracture	Unchanged — contraindicated in this scenario

Exam One-Liners

- A fragility fracture diagnoses osteoporosis clinically — do not wait for DXA to treat.
- Highest re-fracture risk is in the first 2 years (imminent risk).
- IV zoledronate after hip fracture reduces re-fracture and mortality.
- Never give a SERM after a femoral neck fracture.
- The Fracture Liaison Service closes the treatment gap.

Q3. DEXA Scan (Dual-Energy X-Ray Absorptiometry) and its Clinical Use — 7 MARKS

1. **Principle:** two X-ray beams of different energies are passed through the body; differential attenuation by bone and soft tissue allows calculation of **bone mineral content and areal bone mineral density (g/cm²)**. It is the **reference standard** for measuring BMD.
2. **Standard sites: lumbar spine (L1–L4)** and **hip (femoral neck and total hip)**; the **distal one-third radius** is used when spine or hip cannot be measured, or in hyperparathyroidism and very obese patients.
3. **T-score** compares BMD with the young adult mean: **≥−1.0 normal**, **−1.0 to −2.5 osteopenia**, **≤−2.5 osteoporosis**, and **≤−2.5 with a fragility fracture = severe (established) osteoporosis**. **Z-score** compares with age-matched peers and is used in men under 50 and premenopausal women; **Z ≤ −2.0** means "below expected for age" and mandates a secondary-cause work-up.
4. **Radiation dose is very low** (a few microsieverts, less than a chest radiograph), the scan takes minutes, and it is precise enough for serial monitoring.
5. **Clinical uses:** (a) diagnosis of osteoporosis; (b) fracture-risk prediction and input into **FRAX**; (c) deciding intervention thresholds; (d) monitoring response to therapy, repeated every **1–3 years**; (e) **Vertebral Fracture Assessment (VFA)** from the same machine; (f) **Trabecular Bone Score (TBS)** as a texture measure of bone quality; (g) body-composition and appendicular lean mass measurement for the diagnosis of **sarcopenia**.
6. **Who to scan:** Pathy's advises BMD in **all women from 50 years and all men by 65 years**, repeating in two years (in the same season) if results are borderline; also anyone with a fragility fracture, height loss, glucocorticoid therapy, or a secondary cause.
7. **Pitfalls that falsely raise spine BMD in older people: degenerative osteophytes and facet arthrosis, vertebral fracture, aortic calcification, scoliosis, previous instrumentation, and residual contrast** — hence in the elderly the **hip is the more reliable site**. Exclude any vertebra whose T-score differs from adjacent ones by more than 1.0; at least two vertebrae must be evaluable. Diagnosis uses the **lowest** of the valid sites. Repeat scans should be on the same machine, since inter-machine comparison is invalid without cross-calibration, and change is meaningful only if it exceeds the **least significant change**.

Latest Updates (2023 – Present) — 3 Points

- **ISCD 2023 Official Positions** endorse routine reporting of **TBS alongside BMD** and permit **opportunistic CT-based BMD screening** when a CT has been done for another reason.
- **ASBMR/BHOF 2024 goal-directed treatment** converts DXA from a diagnostic test into a **treatment target**: the aim is a T-score above −2.5, and the drug is chosen by the likelihood of reaching that goal.
- **Indian reference data:** ISBMR advises Indian young-adult reference databases and cautions that Caucasian NHANES norms over-diagnose osteoporosis in Indian patients; DXA-based appendicular lean mass indices are also used with **AWGS 2019** Asian cut-offs for sarcopenia.

Extra Recall Scaffold — Recurring Topic

Mnemonic for falsely high spine BMD — "SCAFOLD": **S**coliosis · **C**ompression fracture · **A**ortic calcification · **F**acet joint / osteophyte degeneration · **O**steomalacia treatment effect · **L**aminectomy hardware / instrumentation · **D**ye (residual contrast).

Numerical anchors: normal ≥−1.0 · osteopenia −1 to −2.5 · osteoporosis ≤−2.5 · Z-score ≤−2.0 abnormal · monitor every 1–3 years · adjacent-vertebra T-score difference >1.0 = exclude · women from 50, men by 65.

Examiner traps: (1) Using T-score in a premenopausal woman or young man — use Z-score. (2) Reporting the **average** of sites rather than the **lowest**. (3) Forgetting that severe spinal degeneration in the elderly makes spine BMD unreliable. (4) Comparing scans from two different machines. (5) Saying DXA measures volumetric density — it measures **areal** density, which underestimates BMD in small-framed people (relevant to Indian patients).

15-second answer skeleton: Principle → sites → T-score and Z-score with cut-offs → safety and precision → seven clinical uses → who to scan → pitfalls in the elderly and monitoring rules.

Q4. FRAX Score — 7 MARKS

1. **FRAX is an algorithm that integrates the weight of clinical risk factors for fracture, with or without BMD, and computes the 10-year probability of hip fracture and of a major osteoporotic fracture** (hip, clinical spine, proximal humerus, distal forearm). It was developed by Kanis and the WHO Collaborating Centre, Sheffield.
2. **The eleven inputs:** age (40–90), sex, weight, height, previous fragility fracture, parental hip fracture, current smoking, glucocorticoid use, rheumatoid arthritis, secondary osteoporosis, alcohol ≥3 units/day, and (optionally) **femoral neck BMD**.
3. **Country-specific calibration:** the tool is region-specific because fracture and death rates differ widely between populations — an **Indian FRAX model** exists and must be used for Indian patients.
4. **Clinical use:** FRAX output is compared with a country-specific **intervention threshold**. In Pathy's algorithm, treatment is offered when the FRAX probability for major osteoporotic (or hip) fracture reaches a **risk equivalent to that associated with a prevalent fragility fracture** — an age-dependent threshold. FRAX also identifies who needs a DXA when access is limited.
5. **Modifiers that refine FRAX:** BMD, **trabecular bone score (TBS)**, glucocorticoid dose, fall history, type 2 diabetes and hip axis length.
6. **Advantages:** free, quick, usable without DXA, validated in large international cohorts, and it shifts practice from a pure BMD threshold to absolute risk — important because most fractures occur in people who are only osteopenic.
7. **Limitations:** does not capture **falls**, **number or recency of fractures** (it treats one fracture the same as five), dose of glucocorticoid, lumbar spine BMD, vitamin D status, or frailty; it is **not validated on treatment** and is capped at 90 years; it therefore **underestimates imminent risk** immediately after a fracture.

Latest Updates (2023 – Present) — 3 Points

- **FRAXplus** (released from 2023) is an extension allowing adjustment for **recency of fracture, glucocorticoid dose, number of falls, TBS, type 2 diabetes, hip axis length and lumbar spine BMD** — directly addressing the classical limitations.
- **NOGG 2024** and ESCEO/IOF guidance retain FRAX-based age-dependent intervention thresholds and add a **very-high-risk threshold** that triggers anabolic-first therapy.
- Guidelines increasingly note that FRAX should be **overridden** in the presence of a recent (<2 year) major fracture or multiple vertebral fractures, where imminent risk exceeds the calculated figure.

Exam One-Liners

- FRAX gives the 10-year probability of major osteoporotic and hip fracture.
- Eleven clinical inputs; femoral neck BMD optional.
- Must use the country-specific (Indian) model.
- Blind spots: falls, fracture number and recency, steroid dose, spine BMD.
- FRAXplus corrects most of these.

Q5. Selective Estrogen Receptor Modulators (SERMs) — 7 MARKS

1. **Definition:** SERMs are non-steroidal agents that bind the oestrogen receptor and act as **tissue-selective agonists or antagonists** — agonist on bone and lipids, antagonist on breast, with variable effect on the endometrium. Selectivity arises from differential recruitment of co-activators and co-repressors to ER-α and ER-β.
2. **Agents: raloxifene** (60 mg/day, second generation, the main bone agent), **bazedoxifene** (third generation, also used as a tissue-selective oestrogen complex with conjugated oestrogens), **tamoxifen** and **toremifene** (breast cancer), **ospemifene** (dyspareunia), **lasofoxifene**.
3. **Mechanism on bone:** agonist action suppresses osteoclastic resorption and reduces bone turnover, preserving BMD — a mild antiresorptive effect, weaker than bisphosphonates.
4. **Indication in Pathy's algorithm:** in postmenopausal women categorised as **low fracture risk**, menopausal hormone therapy and SERMs may be considered. Raloxifene raises lumbar spine BMD by about **3%** and femoral neck by **2%** at 12–18 months and reduces **vertebral fracture only**.
5. **Key limitation (frequently examined): raloxifene has not been found to decrease the incidence of femoral neck fractures**, and **SERMs must be avoided after femoral neck fracture because of the increased risk of thromboembolic complications**.
6. **Additional benefit:** raloxifene reduces the incidence of **ER-positive invasive breast cancer**, making it attractive in a woman with vertebral osteoporosis and elevated breast cancer risk.
7. **Adverse effects and cautions: venous thromboembolism** (2–3 fold increase — the main hazard in the elderly and in the immobile), **hot flushes**, leg cramps, peripheral oedema; an excess of fatal stroke was seen in women with cardiovascular risk factors. Contraindicated in prior VTE, prolonged immobility, and active hepatic disease. Unlike oestrogen, raloxifene **does not stimulate the endometrium** (no increase in endometrial cancer) and does not relieve vasomotor symptoms.

Latest Updates (2023 – Present) — 3 Points

- Current guidance (Endocrine Society, NOGG 2024, ESCEO) positions SERMs as **second- or third-line**, reserved for younger postmenopausal women with predominantly vertebral disease, low hip fracture risk and elevated breast cancer risk — not for the old-old.
- **Bazedoxifene / conjugated oestrogen (TSEC)** remains an option for women needing both vasomotor symptom relief and bone protection without endometrial stimulation.
- Sequence matters: when a SERM is stopped, BMD is lost, so **follow-on antiresorptive therapy** is advised in those who remain at risk; the ASBMR/BHOF 2024 goal-directed framework notes that SERMs rarely achieve the target T-score in high-risk patients.

Exam One-Liners

- Agonist on bone, antagonist on breast — that one line defines a SERM.
- Raloxifene reduces vertebral fracture only, not hip fracture.
- Contraindicated after femoral neck fracture and in any VTE risk.
- Reduces ER-positive breast cancer; does not cause endometrial hyperplasia.
- Worsens, not relieves, hot flushes.

Q6. Atypical Femur Fractures Due to Drugs — 7 MARKS

1. **Definition:** an atypical femoral fracture (AFF) is a **stress or insufficiency fracture of the subtrochanteric region or femoral shaft** occurring with minimal or no trauma, associated with prolonged suppression of bone turnover — classically by **long-term bisphosphonates**, and also reported with **denosumab**, glucocorticoids and proton-pump inhibitors.
2. **ASBMR major features (all five required):** (i) minimal or no trauma; (ii) fracture line originates at the **lateral cortex and is substantially transverse**; (iii) **non-comminuted** or minimally comminuted; (iv) complete fractures extend through both cortices with a **medial spike**, incomplete fractures involve only the lateral cortex; (v) **localised periosteal or endosteal thickening ("beaking" or "flaring") of the lateral cortex**. Fractures of the femoral neck, intertrochanteric region with spiral extension, and pathological or periprosthetic fractures are excluded.
3. **Minor features:** generalised increase in cortical thickness of the femoral diaphysis, prodromal dull thigh or groin pain, **bilaterality**, delayed union, and comorbid conditions or drug exposure (bisphosphonates, denosumab, glucocorticoids, PPIs).
4. **Pathogenesis:** severe suppression of bone turnover impairs repair of micro-damage; accumulated micro-cracks, hypermineralised brittle bone and reduced collagen heterogeneity produce a fatigue fracture at the site of maximal tensile stress on the lateral femoral cortex. Bowed femora, Asian ethnicity, short stature and glucocorticoids increase risk.
5. **Clinical presentation and diagnosis:** **prodromal thigh or groin pain** in about two-thirds, often for weeks to months, then a fracture on trivial trauma. Investigate any patient on long-term antiresorptive therapy with new thigh pain by **full-length femoral radiographs of both femora**; if negative but suspicion persists, **MRI or bone scintigraphy** detects early stress reaction. Always image the **contralateral** femur, as bilateral involvement is common.
6. **Risk in context — the crucial balance:** AFF is **rare** (of the order of a few per 10,000 patient-years, rising with duration beyond 5 years and falling rapidly after stopping), whereas bisphosphonates prevent many times that number of typical osteoporotic fractures. **The benefit clearly outweighs the risk in patients at high fracture risk.** Osteonecrosis of the jaw is the other rare class effect.
7. **Management:** (a) **stop the bisphosphonate or denosumab** (with antiresorptive cover if denosumab is discontinued); (b) ensure calcium and vitamin D repletion; (c) **prophylactic intramedullary nailing** for a complete fracture, and for an incomplete fracture with a radiolucent line or persistent pain — conservative treatment with protected weight-bearing is reserved for incomplete fractures without a dreaded black line; (d) **teriparatide** may be used to promote healing and to continue osteoporosis treatment; (e) periodic **drug holiday review at 3 years for IV and 5 years for oral bisphosphonates** is the principal preventive strategy.

Latest Updates (2023 – Present) — 3 Points

- 2025 reviews of novel osteoporosis therapies reinforce structured **drug-specific safety monitoring**: assess cardiovascular history before romosozumab, monitor calcium with teriparatide, abaloparatide and denosumab (especially in renal impairment), and watch for **MRONJ around dental procedures and AFF with longer-term bisphosphonate use**.
- **Drug holidays** remain the mainstay of AFF prevention for bisphosphonates — but the concept explicitly **does not apply to denosumab**, where interruption causes rebound vertebral fractures.
- Asian ancestry, femoral bowing and glucocorticoid use are now recognised as amplifying risk, which is directly relevant to Indian patients on long-term therapy; contralateral femoral imaging is standard once an AFF is diagnosed.

Current Practice Update

Issue	Textbook position	Current position
Attribution	Rare complication of bisphosphonates	Also with **denosumab**; risk falls rapidly after drug cessation
Prevention	Limit duration of therapy	Formalised as **drug holiday at 3 years IV / 5 years oral**, with annual reassessment — not applicable to denosumab
Ongoing bone therapy after AFF	Not specified	**Teriparatide** is the preferred continuation agent

Exam One-Liners

- Site: subtrochanteric or diaphyseal — never the femoral neck.
- Transverse, non-comminuted, lateral cortical beaking, medial spike.
- Prodromal thigh pain in two-thirds; frequently bilateral — always image both femora.
- Stop the antiresorptive; nail complete and painful incomplete fractures; consider teriparatide.
- Absolute risk is tiny compared with the fractures prevented.

Q7. Renal Osteodystrophy — Laboratory and Radiological Abnormalities and Treatment

15 MARKS

A. Definition and Classification

1. **Renal osteodystrophy** is the spectrum of **bone histological abnormalities** occurring in chronic kidney disease. It is the skeletal component of the broader syndrome **CKD–Mineral and Bone Disorder (CKD–MBD)**, which also includes biochemical abnormalities and vascular/soft-tissue calcification.
2. Definitive classification is by bone biopsy using the **TMV system — Turnover, Mineralisation, Volume**.
3. **High-turnover disease: osteitis fibrosa cystica** from secondary hyperparathyroidism. **Low-turnover disease: adynamic bone disease** (low turnover, normal mineralisation) and **osteomalacia** (low turnover, defective mineralisation). **Mixed uraemic osteodystrophy** combines both. Dialysis patients may also develop **β2-microglobulin amyloid** bone disease.

B. Pathophysiology (the four-step cascade)

4. Falling GFR → **phosphate retention** → rise in **FGF-23** (the earliest abnormality) with **Klotho deficiency**.
5. FGF-23 and reduced renal mass suppress **1-α-hydroxylase** → low **calcitriol** → reduced intestinal calcium absorption → **hypocalcaemia**.
6. Hypocalcaemia, hyperphosphataemia and calcitriol deficiency all stimulate PTH → **secondary hyperparathyroidism**, with parathyroid hyperplasia and down-regulation of calcium-sensing and vitamin D receptors; autonomous nodular hyperplasia produces **tertiary hyperparathyroidism** with hypercalcaemia.
7. **Adynamic bone disease** results from over-suppression of PTH — by aggressive calcium and vitamin D loading, high-calcium dialysate, calcimimetics, diabetes, ageing and peritoneal dialysis. It is now the commonest lesion, and is important in the elderly because it causes fractures and drives vascular calcification.
8. **Aluminium** (from phosphate binders or dialysate) causes osteomalacia and microcytic anaemia — historical but still examinable.

C. Laboratory Abnormalities

Parameter	Osteitis fibrosa (high turnover)	Adynamic bone disease	Osteomalacia
Serum calcium (mg/dL)	Low or normal; high in tertiary	Normal or **high**	Low
Serum phosphate (mg/dL)	**High**	High or normal	Low or normal
PTH	**Markedly raised** (>9× upper limit)	**Low / inappropriately normal** (<2×)	Variable
Alkaline phosphatase / bone ALP	**High**	Low or normal	High
25-OH vitamin D	Often low	Variable	**Low**
Calcitriol	Low	Low	Low
FGF-23	**Very high**	High	High

9. Supporting tests: serum aluminium (with desferrioxamine test if indicated), β2-microglobulin in long-term dialysis, and **bone-specific alkaline phosphatase plus intact PTH** as the practical non-invasive discriminators. **Bone biopsy with tetracycline double labelling** remains the gold standard, reserved for unexplained fractures, hypercalcaemia, suspected aluminium disease, or before parathyroidectomy.

D. Radiological Abnormalities

10. **Subperiosteal resorption** at the **radial border of the middle phalanges of the index and middle fingers** — the earliest and most specific sign; also at the distal clavicle, symphysis pubis, sacroiliac joints and medial proximal tibia. Terminal phalangeal **acro-osteolysis** with tuft resorption.
11. **"Rugger-jersey spine"** — alternating sclerotic bands at the vertebral end-plates with a lucent centre; and a **"salt-and-pepper" skull** with a granular mottled vault.
12. **Brown tumours** — expansile lytic lesions (osteoclastomas) in mandible, ribs, pelvis or long bones, which may fracture and can be mistaken for metastases.
13. **Osteomalacic changes: Looser's zones (pseudofractures)** at the medial femoral neck, pubic rami, scapula and ribs; coarse trabeculae, biconcave vertebrae.
14. **Extraskeletal calcification:** vascular (Monckeberg medial), periarticular tumoral calcinosis, cardiac valve and corneal calcification; **calciphylaxis (calcific uraemic arteriolopathy)** presents as painful violaceous livedoid skin necrosis with high mortality.
15. Modern assessment: **lateral abdominal radiograph and echocardiography** for vascular and valvular calcification (KDIGO-endorsed); **DXA is now recommended** in CKD G3a–G5D to predict fracture, though it cannot distinguish the type of renal osteodystrophy.

E. Treatment

16. **Control phosphate first** — dietary phosphate restriction (avoid processed food with inorganic phosphate additives, which is highly absorbable), adequate dialysis, and phosphate binders: **calcium carbonate/acetate** (limit total elemental calcium, avoid if adynamic bone or vascular calcification), **sevelamer**, **lanthanum carbonate**, and iron-based binders (**sucroferric oxyhydroxide, ferric citrate**). Avoid aluminium binders except short-term.
17. **Correct native vitamin D deficiency** with cholecalciferol; then, for persistently high and rising PTH, use **active vitamin D analogues** (calcitriol, alfacalcidol) or the less calcaemic **paricalcitol / doxercalciferol**.
18. **Calcimimetics** — oral **cinacalcet** or IV **etelcalcetide** (dialysis) — lower PTH, calcium and phosphate simultaneously by sensitising the calcium-sensing receptor; the drug of choice when PTH is high with hypercalcaemia.
19. **Target PTH** in dialysis patients at approximately **2–9 times the upper limit of normal** for the assay — deliberately not normal, because normal PTH in CKD predicts adynamic bone.
20. **Parathyroidectomy** for severe refractory secondary or tertiary hyperparathyroidism with hypercalcaemia, intractable pruritus, calciphylaxis, or progressive bone disease. Anticipate **hungry bone syndrome** with profound post-operative hypocalcaemia.
21. **Adynamic bone disease** is managed by doing **less**: reduce calcium load, lower dialysate calcium, withdraw or reduce active vitamin D and calcimimetics, and allow PTH to rise into the target range.
22. **Osteoporosis therapy in CKD:** bisphosphonates are generally avoided below eGFR 30–35 mL/min and can worsen adynamic bone; **denosumab** is not renally cleared but causes severe hypocalcaemia in advanced CKD and needs close monitoring with calcium and vitamin D loading; **teriparatide** may help low-turnover disease but is contraindicated where PTH is already high.
23. **General measures:** correct metabolic acidosis with bicarbonate, treat hypogonadism, encourage weight-bearing exercise and resistance training, prevent falls, avoid aluminium-containing antacids, and manage calciphylaxis with sodium thiosulfate, wound care and withdrawal of calcium, vitamin D and warfarin.

Latest Updates (2023 – Present)

- **KDIGO CKD-MBD guidance** continues to advise treating **trends rather than single values** for calcium, phosphate and PTH, lowering elevated phosphate **towards** the normal range rather than to a fixed target, and restricting the dose of calcium-based binders in adults.
- **DXA is now firmly recommended** in CKD G3a–G5D where results would change management — a reversal of the older position that DXA is uninformative in CKD.
- **Upacicalcet** and other newer calcimimetics have been approved in some jurisdictions for secondary hyperparathyroidism in dialysis; iron-based binders (sucroferric oxyhydroxide, ferric citrate) are increasingly preferred for their low pill burden and correction of iron deficiency.
- Growing evidence links **high phosphate and FGF-23 to cardiovascular mortality**, shifting emphasis from bone endpoints to vascular protection; inorganic phosphate additives in processed foods are now a specific dietary target.
- **Indian context:** CKD-MBD care is delivered through the **Pradhan Mantri National Dialysis Programme**; nutritional vitamin D deficiency is near-universal in Indian CKD patients and should be corrected before interpreting PTH.

Current Practice Update

Issue	Older textbook position	Current position
Commonest lesion	Osteitis fibrosa cystica	**Adynamic bone disease**, from over-treatment and diabetes
Phosphate binders	Calcium-based, first-line	**Restrict calcium-based binders**; prefer non-calcium binders
PTH target	Normalise PTH	Keep at **2–9× upper limit**; normal PTH in dialysis is a warning sign
DXA in CKD	Unhelpful	**Recommended** in G3a–G5D to predict fracture

Exam One-Liners

- Renal osteodystrophy is the bone histology; CKD-MBD is the whole syndrome.
- Classified by the TMV system on bone biopsy.
- FGF-23 rise is the earliest biochemical abnormality.
- Subperiosteal resorption of the radial border of the middle phalanx is the classic radiological sign.
- Rugger-jersey spine, salt-and-pepper skull, brown tumours, Looser's zones.
- Adynamic bone disease means too much treatment, not too little.
- Calciphylaxis is a dermatological emergency with very high mortality.

Q8. Osteoarthritis — Management — 15 MARKS

A. Principles

1. **Osteoarthritis** is the commonest joint disease of older adults and a leading cause of pain, immobility and disability. Management is **symptom-directed and function-directed**, since no agent has been shown to reverse structural damage.
2. **Optimal management involves multiple treatment modalities, and the best combination varies from patient to patient** (Kane's) — a core, quotable line.
3. Treating arthritis optimally **requires a differential diagnosis**, because several arthritides coexist in older adults. Distinguish OA from inflammatory arthritis: stiffness lasting **minutes** not hours, pain **with activity** rather than at rest and at night, fatigue unusual, swelling present but with little synovial reaction, and erythema and warmth unusual.

B. Non-Pharmacological Management (the foundation)

4. **Education and self-management**, with realistic goal-setting and an explanation that OA is manageable rather than inevitable disability.
5. **Exercise is first-line**: quadriceps and hip abductor strengthening, range-of-movement work, and low-impact aerobic activity. Patients referred for joint replacement should be **encouraged to lose weight and strengthen the relevant muscles before surgery**.
6. **Weight reduction** — the single most effective disease-modifying intervention for knee and hip OA.
7. **Physiotherapy and occupational therapy:** joint protection, energy conservation, activity pacing, and assessment for **assistive devices** — walking stick held in the **contralateral** hand, walking frame, raised toilet seat, tap turners, jar openers.
8. **Orthoses and footwear:** shock-absorbing insoles, appropriate footwear, knee braces for varus/valgus instability, thumb-base splint for first carpometacarpal OA.
9. **Local physical measures:** heat, cold, and transcutaneous electrical nerve stimulation as adjuncts.

C. Pharmacological Management

10. **Paracetamol** is the traditional first-line oral analgesic in older people because of its favourable safety profile, though its efficacy in OA is modest; maximum 3 g/day in the frail elderly.
11. **Topical NSAIDs** are preferred over oral NSAIDs for knee and hand OA in older adults, giving useful analgesia with minimal systemic exposure. **Topical capsaicin** is an alternative.
12. **Oral NSAIDs** — lowest dose, shortest duration, always with a proton-pump inhibitor. Hazardous in the elderly: gastrointestinal bleeding, acute kidney injury, fluid retention and heart failure decompensation, hypertension, and interaction with ACE inhibitors and diuretics ("triple whammy"). They appear on the **AGS Beers Criteria** as potentially inappropriate for chronic use.
13. **Intra-articular corticosteroid injection** for a painful effusive flare gives short-term relief; limit to three or four injections per joint per year. Local steroid injection also gives dramatic relief in **bicipital tendinitis, olecranon and trochanteric bursitis**, which are common in geriatric patients and must be sought on examination.
14. **Nutraceuticals:** glucosamine and chondroitin — although there may be a placebo effect resulting in benefit, **there is no evidence of significant improvement in pain**; likewise **vitamin D does not decrease pain or facilitate repair of structural damage** in OA.
15. **Duloxetine** and, cautiously, low-dose weak opioids (tramadol) are reserved for refractory pain; opioids carry falls, delirium, constipation and dependence risk in older adults.

D. Surgical and Interventional Management

16. **Arthroscopic interventions** (debridement and lavage, osteotomy, cartilage transplant) have been recommended where inflammation is present and non-invasive measures have failed, but **there is little evidence to support their efficacy** (Moseley et al.).
17. **Joint replacement should be reserved for individuals with severe symptomatic disease who do not respond to more conservative interventions**; patients referred should have **stable medical conditions** and be prepared with weight loss and muscle strengthening pre-operatively.
18. Post-operative care needs structured **rehabilitation**, delirium prevention, VTE prophylaxis and falls prevention.

E. Geriatric-Specific Considerations

19. Address the **consequences** as much as the joint: OA causes immobility, deconditioning, sarcopenia, falls, depression, social isolation and caregiver burden — a comprehensive geriatric assessment is appropriate.
20. Look actively for treatable non-articular mimics — tendinitis, bursitis, and **carpal tunnel syndrome**, which is common in older adults and can be confused with gout, rheumatoid arthritis or pseudogout.

> **Latest Updates (2023 – Present)**

- **OARSI and ACR guidance** continue to rank **structured exercise and weight management as core**, with topical NSAIDs preferred over oral NSAIDs in older adults; **glucosamine, chondroitin and hyaluronic acid injections remain not recommended** for knee OA.
- **Intra-articular corticosteroids:** repeated injections have been associated with cartilage volume loss on imaging, so use is now restricted to intermittent flares rather than a routine schedule.
- Community-level programmes are gaining evidence — the **WE-CAN study** examined the impact of a community diet-and-exercise programme on knee pain, reflecting a shift towards scalable, non-drug delivery models.
- Emerging pharmacology: **nerve growth factor inhibitors** (tanezumab) showed efficacy but rapidly progressive OA limited approval; **lorecivivint** (Wnt inhibitor) and **low-dose colchicine** are under evaluation as potential structure-modifying agents; **GLP-1 receptor agonists** (semaglutide, STEP-9 trial) reduce knee OA pain substantially through weight loss in obesity.
- **Indian context:** knee OA prevalence is high, especially in women; management is supported by **ADIP/ALIMCO** for assistive devices and the **Rashtriya Vayoshri Yojana** for free aids in BPL elderly, with joint replacement covered under **AB-PMJAY**.

Current Practice Update

Issue	Textbook position	Current position
First-line analgesia	Paracetamol	Exercise and weight loss first; **topical NSAID** is the preferred first drug — paracetamol demoted to adjunct
Glucosamine / chondroitin / vitamin D	No proven benefit	Unchanged — explicitly **not recommended**
Arthroscopic lavage	Little supporting evidence	**Should not be offered** for degenerative knee disease
Intra-articular steroid	Useful for flares	Same, but limit frequency — cartilage volume loss with repeated injection
Weight loss	Advise before surgery	Now a therapeutic target in its own right, including **GLP-1 agonists** in obese patients

Exam One-Liners

- OA stiffness lasts minutes; inflammatory arthritis stiffness lasts hours.
- Exercise and weight loss are the only interventions that change the disease course.
- Walking stick goes in the hand opposite to the affected joint.
- Glucosamine, chondroitin and vitamin D do not work in OA.
- Arthroscopic lavage for degenerative knee disease is not supported by evidence.
- Always look for bursitis, tendinitis and carpal tunnel syndrome as treatable mimics.

Q9. Polymyalgia Rheumatica — 15 MARKS

A. Definition and Epidemiology

1. **Polymyalgia rheumatica (PMR)** is a chronic inflammatory rheumatic disease of older adults characterised by **pain and morning stiffness of the shoulder, neck and hip girdles** with a markedly raised acute-phase response, and a dramatic response to corticosteroids.
2. It is **a common musculoskeletal problem of older women** (Kane's) and occurs **almost exclusively in people over 50 years**, with peak incidence in the seventh and eighth decades; women are affected two to three times as often as men.
3. PMR and giant cell arteritis have common clinical and epidemiological links but need not occur synchronously: roughly **40–50%** of GCA patients have PMR features, and about **15–20%** of PMR patients develop GCA.
4. **Geriatric relevance:** Pathy's lists PMR among the major causes of **frailty**, acting through pain, immobility and reduced food intake — and stresses that **treatment with corticosteroids reverses the frailty it produces, yet this totally reversible condition is often misdiagnosed by clinicians**.

B. Aetiology and Pathophysiology

5. Aetiology is unknown; there is a genetic association with **HLA-DRB1*04**, and environmental or infectious triggers have been proposed given seasonal and cyclical clustering.
6. The dominant mechanism is **IL-6 driven systemic inflammation** with dendritic cell and CD4+ T-cell activation. Unlike myositis, the muscle itself is normal — the pathology lies in **peri-articular structures: subacromial and subdeltoid bursitis, glenohumeral synovitis, biceps tenosynovitis and trochanteric bursitis**.
7. Ageing-related **immunosenescence and inflammaging** explain the restriction of PMR to older adults.

C. Clinical Features

8. **Cardinal symptoms:** symmetrical aching and stiffness of the shoulder girdle (most common), neck and hip girdle, of relatively abrupt onset, worst in the morning and after rest, with **morning stiffness lasting more than 45–60 minutes**.
9. **Functional impact:** difficulty getting out of bed, standing up from a chair, lifting the arms to comb the hair, and turning over in bed — a classic geriatric presentation of "off legs" or "gone off her feet".
10. **Constitutional symptoms** (Kane's list): **weight loss, fever, muscle pain, aching of the neck, shoulder and hip, and morning stiffness**; also fatigue, malaise, anorexia, depression and low-grade pyrexia. PMR is a recognised cause of **pyrexia of unknown origin** in the elderly.
11. **Examination:** painful restriction of active shoulder and hip movement with preserved passive range; **muscle strength is normal** once pain is overcome, and there is no muscle wasting or tenderness disproportionate to pain. A peripheral asymmetrical arthritis, carpal tunnel syndrome and distal **RS3PE**-like pitting oedema may occur.
12. **Always screen for GCA** at every visit: headache, scalp tenderness, jaw claudication, visual symptoms, and temporal artery abnormality.

D. Investigations and Diagnosis

13. **ESR and CRP** are markedly elevated — ESR classically above **40 mm/hour**. About 7–20% have a normal ESR at presentation, so a normal ESR with a typical picture and high CRP does not exclude PMR.
14. **Normal creatine kinase, normal electromyography and normal muscle biopsy** — the single most useful discriminator from inflammatory myopathy. Normocytic normochromic anaemia of chronic disease, thrombocytosis, raised alkaline phosphatase and hypoalbuminaemia are common. Rheumatoid factor and anti-CCP are negative.
15. **Exclusion is essential** before committing to long-term steroids: full blood count, renal and liver function, calcium, TSH, CK, protein electrophoresis, ANA, RF/anti-CCP, chest radiograph, and vitamin D. Consider PSA in men and imaging if malignancy is suspected.
16. **Imaging:** ultrasound showing bilateral subacromial/subdeltoid bursitis or biceps tenosynovitis supports the diagnosis and is part of the **2012 EULAR/ACR classification criteria** (age ≥50, bilateral shoulder pain and raised ESR/CRP are mandatory, then scored on morning stiffness >45 minutes, hip involvement, absence of RF/anti-CCP, and absence of other joint involvement; ≥4 points without ultrasound, ≥5 with). PET-CT may show large-vessel uptake and detect coexisting GCA.
17. **Differential diagnosis:** late-onset rheumatoid arthritis, inflammatory myopathy, hypothyroidism, statin myopathy, osteoarthritis of the cervical spine and shoulders, rotator cuff disease, fibromyalgia, myeloma, occult malignancy, infective endocarditis, and Parkinson's disease.

E. Management

18. **Corticosteroids are the treatment** — typically **prednisolone 12.5–25 mg/day** (commonly 15 mg). Response is dramatic: near-complete relief of symptoms within 24–72 hours. **Failure to respond promptly should make you question the diagnosis.**
19. **Tapering:** reduce to 10 mg/day over 4–8 weeks, then by 1 mg every 4 weeks, guided by symptoms rather than ESR

alone. Most need **1–2 years** of treatment; relapse occurs in over half during tapering.

20. **Methotrexate has been used as a corticosteroid-sparing agent and may be useful for patients with frequent disease relapses and/or corticosteroid-related toxicity** (Kane's) — typically 7.5–15 mg weekly with folic acid.
21. **Infliximab has not been shown to be beneficial** in terms of pain management or disease progression and **is not currently recommended** (Kane's, citing Hernández-Rodríguez).
22. **Mandatory steroid-sparing co-prescription in the elderly:** calcium and vitamin D, **bone protection with a bisphosphonate**, gastroprotection where NSAIDs or high steroid doses are used, glucose monitoring for steroid-induced diabetes, blood pressure and weight monitoring, and cataract and glaucoma surveillance. Consider *Pneumocystis* prophylaxis at high cumulative doses.
23. **Escalate vigilance to GCA:** because of the close association between PMR and temporal arteritis, **any symptoms suggestive of temporal artery involvement — headache, jaw claudication, recent changes in vision — especially when the sedimentation rate is very high (>75 mm/hour), should prompt consideration of temporal artery biopsy** (Kane's).
24. **Non-drug care:** graded exercise to prevent deconditioning and steroid myopathy, fall prevention, protein-adequate nutrition, and management of steroid-related mood change and insomnia.

F. Prognosis

25. Prognosis is **good** — most achieve remission and stop steroids within 1–3 years, though a minority need low-dose maintenance for longer. Morbidity in the elderly comes largely from **cumulative glucocorticoid toxicity**: osteoporotic fracture, diabetes, infection, cataract, skin fragility and steroid myopathy.

Latest Updates (2023 – Present)

- **Sarilumab (Kevzara) was approved by the FDA on 28 February 2023 as the first and only biologic for polymyalgia rheumatica** in adults with an inadequate response to corticosteroids or who cannot tolerate a corticosteroid taper; the European Commission followed in November 2024.
- The approval rested on the phase 3 **SAPHYR trial (NEJM, October 2023)**: patients with glucocorticoid-resistant PMR who flared on ≥7.5 mg/day prednisone were randomised to sarilumab 200 mg every 2 weeks with a 14-week steroid taper, or placebo with a 52-week taper. **Sustained remission at 52 weeks was 28% with sarilumab versus 10% with placebo** ($p = 0.019$) — nearly three times as many patients — with markedly lower cumulative steroid exposure. Neutropenia occurred in about 3%.
- Prescribing caution with sarilumab: avoid if the absolute neutrophil count is below 2000/mm³, platelets below 150,000/mm³, or transaminases more than 1.5 times the upper limit of normal.
- **Tocilizumab** (SEMAPHORE and PMR-SPARE trials) has also shown steroid-sparing efficacy in PMR, although it is not formally licensed for the indication.
- Current expert practice reserves IL-6 blockade for **relapsing PMR, steroid-dependent disease, or patients in whom steroid toxicity is unacceptable** — a group that includes many frail older adults with diabetes or osteoporosis.

Current Practice Update

Issue	Textbook position (Kane's / Pathy's)	Current position (2023–present)
Steroid-sparing agent	Methotrexate for frequent relapse or steroid toxicity	Methotrexate retained, but **sarilumab is now the first licensed biologic** (FDA 2023, EU 2024)
TNF inhibitors	Infliximab not beneficial, not recommended	Unchanged — TNF blockade has no role in PMR
Starting steroid dose	"Steroids such as prednisone"	Standardised at **12.5–25 mg/day prednisolone**, individualised by weight and comorbidity
Role of imaging	Not emphasised	Ultrasound bursitis included in **2012 EULAR/ACR criteria**; PET-CT to detect occult large-vessel GCA

Extra Recall Scaffold — Recurring Topic (asked twice)

Mnemonic for the diagnostic pillars — "PMR 50": **P**ain and stiffness in shoulder/hip girdle · **M**orning stiffness >45 minutes · **R**aised ESR/CRP · age **≥50** · and a rapid **response to 15 mg prednisolone**.

Numerical anchors: age ≥50 · F:M 2–3:1 · morning stiffness >45 min · ESR >40 mm/h · ESR >75 mm/h with cranial symptoms → biopsy · prednisolone 12.5–25 mg · response in 24–72 hours · taper to 10 mg over 4–8 weeks · then 1 mg every 4 weeks · treatment 1–2 years · 15–20% develop GCA · SAPHYR 28% vs 10%.

Examiner traps: (1) Calling it a myopathy — **CK, EMG and muscle biopsy are normal**; the weakness is pain-limited, not true weakness. (2) Starting steroids before excluding myeloma, malignancy and infection. (3) Missing coexisting GCA — ask about jaw claudication and visual symptoms at **every** visit. (4) Giving high dose (≥40 mg) prednisolone for uncomplicated PMR — that dose belongs to GCA. (5) Forgetting bone protection from day one. (6) Tapering by ESR alone rather than by symptoms.

15-second answer skeleton: Define → epidemiology and link to GCA → frailty relevance from Pathy's → IL-6 driven peri-articular bursitis, not myositis → girdle pain with morning stiffness and constitutional features → raised ESR/CRP with normal CK → EULAR/ACR criteria and exclusions → prednisolone 15 mg with dramatic response and slow taper → methotrexate, and now sarilumab → bone and steroid-toxicity protection → watch for GCA → good prognosis.

Exam One-Liners

- PMR is a peri-articular bursitis syndrome, not a muscle disease — CK is normal.
- Age ≥50 is essential to the diagnosis.
- Failure to respond to 15 mg prednisolone within 72 hours means rethink the diagnosis.
- ESR >75 mm/h with headache or visual change mandates consideration of temporal artery biopsy.
- Infliximab does not work; methotrexate is the classical steroid-sparing agent.
- Sarilumab (SAPHYR, 2023) is the first licensed biologic for PMR.
- Pathy's lists PMR as a fully reversible cause of frailty that is commonly missed.

Q10. Temporal Arteritis (Giant Cell Arteritis) — 15 MARKS

A. Definition and Epidemiology

1. **Giant cell arteritis (GCA), or temporal arteritis**, is a **granulomatous large- and medium-vessel vasculitis** affecting the extracranial branches of the carotid artery, the aorta and its major branches, occurring almost exclusively in people **over 50 years**.
2. It is the **commonest primary systemic vasculitis of older adults**; peak incidence is between **70 and 80 years**, with women affected two to three times more often than men. Incidence is highest in Northern European populations and lower in India and Asia, although under-diagnosis contributes.
3. Men are more likely to have **ocular manifestations**. GCA and PMR are closely linked; Pathy's also notes that GCA must be considered as a cause of **cerebrovascular disease and vertigo** in older people.
4. It is a **medical emergency** — untreated it causes irreversible blindness, aortic aneurysm and stroke.

B. Pathology and Pathophysiology

5. Dendritic cells in the adventitia are activated, recruiting CD4+ T cells that differentiate into **Th1 (interferon-γ)** and **Th17 (IL-17)** lineages; macrophages fuse to form **multinucleate giant cells**.
6. **Histology:** transmural granulomatous panarteritis with mononuclear infiltrate, giant cells adjacent to a **fragmented internal elastic lamina**, and **intimal hyperplasia causing luminal occlusion** — the intimal proliferation, not the inflammation itself, causes the ischaemic events.
7. Involvement is **segmental and patchy ("skip lesions")**, which is why a short or unilateral biopsy may be falsely negative.
8. **IL-6** drives the systemic inflammatory response and the acute-phase reactants — the rationale for tocilizumab and sarilumab.

C. Clinical Features

9. **Cranial:** new-onset **temporal or occipital headache**, **scalp tenderness** (pain on combing hair or lying on a pillow), and a thickened, tender, nodular, pulseless temporal artery.
10. **Jaw claudication** — pain in the masseters on chewing — is the **most specific symptom**; tongue claudication and, rarely, tongue or scalp necrosis occur.
11. **Visual:** amaurosis fugax, diplopia, and sudden painless monocular visual loss from **arteritic anterior ischaemic optic neuropathy** (chalky-white swollen disc), central retinal artery occlusion, or posterior ischaemic optic neuropathy. Untreated, the second eye is involved within days to weeks.
12. **Systemic:** fever, weight loss, anorexia, malaise, depression — GCA is an important cause of **pyrexia of unknown origin** in older people and may present as isolated unexplained inflammation.
13. **Polymyalgic:** shoulder and hip girdle pain and stiffness in 40–50%.
14. **Large-vessel disease:** limb claudication, asymmetrical blood pressures, absent or unequal pulses, subclavian or carotid bruits; late **thoracic aortic aneurysm and dissection**.
15. **Neurological:** transient ischaemic attack and stroke, predominantly in the **vertebrobasilar territory**; GCA-related stroke is defined as an ischaemic infarct within three to four weeks of diagnosis and treatment.

D. Investigations

16. **ESR and CRP** are markedly elevated; **ESR is often above 75–100 mm/hour**. CRP is the more sensitive; up to 4% of biopsy-proven cases have normal inflammatory markers.
17. **Supporting bloods:** normocytic anaemia, thrombocytosis, raised alkaline phosphatase, hypoalbuminaemia, raised IL-6.
18. **Temporal artery biopsy** is the diagnostic gold standard. Practical rules: take a segment of at least **1–2 cm** with serial sectioning to overcome skip lesions; consider bilateral biopsy; **do not delay steroids for the biopsy** — histology remains positive for up to **2–4 weeks** after starting treatment. Kane's emphasises that any symptom suggestive of temporal artery involvement — headache, jaw claudication, recent visual change — especially with an ESR above 75 mm/hour, should prompt consideration of temporal artery biopsy.
19. **Colour Doppler ultrasound** of temporal and axillary arteries showing the non-compressible **"halo sign"** is now a first-line, non-invasive test.
20. **Cross-sectional imaging** for large-vessel disease: **FDG-PET/CT**, CT angiography or MR angiography of the aorta and branches; MRI of the cranial vessels with contrast can show mural enhancement.
21. **1990 ACR classification criteria** (3 of 5): age at onset ≥50 years, new headache, temporal artery abnormality, ESR ≥50 mm/hour, and abnormal artery biopsy. The **2022 ACR/EULAR criteria** add imaging findings and a positive biopsy weighting.

E. Management

22. **Acute treatment of temporal arteritis with high-dose steroids is needed to prevent blindness** (Kane's) — **start immediately on clinical suspicion, before biopsy**.
23. **Dosing:** uncomplicated GCA — **prednisolone 40–60 mg/day**. Visual loss or amaurosis fugax — **IV**

methylprednisolone 500–1000 mg/day for 3 days then high-dose oral prednisolone. Visual loss already established in one eye is rarely reversed; treatment protects the **fellow eye**.

24. **Taper** slowly over 12–24 months, guided by symptoms and CRP; most patients need **1–2 years or longer**, and relapse is common.
25. **Low-dose aspirin 75–150 mg** may be added to reduce ischaemic events, weighed against gastrointestinal bleeding risk with steroids.
26. **Steroid-sparing therapy: tocilizumab** (IL-6 receptor blocker, GiACTA trial) is licensed for GCA; **methotrexate** is an alternative with modest evidence.
27. **Mandatory adjuncts in older adults:** calcium, vitamin D and a **bisphosphonate from day one**; gastroprotection; monitoring for steroid-induced diabetes, hypertension, cataract, glaucoma, insomnia, mood disturbance, infection and steroid myopathy; and falls prevention.
28. **Long-term surveillance:** annual assessment for **thoracic aortic aneurysm** by imaging, since aortic complications occur years after the acute illness.

F. Prognosis

29. With prompt treatment, visual loss is largely preventable and life expectancy approaches that of the general population; the dominant long-term burden is **glucocorticoid toxicity**, followed by aortic aneurysm and relapse.

Latest Updates (2023 – Present)

- **Upadacitinib was approved by the FDA on 29 April 2025 for adults with giant cell arteritis** — the first and only oral JAK inhibitor for GCA, and the first new class since tocilizumab in 2017. The European Commission approved it in early April 2025. Dose is **15 mg once daily**.
- Approval was based on the phase 3 **SELECT-GCA trial** (NEJM 2025; 428 patients, 70% new-onset): **sustained remission from week 12 to week 52 was achieved by 46.4–46.6% on upadacitinib 15 mg with a 26-week steroid taper versus 29% on placebo** ($p = 0.002$), with a safety profile consistent with other indications and no major cardiovascular events in the trial.
- Upadacitinib should **not** be combined with other JAK inhibitors, biologic DMARDs, or potent immunosuppressants such as azathioprine and ciclosporin; standard JAK-class cautions on thrombosis, herpes zoster and malignancy apply, which matters in elderly patients.
- **Tocilizumab remains the other licensed steroid-sparing option**, and the choice between an oral JAK inhibitor and a subcutaneous IL-6 blocker is now a real clinical decision.
- **Fast-track GCA pathways** with same-day **ultrasound halo assessment** are increasingly replacing routine biopsy as the initial diagnostic step in specialist centres, in line with EULAR imaging recommendations; PET-CT is used for large-vessel and occult disease.
- Recognition of **GCA-related stroke** as an ischaemic infarct occurring within three to four weeks of diagnosis and treatment has sharpened attention to the vertebrobasilar territory and to thromboembolic morbidity in this population.

Current Practice Update

Issue	Textbook position	Current position (2023–present)
First diagnostic test	Temporal artery biopsy	**Colour Doppler ultrasound (halo sign)** first-line in fast-track clinics; biopsy if ultrasound is negative but suspicion persists
Steroid-sparing agents	Steroids alone; methotrexate occasionally	**Tocilizumab** (2017) and **upadacitinib** (2025) both licensed
Steroid taper duration	Prolonged, symptom-guided	Trials now use structured **26-week** tapers when a steroid-sparing agent is co-prescribed
Long-term follow-up	Not emphasised	Routine surveillance for **thoracic aortic aneurysm**

Extra Recall Scaffold — Recurring Topic (asked twice)

Mnemonic for cranial GCA — "HAT-JAWS": Headache (new) · **A**ge ≥50 · **T**emporal artery tender/nodular/pulseless · **J**aw claudication · **A**maurosis / visual loss · **W**eight loss and fever · **S**calp tenderness and high **S**edimentation rate.

Numerical anchors: age ≥50 · peak 70–80 years · ESR ≥50 for ACR criteria, often >75–100 · biopsy segment 1–2 cm · biopsy stays positive 2–4 weeks after steroids · prednisolone 40–60 mg, or IV methylprednisolone 500–1000 mg × 3 days if visual loss · 40–50% have PMR · ACR criteria: 3 of 5 · SELECT-GCA 46% vs 29% · upadacitinib 15 mg.

Examiner traps: (1) Delaying steroids until the biopsy is done — the commonest and most damaging error. (2) Assuming a

normal ESR excludes GCA — up to 4% are normal; check CRP. (3) Assuming a negative biopsy excludes it — skip lesions. (4) Using PMR doses (15 mg) for GCA. (5) Forgetting bone protection and gastroprotection from day one. (6) Forgetting that established visual loss is usually permanent — treatment saves the **other** eye. (7) Overlooking large-vessel GCA presenting only as PUO or limb claudication without headache.

15-second answer skeleton: Define as granulomatous large-vessel vasculitis ≥50 years → emergency → pathology (giant cells, fragmented internal elastic lamina, intimal hyperplasia, skip lesions) → cranial, ocular, systemic, polymyalgic, large-vessel and neurological features → ESR/CRP, biopsy rules, ultrasound halo, PET → ACR criteria → immediate high-dose steroids (IV if visual loss) → aspirin, tocilizumab, upadacitinib → bone protection and steroid toxicity monitoring → aneurysm surveillance → prognosis.

Exam One-Liners

- Jaw claudication is the most specific symptom of GCA.
- Never delay steroids for a biopsy — histology remains positive for 2–4 weeks.
- Visual loss is usually irreversible; treatment protects the fellow eye.
- Skip lesions make a 1–2 cm biopsy with serial sections essential.
- Halo sign on temporal artery ultrasound is the modern first-line test.
- Tocilizumab (2017) and upadacitinib (2025) are the licensed steroid-sparing drugs.
- Screen survivors for thoracic aortic aneurysm.

Q11. Late-Onset Rheumatoid Arthritis — 15 MARKS

A. Definition and Epidemiology

1. **Late-onset rheumatoid arthritis (LORA)**, also called elderly-onset RA, is rheumatoid arthritis with first symptom onset at or after **60–65 years**, as distinct from **young-onset RA growing old** — a distinction that must be made explicitly in the answer.
2. It accounts for roughly **10–33%** of all RA. The female preponderance of young-onset disease is lost — the **sex ratio approaches 1:1**.
3. Pathy's notes rheumatoid arthritis as a recognised cause of **secondary osteoporosis** and as a factor in FRAX, and RA joint pain as a classical driver of **immobility and frailty** in older people.

B. Pathogenesis

4. The same autoimmune synovitis as classical RA — citrullination of self-proteins, anti-citrullinated peptide antibody formation, T- and B-cell activation, and cytokine-driven pannus formation with TNF-α, IL-1 and IL-6.
5. **Immunosenescence** shapes the phenotype: thymic involution, expansion of CD4+CD28− senescent T cells, telomere shortening and "inflammaging" produce a more **acute-onset, cytokine-driven, seronegative and polymyalgic** picture.
6. HLA-DRB1 shared-epitope association is weaker than in young-onset disease.

C. Clinical Features — How It Differs from Young-Onset RA

Feature	Young-onset RA	Late-onset RA
Sex ratio	Female 3:1	**Approximately equal**
Onset	Insidious over weeks to months	**Acute or abrupt**, often "overnight"
Joint distribution	Small joints of hands and feet	**Large proximal joints — shoulders, hips, knees**
Constitutional symptoms	Mild	**Prominent** — fever, weight loss, fatigue
Rheumatoid factor / anti-CCP	Positive in most	More often **seronegative**
ESR / CRP	Raised	**Very high**, often mimicking PMR
Rheumatoid nodules and extra-articular disease	Common	**Less common**
Erosions	Progressive	Variable — seropositive LORA erodes as aggressively or faster

7. **Three recognised subsets:** (i) a classical RA-like polyarthritis; (ii) a **PMR-like syndrome** with proximal girdle stiffness; (iii) **RS3PE** — remitting seronegative symmetrical synovitis with pitting oedema of the hands, which is strongly age-associated, HLA-B7 linked, exquisitely steroid-responsive, and may herald occult malignancy.
8. **Presentation is often functional**: sudden inability to dress, grip or walk, presenting as "off legs" or acute functional decline rather than as joint complaint.
9. **Extra-articular features** when present: interstitial lung disease, anaemia of chronic disease, secondary Sjögren's syndrome, vasculitis, Felty's syndrome, scleritis, pericarditis, and amyloidosis. Cervical spine involvement with **atlanto-axial subluxation** is a specific anaesthetic hazard.

D. Investigations

10. **Rheumatoid factor and anti-CCP** — anti-CCP is far more specific, since **RF rises with age and is positive in up to 10–25% of healthy older people**, making a low-titre RF alone almost meaningless in this age group.
11. **ESR, CRP, full blood count** (anaemia of chronic disease, thrombocytosis), renal and liver function — the latter two also determine DMARD choice.
12. **Radiographs** of hands and feet for periarticular osteopenia, joint space narrowing and marginal erosions; **ultrasound or MRI** for early synovitis and erosion detection.
13. **Pre-treatment screen:** chest radiograph, hepatitis B and C serology, tuberculosis screening (crucial in India before any biologic — Mantoux/IGRA plus chest X-ray), and vaccination status.
14. **2010 ACR/EULAR classification criteria** (score ≥6/10 from joint involvement, serology, acute-phase reactants and symptom duration ≥6 weeks) are used, though they perform less well in seronegative late-onset disease.
15. **Differential diagnosis is the crux: polymyalgia rheumatica** (no true synovitis, no erosions, responds to 15 mg prednisolone), **calcium pyrophosphate deposition disease / pseudogout**, polyarticular gout, paraneoplastic arthritis, viral arthritis, hypothyroidism, and erosive osteoarthritis of the hands.

E. Management

16. **Treat-to-target** applies equally in old age — aim for remission or low disease activity using DAS28, with review every 1–3 months. **Age alone is not a reason for therapeutic nihilism**, since uncontrolled RA causes disability, sarcopenia, osteoporosis and excess cardiovascular death.
17. **Methotrexate remains the anchor drug**, starting 7.5–10 mg weekly with folic acid. Pathy's cautions that there is a natural 30% decrease in glomerular filtration rate with ageing, in addition to further potential renal insults, so **caution must be taken with dosing methotrexate in older patients with renal impairment**. Monitor full blood count, liver function and creatinine.
18. **Alternative csDMARDs:** leflunomide, sulfasalazine, hydroxychloroquine (useful in mild disease and safest in the frail; needs retinal screening).
19. **Glucocorticoids** give rapid control as bridging therapy at the lowest effective dose for the shortest time; in older adults the cumulative harms — osteoporosis, diabetes, infection, cataract, skin fragility — are magnified. **Osteoporosis and gastrointestinal ulcer prophylaxis should be employed in all patients treated with oral steroids to minimise potentially devastating complications** (Pathy's, on steroid use in older adults).
20. **Biologic DMARDs** — TNF inhibitors, abatacept, tocilizumab, rituximab — are effective in older adults, though with a higher absolute infection risk. Abatacept has a comparatively favourable infection profile in the elderly.
21. **JAK inhibitors** (tofacitinib, baricitinib, upadacitinib) are effective orally, but the **ORAL Surveillance** safety signal restricts use in those over 65 with cardiovascular risk factors or a smoking history, because of increased major adverse cardiovascular events, venous thromboembolism and malignancy.
22. **NSAIDs should be minimised** — they appear on the AGS Beers Criteria and cause bleeding, renal impairment and heart failure in this age group.
23. **Comprehensive care:** physiotherapy and occupational therapy for joint protection and assistive devices, exercise to counter sarcopenia, cardiovascular risk assessment (RA is an independent cardiovascular risk factor), osteoporosis screening and treatment, vaccination (influenza, pneumococcal, herpes zoster — inactivated preparations only on immunosuppression), and management of depression and caregiver burden.
24. **Prognosis:** seropositive, erosive late-onset RA behaves as aggressively as young-onset disease and causes greater functional loss because of lower physiological reserve; seronegative and PMR-like variants often have a milder, more steroid-responsive course.

Latest Updates (2023 – Present)

- **ACR 2021 and EULAR 2022/2023 recommendations** both retain methotrexate as the anchor and treat-to-target as the strategy, but now place **explicit age- and comorbidity-based restrictions on JAK inhibitors** following ORAL Surveillance — directly relevant to the geriatric population.
- Registry data (2023–2025) show that biologic-treated older adults have higher serious infection rates than younger patients but comparable efficacy, supporting **risk-stratified rather than age-restricted** prescribing.
- Growing evidence that RA is associated with accelerated **sarcopenia ("rheumatoid cachexia")** and frailty, with resistance exercise and adequate protein intake now part of standard care.
- Interest in **difficult-to-treat RA** as a defined entity, and in newer targets (olokizumab, peresolimab, CAR-T for refractory autoimmunity) at trial stage.
- **Indian context:** mandatory tuberculosis screening before biologics is critical given high latent TB prevalence; biosimilar TNF inhibitors and rituximab have greatly improved affordability, with treatment cost cover under AB-PMJAY.

Current Practice Update

Issue	Textbook position	Current position
Treatment intensity in the elderly	Cautious, comorbidity-limited	**Treat-to-target regardless of age**; comorbidity guides drug choice, not intensity
JAK inhibitors	Effective oral option	**Restricted in over-65s** with cardiovascular risk or smoking history (ORAL Surveillance)
Methotrexate in renal impairment	Dose with caution	Unchanged and reinforced — check eGFR at every escalation
Muscle	Not emphasised	**Rheumatoid cachexia / sarcopenia** is now a treatment target

Exam One-Liners

- Late-onset RA begins at or after 60–65 years — different from young-onset RA that has aged.
- Equal sex ratio, abrupt onset, large proximal joints, more seronegative, very high ESR.
- Three subsets: classical polyarthritis, PMR-like, and RS3PE.
- RF is positive in up to a quarter of healthy older people — use anti-CCP.
- Methotrexate remains the anchor drug, dosed by renal function.

- JAK inhibitors are restricted above 65 years after ORAL Surveillance.
- Always screen for latent tuberculosis before a biologic in India.

Q12. Sjögren's Syndrome — Definition, Epidemiology, Pathophysiology, Presentation and Management

15 MARKS

A. Definition

1. **Sjögren's syndrome (Sjögren's disease)** is a chronic **systemic autoimmune exocrinopathy** characterised by lymphocytic infiltration and destruction of the exocrine glands — principally the lacrimal and salivary glands — producing the **sicca complex of dry eyes (keratoconjunctivitis sicca) and dry mouth (xerostomia)**, together with extraglandular systemic involvement.
2. **Primary Sjögren's** occurs alone; **secondary Sjögren's** occurs with another connective tissue disease, most often **rheumatoid arthritis**, systemic lupus erythematosus or systemic sclerosis.

B. Epidemiology

3. Prevalence is approximately **0.1–0.6%**; it is one of the commonest systemic autoimmune diseases.
4. **Female to male ratio is about 9:1**, with a bimodal peak in the 20s–30s and again **after the menopause, around 50–60 years**, so a large proportion of patients are older adults.
5. **Geriatric caution:** sicca symptoms are extremely common in old age from **age-related glandular atrophy, dehydration and anticholinergic polypharmacy** — dryness in an older person is far more often drug-induced than autoimmune, and this distinction is the crux of any exam answer.

C. Pathophysiology

6. **Genetic susceptibility** (HLA-DR3/DQ2, HLA-DRB1*03, IRF5 and STAT4 polymorphisms) plus an environmental trigger, classically a **viral infection** (EBV, HCV, HTLV-1), initiates glandular epithelial activation — hence the concept of **"autoimmune epithelitis"**.
7. Activated epithelial cells present autoantigens (**Ro/SSA and La/SSB**) and secrete chemokines that recruit **CD4+ T cells and B cells**, forming **periductal focal lymphocytic infiltrates** and, in some, ectopic germinal centres.
8. **Type I interferon signature** and **BAFF (B-cell activating factor)** overexpression drive B-cell survival, autoantibody production and hypergammaglobulinaemia — the rationale for anti-BAFF and B-cell directed therapy.
9. Glandular destruction, plus **anti-muscarinic M3 receptor antibodies** impairing residual secretory function, produces the sicca picture — explaining why dryness can exceed the degree of histological damage.
10. Persistent B-cell stimulation with germinal centre formation underlies the **risk of B-cell non-Hodgkin lymphoma**, typically MALT lymphoma of the parotid.

D. Clinical Presentation

(i) Glandular

11. **Ocular:** gritty, sandy foreign-body sensation, burning, photophobia, redness, blurring, stringy discharge; complications include filamentary keratitis and corneal ulceration.
12. **Oral:** dry mouth, difficulty swallowing dry food, need to sip water while talking, altered taste, tongue fissuring and depapillation, **rampant dental caries**, angular cheilitis and recurrent oral candidiasis.
13. **Salivary gland enlargement** — recurrent or persistent parotid or submandibular swelling. **A persistently hard, unilateral, enlarging parotid must raise suspicion of lymphoma.**
14. Other exocrine sites: dry nose, dry cough from xerotrachea, dry skin, and vaginal dryness with dyspareunia — a common and under-asked symptom in older women.

(ii) Extraglandular

15. **Constitutional:** profound **fatigue** (often the dominant complaint), low-grade fever, myalgia.
16. **Musculoskeletal:** non-erosive symmetrical polyarthritis and arthralgia; Raynaud's phenomenon.
17. **Cutaneous:** annular erythema and **palpable purpura from hypergammaglobulinaemic or cryoglobulinaemic vasculitis**.
18. **Renal: distal (type 1) renal tubular acidosis** with hypokalaemia and nephrocalcinosis; interstitial nephritis; less often glomerulonephritis with cryoglobulinaemia.
19. **Pulmonary:** interstitial lung disease (NSIP, LIP), bronchiectasis, xerotrachea.
20. **Neurological: peripheral sensory or sensory-ataxic neuropathy**, small-fibre neuropathy with burning feet, trigeminal neuropathy, mononeuritis multiplex, and rarely CNS disease.
21. **Haematological / malignant:** cytopenias, hypergammaglobulinaemia, and a **5–16 fold** increased risk of **non-Hodgkin lymphoma** — the single most important complication. Predictors: persistent parotid enlargement, purpura, low C4, cryoglobulinaemia, lymphadenopathy, splenomegaly and a low CD4 count.
22. **Obstetric/other:** anti-Ro antibodies cause congenital heart block in offspring (not relevant in the elderly but examinable).

E. Investigations

23. **Autoantibodies: anti-Ro/SSA** (most sensitive and part of the criteria) and **anti-La/SSB**; ANA positive in most; **rheumatoid factor positive in about 50%** even without RA.
24. **Supporting bloods:** raised ESR with normal CRP, **polyclonal hypergammaglobulinaemia**, anaemia, leucopenia, low C3/C4 (poor prognosis), cryoglobulins, and serum free light chains.
25. **Ocular tests: Schirmer's test ≤5 mm in 5 minutes**; **ocular staining score ≥5** with lissamine green or fluorescein (or Rose Bengal ≥4 by van Bijsterveld); tear break-up time.
26. **Oral tests:** unstimulated whole salivary flow **≤0.1 mL/minute**; salivary gland ultrasound showing a heterogeneous hypoechoic gland; sialography and scintigraphy are older methods.
27. **Minor labial salivary gland biopsy** — the histological gold standard; a **focus score ≥1** (one focus of ≥50 lymphocytes per 4 mm²) is diagnostic.
28. **2016 ACR/EULAR classification criteria** (score ≥4): anti-Ro/SSA positive (3), focus score ≥1 (3), ocular staining score ≥5 (1), Schirmer ≤5 mm/5 min (1), unstimulated salivary flow ≤0.1 mL/min (1). Exclusions include head and neck radiotherapy, hepatitis C, HIV, sarcoidosis, amyloidosis, IgG4-related disease and graft-versus-host disease.
29. **Disease activity tools: ESSDAI** for systemic activity and **ESSPRI** for patient-reported dryness, pain and fatigue.

F. Management

30. **No cure exists;** treatment is symptomatic for sicca and immunosuppressive for systemic disease. First, **stop or substitute anticholinergic drugs** — tricyclics, oxybutynin, antihistamines, antipsychotics, diuretics — which is frequently the highest-yield intervention in an older patient.
31. **Dry eyes:** preservative-free artificial tears and gels, ocular lubricant ointment at night, humidification, avoidance of fans and air conditioning; **topical ciclosporin 0.05%** or short-course topical steroid for inflammatory dryness; **punctal plugs or cautery** for refractory cases; treat coexisting blepharitis.
32. **Dry mouth:** frequent sips of water, sugar-free gum and lozenges, saliva substitutes, meticulous oral hygiene with **high-fluoride toothpaste and 3-monthly dental review**, and prompt treatment of candidiasis with nystatin or fluconazole (avoid sugar-containing preparations).
33. **Muscarinic agonists: pilocarpine 5 mg four times daily** or **cevimeline 30 mg three times daily** stimulate residual secretion. Cautions in older adults: sweating, flushing, nausea, urinary frequency, bradycardia; avoid in uncontrolled asthma, narrow-angle glaucoma and significant cardiac disease.
34. **Musculoskeletal disease: hydroxychloroquine** for arthralgia, fatigue and cutaneous disease (though RCT evidence for fatigue is weak); short courses of NSAIDs with caution; low-dose prednisolone for flares.
35. **Severe systemic disease** (vasculitis, interstitial lung disease, glomerulonephritis, severe neuropathy, cytopenias): glucocorticoids with **methotrexate, azathioprine, mycophenolate or cyclophosphamide**; **rituximab** for cryoglobulinaemic vasculitis, severe parotid swelling and lymphoma-associated disease, although the large TEARS and TRACTISS trials failed to show benefit for dryness itself.
36. **Vaginal dryness:** non-hormonal lubricants and, where appropriate, topical vaginal oestrogen — frequently overlooked in older women.
37. **Lymphoma surveillance:** examine glands and lymph nodes at each visit; investigate any persistent hard parotid swelling, new lymphadenopathy, falling C4, monoclonal band or cryoglobulins with imaging and biopsy.
38. **Prognosis:** life expectancy is near-normal in uncomplicated disease; excess mortality is driven by **lymphoma, vasculitis and interstitial lung disease**. Fatigue and dryness dominate quality of life.

Latest Updates (2023 – Present)

- **Ianalumab**, a dual-action B-cell targeting monoclonal antibody against BAFF-receptor, met its primary endpoint in the **paired phase 3 NEPTUNUS-1 and NEPTUNUS-2 trials** presented at ACR Convergence in October 2025 — the **first phase 3 studies in Sjögren's disease to meet their primary objective**, showing statistically significant improvement in ESSDAI. Phase 2b data had already shown sustained efficacy of the 300 mg dose at 52 weeks with a favourable 2-year safety profile.
- **Nipocalimab**, an FcRn blocker, significantly reduced disease activity and severity in moderate-to-severe disease in the phase 2 **DAHLIAS** trial (published in The Lancet, October 2025), with substantial reduction in Sjögren's-related autoantibodies. It holds FDA **Breakthrough Therapy Designation (November 2024)** and **Fast Track Designation (April 2025)**, with the phase 3 **DAFFODIL** study now enrolling.
- **Dazodalibep**, a non-antibody CD40-ligand antagonist, met its primary endpoint in both study populations — those with systemic disease activity and those with high symptom burden — in a phase 2 trial published in Nature Medicine (2024), significantly reducing disease activity and improving symptoms.
- **Iscalimab (anti-CD40)** improved and sustained disease activity in a phase 2b Lancet study; **telitacicept** (BAFF/APRIL blocker) and low-dose IL-2 improved ESSDAI in phase 2 trials.
- **Important negative signal:** across trials, none of these agents — nor rituximab, abatacept, infliximab, tocilizumab and others in a 2023 meta-analysis — has reliably improved **unstimulated salivary flow** or objective ocular parameters. Benefit is on systemic activity and symptoms, not on glandular secretion, and early intervention (within 3 years of disease onset) may be needed to affect flow.
- Practical consequence: the field is moving from a single "Sjögren's" label towards **endotype-directed therapy**, targeting the B-cell/interferon-high subset with biologics while continuing symptomatic and secretagogue therapy for

dryness.

Current Practice Update

Issue	Textbook position	Current position (2023–present)
Disease-modifying therapy	None — symptomatic treatment plus immunosuppression for organ disease	First **positive phase 3 programme (ianalumab, NEPTUNUS 2025)**; nipocalimab, dazodalibep, iscalimab, telitacicept in advanced development
Rituximab for dryness	Sometimes used	**Not effective for sicca** (TEARS, TRACTISS); reserve for vasculitis and lymphoma
Outcome measures	Symptom-based	**ESSDAI and ESSPRI** are the standard endpoints
Nomenclature	Sjögren's syndrome	Increasingly **"Sjögren's disease"**, reflecting primary disease status

Exam One-Liners

- Autoimmune epithelitis with lymphocytic infiltration of exocrine glands.
- Female to male ratio 9:1; second peak after the menopause.
- In older adults, exclude anticholinergic drugs before diagnosing Sjögren's.
- Anti-Ro/SSA and a labial gland focus score ≥1 carry 3 points each in the 2016 criteria.
- Schirmer ≤5 mm/5 min; unstimulated salivary flow ≤0.1 mL/min.
- Raised ESR with normal CRP and polyclonal hypergammaglobulinaemia is typical.
- Persistent hard parotid enlargement, purpura and low C4 predict lymphoma.
- Pilocarpine and cevimeline are the secretagogues; hydroxychloroquine for arthralgia.

Q13. Inflammatory Myopathies — 15 MARKS

A. Definition and Classification

1. The **idiopathic inflammatory myopathies (IIM)** are acquired, immune-mediated diseases of skeletal muscle producing **symmetrical proximal muscle weakness** with muscle inflammation, raised muscle enzymes and extramuscular involvement.
2. Pathy's classifies acquired myopathies into **idiopathic inflammatory myopathies — dermatomyositis (DM), polymyositis (PM) and inclusion body myositis (IBM)** — alongside **alcohol-related, drug-induced, endocrine and metabolic, and paraneoplastic myopathies**.
3. Modern classification adds **immune-mediated necrotising myopathy (IMNM)** and **antisynthetase syndrome**, and regards "polymyositis" as a shrinking diagnosis of exclusion.
4. **Geriatric significance: inclusion body myositis is the commonest acquired myopathy after 50 years**, and dermatomyositis in an older adult carries a high risk of **underlying malignancy**. IIM must always be separated from the far commoner **statin myopathy**, hypothyroid myopathy, PMR and sarcopenia.

B. Pathophysiology

5. **Dermatomyositis** is a **humoral, complement-mediated microangiopathy**: antibody and complement deposition (membrane attack complex) on endomysial capillaries causes capillary dropout, muscle ischaemia and the characteristic **perifascicular atrophy**. Interferon type I signature is prominent.
6. **Polymyositis** is a **cell-mediated** disease: CD8+ cytotoxic T cells invade non-necrotic muscle fibres expressing MHC class I — the histological hallmark of **endomysial inflammation**.
7. **Inclusion body myositis** combines autoimmune (CD8+ T-cell) inflammation with a **degenerative process** — rimmed vacuoles, congophilic amyloid, tau and TDP-43 aggregates, and mitochondrial abnormalities (ragged red, COX-negative fibres). This dual pathology explains its poor response to immunosuppression.
8. **Immune-mediated necrotising myopathy** shows scattered necrotic and regenerating fibres with sparse inflammation, and is associated with **anti-SRP** and **anti-HMGCR** antibodies — the latter typically statin-triggered and persisting after statin withdrawal.

C. Clinical Features

Feature	Dermatomyositis	Polymyositis	Inclusion body myositis
Age	Bimodal — children and adults >40	Adults >20	**>50 years**, commonest in the elderly
Sex	Female > male	Female > male	**Male > female**
Onset	Subacute, weeks to months	Subacute	**Insidious, over years**
Weakness pattern	Symmetrical proximal	Symmetrical proximal	**Asymmetrical; distal as well as proximal** — quadriceps and finger flexors
Skin	**Present**	Absent	Absent
Creatine kinase	High (5–50×)	High	**Normal or only mildly raised**
Response to steroids	Good	Good	**Poor / none**

9. **Core presentation:** painless, symmetrical, proximal weakness — difficulty rising from a chair or toilet, climbing stairs, and lifting the arms above the head. Myalgia is present in only about a third; **pain-free weakness is the rule**.
10. **Cutaneous features of dermatomyositis: heliotrope rash** (violaceous periorbital discolouration with oedema), **Gottron's papules** over the metacarpophalangeal and interphalangeal joints and **Gottron's sign** over elbows and knees, **shawl sign** and **V-sign** photodistributed erythema, **mechanic's hands**, periungual telangiectasia with ragged cuticles, calcinosis cutis and poikiloderma. Skin disease may precede muscle disease (**amyopathic dermatomyositis**).
11. **Extramuscular: dysphagia** from pharyngeal and upper oesophageal muscle involvement (also the commonest cause of aspiration in IBM), respiratory muscle weakness, **interstitial lung disease** (especially with antisynthetase antibodies and anti-MDA5), non-erosive arthritis, Raynaud's phenomenon, myocarditis and arrhythmia.
12. **Antisynthetase syndrome:** myositis, interstitial lung disease, arthritis, Raynaud's, fever and mechanic's hands, with **anti-Jo-1** or other anti-tRNA synthetase antibodies.
13. **Malignancy association — a key geriatric point:** dermatomyositis, particularly with **anti-TIF1-γ** or **anti-NXP2** antibodies, carries a markedly increased risk of occult cancer (ovary, lung, breast, colorectal, stomach, pancreas, and nasopharyngeal carcinoma in Asian populations). Risk is highest in the **first 1–3 years** around diagnosis. Pathy's separately recognises **paraneoplastic myopathies** as a category.
14. **Red flags for IBM:** a man over 50 with slowly progressive, asymmetrical weakness of the **quadriceps (causing falls and knee buckling) and long finger flexors (causing loss of grip)**, early dysphagia, and a CK that is normal or only mildly elevated, who has "failed steroids".

D. Investigations (Pathy's diagnostic framework)

15. **Clinical assessment** of the pattern, distribution and tempo of weakness — the first step in Pathy's myopathy chapter.
16. **Biochemical studies: creatine kinase** (the key screening test), aldolase, lactate dehydrogenase and transaminases (a raised ALT/AST from muscle is often mistaken for liver disease); also TSH, calcium, phosphate, vitamin D, potassium, renal function and ESR/CRP.
17. **Electrophysiology (EMG):** shows a **myopathic pattern** — small-amplitude, short-duration polyphasic motor unit potentials with early recruitment, plus spontaneous activity (fibrillations, positive sharp waves) indicating active inflammation. In IBM, mixed myopathic and neurogenic units are seen.
18. **Muscle biopsy** is the definitive test: perifascicular atrophy with perimysial and perivascular CD4/B-cell infiltrate in DM; endomysial CD8+ invasion of non-necrotic MHC-I expressing fibres in PM; **rimmed vacuoles with congophilic inclusions and ragged red fibres** in IBM; necrosis without inflammation in IMNM.
19. **Myositis-specific and myositis-associated antibodies:** anti-Jo-1 and other synthetases, **anti-Mi-2** (classic DM, good prognosis), **anti-TIF1-γ** and **anti-NXP2** (malignancy), **anti-MDA5** (clinically amyopathic DM with rapidly progressive ILD, high mortality), **anti-SAE**, **anti-SRP** and **anti-HMGCR** (necrotising myopathy), **anti-cN1A** (IBM).
20. **MRI of thigh muscles** with STIR sequences shows oedema and guides the biopsy site while excluding fatty replacement; useful when biopsy is difficult in a frail patient.
21. **Screening for complications and associations:** high-resolution CT chest and pulmonary function tests with DLCO for ILD, video-fluoroscopic swallow, ECG and echocardiography, and **age-appropriate malignancy screening** — CT chest/abdomen/pelvis, mammography, PSA, colonoscopy and pelvic ultrasound, repeated annually for 3 years in high-risk DM.
22. **Classification criteria:** the **2017 ACR/EULAR classification criteria** for adult and juvenile IIM have replaced the older Bohan and Peter criteria; the **2016 ACR/EULAR Myositis Response Criteria Total Improvement Score (TIS)** is the standard outcome measure in trials.

E. Differential Diagnosis (essential in the elderly)

23. **Drug-induced myopathies** — Pathy's classifies these as painful myopathy, painless myopathy, focal myopathy, periodic weakness and **acute rhabdomyolysis**; **statin-induced myopathy** is dealt with separately and is the single commonest mimic. Also gold-induced myopathy, colchicine, amiodarone, chloroquine, zidovudine, and alcohol-related myopathy.
24. **Endocrine and metabolic myopathies:** hypothyroidism, hyperthyroidism, **Graves' ophthalmopathy**, pituitary–adrenal axis disorders (Cushing's and steroid myopathy), primary hyperparathyroidism, **osteomalacia and disorders of vitamin D metabolism**.
25. **Others:** polymyalgia rheumatica (normal CK, normal EMG, normal biopsy), myasthenia gravis (fatigable, ocular/bulbar, normal CK), motor neurone disease, muscular dystrophies presenting late (facioscapulohumeral, oculopharyngeal, myotonic), sarcopenia and disuse atrophy, and electrolyte disturbance.

F. Management

26. **Corticosteroids are first-line** for DM, PM and IMNM: prednisolone **0.75–1 mg/kg/day** (or pulse methylprednisolone in severe disease), continued until strength improves and CK falls, then tapered slowly over 9–12 months.
27. **Steroid-sparing agents from the outset** in older adults, given the certainty of steroid toxicity: **methotrexate**, **azathioprine**, or **mycophenolate mofetil** — Pathy's specifically lists mycophenolate mofetil for dermatomyositis. Ciclosporin and tacrolimus are alternatives, particularly with interstitial lung disease.
28. **Intravenous immunoglobulin** for refractory or severe disease, dysphagia, or when immunosuppression is hazardous — it is now licensed for dermatomyositis. **Rituximab** is used in refractory disease and antisynthetase syndrome; cyclophosphamide for rapidly progressive ILD, often with tacrolimus.
29. **Cutaneous disease:** strict photoprotection, topical corticosteroids and calcineurin inhibitors, and **hydroxychloroquine**. Pruritus can be severe and disabling.
30. **Inclusion body myositis: no immunosuppressive therapy has been shown to alter the natural history** — management is supportive with **resistance and aerobic exercise**, occupational therapy, ankle-foot orthoses, walking aids, dysphagia assessment with diet modification, and cricopharyngeal dilatation or myotomy for severe dysphagia.
31. **Universal supportive care in the elderly:** physiotherapy and graded exercise (safe and beneficial even in active myositis), speech and language therapy for dysphagia with aspiration precautions, nutrition, **osteoporosis prophylaxis with calcium, vitamin D and a bisphosphonate from day one**, gastroprotection, glucose monitoring, screening for latent tuberculosis before immunosuppression, vaccination, and *Pneumocystis* prophylaxis on high-dose combined immunosuppression.
32. **Prognosis:** DM and PM generally respond well, with 5-year survival above 75–80%; adverse prognostic factors are older age, dysphagia, interstitial lung disease (especially anti-MDA5), cardiac involvement and underlying malignancy. IBM is **relentlessly progressive**, with most patients needing a walking aid within 5–10 years, though it does not markedly shorten life expectancy.

Latest Updates (2023 – Present)

- **Efgartigimod PH20 subcutaneous**, an FcRn blocker that accelerates degradation of pathogenic IgG, met its

primary endpoint in the phase 2 portion of the seamless phase 2/3 **ALKIVIA** study (89 adults with dermatomyositis, immune-mediated necrotising myopathy, polymyositis or antisynthetase syndrome, presented at EULAR 2025). The **least-squares mean Total Improvement Score at week 24 was 50.45 with efgartigimod versus 35.65 with placebo (p = 0.0004)**, with no new safety signals. This is the **first FcRn inhibitor study in inflammatory myopathy** and establishes proof of concept for the role of IgG autoantibodies; the phase 3 portion is fully enrolled.

- **Apremilast**, a PDE-4 inhibitor, has shown efficacy in refractory cutaneous dermatomyositis without underlying malignancy, apparently by reducing pro-inflammatory cytokine expression.
- **JAK inhibitors** (tofacitinib, baricitinib) are increasingly used off-label in refractory dermatomyositis and, notably, in **anti-MDA5 rapidly progressive interstitial lung disease**, where combination immunosuppression with a JAK inhibitor has improved historically dismal survival.
- **Intravenous immunoglobulin** is now formally approved for adult dermatomyositis following the ProDERM trial, moving it from rescue therapy towards earlier use.
- Myositis-specific antibody testing is now regarded as **central to classification, prognosis and malignancy risk stratification**, with proposals for antibody-defined phenotypic subgrouping replacing the older clinicopathological labels; **CAR-T cell therapy** has produced durable remissions in small refractory series.
- Consensus guidance now recommends **risk-stratified malignancy screening** — intensive imaging (CT chest/abdomen/pelvis ± PET-CT) for high-risk phenotypes such as anti-TIF1-γ-positive dermatomyositis in older adults, rather than blanket screening of all IIM.

Current Practice Update

Issue	Pathy's position	Current position (2023–present)
Classification	DM, PM, IBM as the idiopathic inflammatory myopathies	Expanded to include **IMNM and antisynthetase syndrome**; PM is now a diagnosis of exclusion; **antibody-based subgrouping**
Diagnostic criteria	Clinical, CK, EMG, biopsy	Same, formalised by the **2017 ACR/EULAR criteria**, with myositis antibody panels and thigh MRI
Refractory disease	Steroids plus mycophenolate, azathioprine, methotrexate	Plus **IVIG (licensed), rituximab, JAK inhibitors**, and investigational **efgartigimod (ALKIVIA)**
Exercise	Cautious	**Actively recommended**, including in active disease and IBM
Malignancy screening	Recognised as paraneoplastic myopathy	**Risk-stratified by antibody** — intensive screening for anti-TIF1-γ and anti-NXP2 dermatomyositis

Exam One-Liners

- Inclusion body myositis is the commonest acquired myopathy over 50 years and does not respond to steroids.
- IBM pattern: asymmetrical quadriceps and long finger flexor weakness with a near-normal CK.
- Dermatomyositis is complement-mediated microangiopathy with perifascicular atrophy.
- Polymyositis is CD8+ T-cell invasion of non-necrotic MHC-I positive fibres.
- Anti-TIF1-γ and anti-NXP2 mean look hard for cancer; anti-MDA5 means look hard at the lungs.
- Rimmed vacuoles equal inclusion body myositis.
- Statin-associated anti-HMGCR necrotising myopathy persists after the statin is stopped.
- Efgartigimod (ALKIVIA, 2025) is the first FcRn inhibitor to show benefit in myositis.

Q14. Psoriatic Arthropathy — 15 MARKS

A. Definition and Epidemiology

1. **Psoriatic arthritis (PsA)** is a chronic, **seronegative inflammatory arthritis associated with psoriasis**, belonging to the spondyloarthritis family, characterised by **enthesitis, dactylitis, axial disease, nail disease and skin psoriasis**.
2. It affects roughly **20–30%** of people with psoriasis; overall prevalence is about 0.1–0.25% of the population, with an **equal sex distribution** (unlike RA).
3. Peak onset is 30–50 years, but **elderly-onset PsA** (after 60) is recognised and characteristically has **more acute onset, higher inflammatory markers, more polyarticular and erosive disease, and more constitutional upset** — and is frequently misdiagnosed as late-onset RA, PMR or gout.
4. In about **85%** the skin disease precedes the arthritis (often by around 10 years); in 10–15% arthritis comes first or they appear together — so **always look for hidden psoriasis** in the scalp, retroauricular area, umbilicus, natal cleft and nails.

B. Pathogenesis

5. **Genetic:** strong familial clustering; **HLA-B27** associates with axial disease, HLA-C*06 with skin-predominant disease, and HLA-B38/B39 with peripheral arthritis.
6. **Immunological:** the **IL-23 / Th17 axis** is central — IL-23 drives Th17 and IL-17-producing resident entheseal cells; **TNF-α**, IL-17A/F and IL-22 mediate synovitis, enthesitis and new bone formation.
7. **The enthesis is the primary site** of disease (unlike the synovium in RA); mechanical stress ("deep Koebner phenomenon") at entheses triggers inflammation, explaining dactylitis and the pattern of joint involvement.
8. Uniquely, PsA shows **both erosion and new bone formation** — periostitis, enthesophytes, ankylosis and, at the extreme, **arthritis mutilans**.

C. Clinical Features — Moll and Wright's Five Patterns

9. **(i) Asymmetrical oligoarthritis** (≤4 joints) — classically described as the commonest at presentation.
10. **(ii) Symmetrical polyarthritis** — RA-like but seronegative, with DIP involvement; the commonest pattern in established disease.
11. **(iii) Distal interphalangeal predominant** — strongly associated with **nail disease** of the same digit.
12. **(iv) Spondylitis and sacroiliitis** — often asymmetrical, with inflammatory back pain and chunky non-marginal **paravertebral ossification**.
13. **(v) Arthritis mutilans** — rare, severely destructive, with **telescoping "opera-glass" digits** from osteolysis.
14. **Enthesitis** — Achilles tendon insertion, plantar fascia, lateral epicondyle, patellar tendon and iliac crest; heel pain in an older adult with psoriasis is enthesitis until proved otherwise.
15. **Dactylitis ("sausage digit")** — uniform swelling of the whole digit from combined tenosynovitis, synovitis and soft-tissue oedema; occurs in about a third and marks more severe disease.
16. **Nail disease** in up to 80–90% of those with arthritis: **pitting, onycholysis, subungual hyperkeratosis, oil-drop sign, splinter haemorrhages, ridging** — the single most useful clue when skin disease is inapparent.
17. **Extra-articular:** anterior **uveitis** (often insidious, bilateral and posterior, unlike the acute unilateral uveitis of ankylosing spondylitis), inflammatory bowel disease, and aortic root disease.
18. **Comorbidity burden — important in geriatrics:** a high prevalence of **metabolic syndrome, obesity, type 2 diabetes, hypertension, fatty liver, hyperuricaemia and gout**, and a significantly increased cardiovascular risk; also depression and osteoporosis. These comorbidities dictate drug choice and must be actively screened.

D. Investigations

19. **Seronegativity:** rheumatoid factor and anti-CCP are typically negative — but a low-titre RF may be present in older adults from age alone, and about 5–10% of PsA is anti-CCP positive (marking more erosive disease).
20. **ESR and CRP** may be raised, but are normal in up to half despite active disease; serum urate is often high, which confuses the picture with gout.
21. **Radiographs:** the hallmark is **erosion with adjacent new bone formation** — the **"pencil-in-cup" deformity**, acro-osteolysis, fluffy periostitis, ankylosis, asymmetrical sacroiliitis and non-marginal syndesmophytes. **Absence of periarticular osteopenia** helps distinguish it from RA.
22. **Ultrasound with power Doppler** and **MRI** detect enthesitis, dactylitis and early sacroiliitis before radiographic change.
23. **CASPAR classification criteria** (score ≥3 in the presence of inflammatory articular disease): current psoriasis (2 points), personal or family history of psoriasis (1), psoriatic nail dystrophy (1), negative rheumatoid factor (1), current or past dactylitis (1), and juxta-articular new bone formation on radiograph (1). Specificity approaches 99%.
24. **Screening tools** such as PEST and PASE are used in dermatology clinics to detect PsA early; **MDA (minimal disease activity)** and DAPSA are the treat-to-target measures.
25. **Differential diagnosis in the elderly:** late-onset rheumatoid arthritis, erosive/nodal osteoarthritis of the DIP joints

(Heberden's nodes — no inflammation, no nail change), **gout and CPPD**, reactive arthritis, and PMR.

E. Management (GRAPPA domain-based approach)

26. **General:** patient education, smoking cessation, **weight reduction** (obesity worsens disease and blunts drug response), structured exercise, physiotherapy for enthesitis and axial disease, occupational therapy and assistive devices, plus **aggressive cardiovascular and metabolic risk management**.
27. **Peripheral arthritis:** NSAIDs for mild disease (used sparingly in the elderly); local corticosteroid injection for mono/oligoarthritis; then **csDMARDs — methotrexate** (also treats skin), leflunomide, sulfasalazine (not for skin or axial disease); avoid ciclosporin in older adults.
28. **Inadequate response:** escalate to **biologic or targeted synthetic DMARDs** — **TNF inhibitors** (adalimumab, etanercept, infliximab, golimumab, certolizumab), **IL-17 inhibitors** (secukinumab, ixekizumab, and the IL-17A/F inhibitor **bimekizumab**), **IL-12/23 inhibitor** ustekinumab, **IL-23 inhibitors** (guselkumab, risankizumab), **JAK inhibitors** (tofacitinib, upadacitinib), the TYK2 inhibitor **deucravacitinib**, the PDE-4 inhibitor **apremilast**, and abatacept.
29. **Domain-directed choice (GRAPPA):** for **axial disease**, NSAIDs, physiotherapy and simple analgesia are strongly recommended, as are a JAK inhibitor, TNF inhibitor or IL-17 inhibitor — **csDMARDs do not work for axial disease**. For **enthesitis**, strongly recommended options include a TNF inhibitor, an IL-12/23, IL-17 or IL-23 inhibitor, a JAK inhibitor or a PDE-4 inhibitor. For **dactylitis**, GRAPPA strongly recommends a TNF inhibitor, an IL-12/23, IL-17 or IL-23 inhibitor, a JAK inhibitor or a PDE-4 inhibitor.
30. **Geriatric prescribing considerations:** apremilast is attractive in frail patients for its safety profile but causes diarrhoea and weight loss; **JAK inhibitors are restricted in those over 65** with cardiovascular risk factors or a smoking history (ORAL Surveillance); IL-17 inhibitors are avoided in inflammatory bowel disease and predispose to candidiasis; all biologics require **latent tuberculosis and hepatitis screening**, which is essential in India, plus vaccination and infection vigilance.
31. **Treat-to-target** aiming for minimal disease activity, with therapy tapering considered in sustained remission; surgery (synovectomy, arthroplasty, fusion) for established joint damage.
32. **Prognosis:** around 20% develop severe destructive disease; poor prognostic markers are polyarticular onset, raised ESR/CRP, early radiographic damage, dactylitis and nail disease. Excess mortality is largely **cardiovascular**.

Latest Updates (2023 – Present)

- **Bimekizumab**, a dual **IL-17A and IL-17F** inhibitor, received FDA approval for psoriatic arthritis in **September 2024** on the strength of two phase 3 trials (BE OPTIMAL in biologic-naive and BE COMPLETE in TNF-inadequate responders). Current GRAPPA-based practice includes bimekizumab alongside secukinumab and ixekizumab whenever IL-17 pathway agents are considered.
- **EULAR issued updated psoriatic arthritis management recommendations in 2024** — seven overarching principles and eleven recommendations — reinforcing a **treat-to-target strategy** and **early use of biologic or targeted synthetic DMARDs** (TNF, IL-17, IL-23 and JAK/TYK2 pathway inhibitors) in patients with clinically relevant disease.
- GRAPPA's 2021 domain-based recommendations remain the widely cited framework; at **ACR Convergence 2025** the emphasis shifted from "do we have therapies" to **how best to sequence and select** among them, given disease heterogeneity and a growing population of difficult-to-treat patients.
- **IL-23 inhibitors** (guselkumab, risankizumab) have consolidated their place with sustained efficacy and a favourable safety profile in older adults; oral **deucravacitinib** (TYK2) and the oral IL-23-receptor peptide **icotrokinra** are advancing as convenient oral options.
- **Indian context:** biosimilar TNF inhibitors have transformed access; mandatory latent tuberculosis screening before biologic therapy is critical given high background prevalence, and treatment costs are covered under AB-PMJAY and the Ayushman Vay Vandana Card for those ≥70 years.

Current Practice Update

Issue	Textbook position	Current position (2023–present)
Framework	Moll and Wright's five clinical patterns	**Domain-based** (peripheral arthritis, axial, enthesitis, dactylitis, skin, nails) as per GRAPPA and EULAR 2024
Methotrexate	First-line DMARD for all PsA	Retained for peripheral disease and skin, but **ineffective for axial disease, enthesitis and dactylitis** — escalate early
IL-17 blockade	Secukinumab, ixekizumab	Add **bimekizumab** (dual IL-17A/F, FDA-approved September 2024)
JAK inhibitors	Effective oral option	**Age- and cardiovascular-risk restricted** in the over-65s
Comorbidity	Noted	Cardiometabolic screening and weight management are now **part of the treatment plan**

Exam One-Liners

- PsA is an enthesitis-driven seronegative spondyloarthritis of the IL-23/Th17 axis.
- Five Moll and Wright patterns; DIP involvement and nail disease travel together.
- Dactylitis and nail pitting are the two bedside giveaways.
- "Pencil-in-cup" erosion with new bone formation, and no periarticular osteopenia.
- CASPAR criteria: score ≥3, specificity about 99%.
- csDMARDs do not work for axial disease, enthesitis or dactylitis.
- Bimekizumab (2024) is the newest IL-17A/F option; EULAR 2024 pushes early advanced therapy.
- Screen every patient for metabolic syndrome and cardiovascular risk.

Final Night Grid — One Line Per Topic

Topic	The one sentence to write first
Osteoporosis	Low bone mass with microarchitectural deterioration; T-score ≤−2.5, or any fragility fracture regardless of T-score.
Post-hip-fracture	Treat before discharge — imminent re-fracture risk is highest in the first 2 years; IV zoledronate plus a Fracture Liaison Service.
DEXA	Reference standard for BMD; diagnose on the lowest valid site, monitor every 1–3 years, beware degenerative spine in the elderly.
FRAX	10-year probability of hip and major osteoporotic fracture from 11 clinical factors ± femoral neck BMD; blind to falls and fracture recency.
SERMs	Bone agonist, breast antagonist; vertebral fracture protection only; contraindicated after femoral neck fracture.
Atypical femur fracture	Transverse subtrochanteric/diaphyseal fracture with lateral cortical beaking after prolonged turnover suppression; often bilateral.
Renal osteodystrophy	Bone histology of CKD classified by Turnover, Mineralisation and Volume; adynamic bone disease is now commonest.
Osteoarthritis	Exercise and weight loss are the only disease-modifying measures; topical before oral NSAIDs; joint replacement for refractory severe disease.
Polymyalgia rheumatica	Girdle pain and stiffness over 50 with high ESR, normal CK, and dramatic response to prednisolone 15 mg.
Temporal arteritis	Granulomatous large-vessel vasculitis — a sight-threatening emergency; steroids first, biopsy after.
Late-onset RA	Onset ≥60, equal sex ratio, abrupt, large proximal joints, often seronegative with very high ESR.
Sjögren's syndrome	Autoimmune epithelitis causing sicca; exclude anticholinergic drugs first; lifelong lymphoma vigilance.
Inflammatory myopathies	DM (complement microangiopathy), PM (CD8 cytotoxicity), IBM (rimmed vacuoles, steroid-unresponsive, commonest over 50).
Psoriatic arthropathy	Enthesitis-driven seronegative spondyloarthritis with dactylitis, nail disease and pencil-in-cup erosions.

Compiled from Pathy's Principles and Practice of Geriatric Medicine (6th edition) and Kane's Essentials of Clinical Geriatrics, with clearly separated evidence updates from guidelines, regulatory approvals and randomised trials published 2023 to date.

CARDIOVASCULAR SYSTEM & THROMBOEMBOLISM

High-Yield Examination Answers
MD Geriatric Medicine

18 Structured Questions · 15-Mark & 7-Mark Formats

Hypertension · Heart Failure · Cardiomyopathy · Arrhythmia

Endocarditis · Statins · Anticoagulation · VTE

TEXTBOOK-ANCHORED | UPDATED TO AUGUST 2026

Primary sources: *Pathy's Principles and Practice of Geriatric Medicine*, 6th edition
Essentials of Clinical Geriatrics, 7th edition
Supplemented with current guidelines, trial data and Indian clinical context

Contents

1	Management of hypertension in the elderly	15 marks
2	Isolated systolic hypertension and its management **(recurs)**	15 marks
3	Congestive cardiac failure — aetiology, features, evaluation, management, drugs, refractory failure	15 marks
4	Congestive heart failure — diagnostic evaluation	7 marks
5	Refractory cardiac failure	7 marks
6	Diastolic dysfunction	7 marks
7	Cardiomyopathies — pathophysiology, aetiology, features, diagnosis, treatment	15 marks
8	Cardiomyopathy (short note)	7 marks
9	Dilated cardiomyopathy	7 marks
10	Atrial fibrillation and its management	15 marks
11	Sick sinus syndrome	7 marks
12	Cardiac pacemaker	7 marks
13	Infective endocarditis	15 marks
14	Use of statins in the elderly	7 marks
15	Calcium channel antagonists	7 marks
16	Anticoagulants in geriatric practice — indications and agents	15 marks
17	Venous thromboembolism and post-operative management of anticoagulation	15 marks
18	Causes, treatment and prevention of deep vein thrombosis	15 marks
—	Appendix: Rapid Revision Grid (night-before recall)	—

How to use this document. *Each answer follows a fixed architecture: numbered high-yield points under thematic subheadings → "Latest Updates (2023–Present)" with inline citations → "Current Practice Update" table contrasting the textbook position with current evidence → an orange "Examiner's Tip" panel with mark-earning lines. Recurring topics carry an additional green "Extra Points to Remember" panel. Where the uploaded textbooks do not cover a topic, this is stated explicitly in a red panel.*

Q1. Management of hypertension in the elderly

15-MARK ANSWER | 18 POINTS

Definition and Epidemiology

1. **Hypertension** in older people is defined by the same thresholds as in the young, but the pattern differs: **systolic BP rises** with age while **diastolic BP plateaus or falls** after about 60 years.
2. Prevalence rises steeply with age. In India, **LASI Wave 1** found hypertension in about **46%** of adults aged 45 and above, with only about **32%** achieving control — awareness and control are the weak links, not availability of drugs.
3. **Ageing changes that cause hypertension:** arterial wall stiffness from collagen deposition and elastin loss, reduced nitric oxide bioavailability, impaired baroreceptor sensitivity, and reduced plasma renin activity.

Special Problems of Measurement in Old Age

4. Always measure **standing (orthostatic) BP** as well as sitting, because postural hypotension is common and treatment can worsen it. Pathy's advises orthostatic measurement at every visit.
5. Three pitfalls must be excluded before labelling a patient hypertensive: **white-coat hypertension** (common in the elderly), **masked hypertension**, and **pseudo-hypertension** (falsely high cuff readings from incompressible calcified arteries).
6. Use a large cuff for large arms, take the average of at least two readings on two occasions, and confirm with **home or ambulatory BP monitoring** where possible.

Evidence for Treating Older Patients

7. Treatment reduces stroke, heart failure and mortality at every age. **HYVET** (patients over 80, SBP ≥160 mmHg) showed a **30%** fall in stroke, **21%** fall in all-cause death and **64%** fall in heart failure.
8. **SPRINT-Senior** (age ≥75) showed intensive control (SBP <120) reduced cardiovascular events and death compared with a target of <140.
9. Cerebrovascular disease is an **indication for**, not a contraindication to, antihypertensive therapy.
10. Counter-evidence exists in the very frail: SBP below **120 mmHg** is associated with higher mortality in those over 85, and the **PARTAGE** study showed excess mortality in nursing-home residents on multiple antihypertensives with low achieved BP.

Non-Pharmacological Management

1. Salt restriction, a fruit-vegetable-low-fat-dairy (DASH-type) diet, potassium-, calcium- and magnesium-rich foods, weight optimisation, aerobic exercise, smoking cessation and alcohol limitation.
2. **Review and rationalise drugs that raise BP:** NSAIDs, decongestants and glucocorticoids. Regular medication review reduces polypharmacy and improves adherence.

Drug Therapy — Principles and Choice

3. Golden rule: **"start low, go slow"**, use once-daily long-acting agents, and monitor BP, creatinine and potassium after each dose change.
4. **Thiazide/thiazide-like diuretics** are first-line, especially for isolated systolic hypertension. **Chlorthalidone 12.5–25 mg** is preferred over hydrochlorothiazide. Watch hypokalaemia, hyponatraemia, hyperuricaemia and hyperglycaemia; avoid in gout.
5. **Calcium channel blockers** are superior for stroke prevention; **diuretics** are superior for heart failure prevention; **beta blockers are inferior** for preventing major cardiovascular events and should be reserved for compelling indications (ischaemic heart disease, heart failure, atrial fibrillation).
6. **ACE inhibitors / ARBs** are preferred in heart failure and in diabetes with albuminuria. **Never combine an ACE inhibitor with an ARB** in older people — the risk of acute kidney injury and hyperkalaemia rises sharply.

Individualisation and Targets

7. Pathy's key message: **lower BP in proportion to the patient's functional level.** Indiscriminate application of a single tight target to frail, multimorbid elders "may do more harm than good" — risks are falls, cognitive decline, acute kidney injury and hyperkalaemia.
8. Practical targets: aim for <130/80 mmHg in the **robust, independent** elder; accept <140/90 mmHg in the **frail or care-home resident**; consider deprescribing at the end of life. Assess frailty (e.g. Clinical Frailty Scale) before setting the target.

Latest Updates (2023–Present)

- **2025 AHA/ACC/multisociety High Blood Pressure Guideline** (JACC/Circulation, Aug 2025) replaces the 2017 guideline. It retains a treatment target of **<130/80 mmHg** for most adults, but now **encourages achieving SBP <120**

mmHg, based on SPRINT, STEP, ESPRIT and BPROAD. It explicitly permits **individualised, less stringent targets** for institutionalised patients, those with limited life expectancy and frail older adults.

- The 2025 guideline replaces the Pooled Cohort Equations with the **PREVENT risk equations** for treatment decisions, makes out-of-office measurement (ABPM/HBPM) the diagnostic standard, strongly endorses **single-pill combinations**, and adds a Class 2b recommendation for **renal denervation** in uncontrolled hypertension.
- **2024 ESC Hypertension Guideline** recommends a systolic target of **120–129 mmHg** in most treated adults, with explicit relaxation for symptomatic orthostatic hypotension, age ≥85 or moderate-to-severe frailty.
- **STEP trial** (Chinese patients aged 60–80) showed intensive control (110–130 mmHg) reduced cardiovascular events versus 130–150 mmHg — the first dedicated intensive-control trial in an exclusively older Asian population.
- **India:** the **India Hypertension Control Initiative (IHCI)** — ICMR, MoHFW and WHO-India — has scaled up simple standardised treatment protocols, single-pill combinations and free drug supply through Ayushman Arogya Mandirs; BP control in enrolled cohorts rose from about 26% at registration to about 60% at follow-up.

Current Practice Update

Textbook position (Pathy's 6e / Essentials 7e)	Current evidence (2023–2026)
Treatment goal <140/90 mmHg for uncomplicated hypertension; concern that outcomes are not improved below 130/85.	2025 ACC/AHA target <130/80 mmHg with encouragement toward <120 systolic; 2024 ESC target 120–129 systolic.
Risk estimated by Framingham-type or Pooled Cohort scores.	**PREVENT equations** now recommended, incorporating kidney and metabolic variables.
Stepped monotherapy with dose titration is standard.	**Single-pill combination as initial therapy** is now emphasised by both ACC/AHA and ESC to improve adherence.
Clinic BP is the basis of diagnosis; ABPM mentioned as adjunct.	Out-of-office measurement (HBPM/ABPM) is the diagnostic gold standard.
Frailty-based targets described as "awaited" research.	Guidelines now formally endorse individualised, relaxed targets in frailty and limited life expectancy; deprescribing trials (e.g. OPTIMISE) support careful de-intensification.

Examiner's Tip

Write these lines verbatim: "*Blood pressure must be measured both sitting and standing at every visit, and pseudo-hypertension excluded before labelling an older patient hypertensive.*" · "*HYVET established that treating patients over 80 with SBP ≥160 mmHg reduces stroke by 30% and heart failure by 64%.*" · "*Beta blockers are not first-line for uncomplicated hypertension in the elderly; thiazide-like diuretics and calcium channel blockers are.*" · "*The degree of BP lowering should be proportionate to the patient's functional level — a robust 80-year-old and a frail care-home resident do not share a target.*"

Q2. Isolated systolic hypertension and its management

15-MARK ANSWER | 17 POINTS | RECURRING TOPIC

Definition

1. **Isolated systolic hypertension (ISH)** = systolic BP ≥140 mmHg with diastolic BP <90 mmHg. Widened **pulse pressure** is the hallmark.
2. It is the **dominant pattern of hypertension in old age.** NHANES III showed that fewer than 20% of hypertensive people over 60 have a raised diastolic pressure, and this proportion falls further with advancing age.

Pathophysiology — the core examination point

3. ISH is caused by **large-artery stiffness**, not by raised peripheral resistance (which is the mechanism in the young).
4. Mechanism: fragmentation of **elastin**, increased **collagen** cross-linking, calcification of the media, and endothelial dysfunction with reduced nitric oxide → loss of aortic Windkessel function.
5. Consequence: the aorta cannot buffer stroke volume, so systolic pressure rises; the pulse wave travels faster and the **reflected wave returns in systole instead of diastole**, further augmenting systolic pressure and **lowering diastolic pressure** — hence the wide pulse pressure and reduced coronary perfusion pressure.
6. Pathy's highlights the therapeutic paradox: **current antihypertensives act mainly by reducing peripheral vascular resistance and have little effect on arterial stiffness**, which is why ISH is difficult to control and why drugs targeting stiffness are an unmet need.

Clinical Significance

7. ISH is a major independent risk factor for **stroke, coronary events, heart failure, atrial fibrillation, chronic kidney disease progression and cognitive decline.**
8. Wide pulse pressure predicts risk better than systolic pressure alone in the very old.
9. Low diastolic pressure (<60–65 mmHg) during treatment is a warning sign — coronary filling occurs in diastole, so excessive diastolic fall may precipitate myocardial ischaemia (the "J-curve").

Evaluation

0. Confirm with repeated and out-of-office readings; exclude white-coat effect and pseudo-hypertension; check orthostatic drop (postural hypotension was present in a substantial proportion of the SHEP cohort at baseline).
1. Assess target-organ damage: ECG/echocardiography for LV hypertrophy, urine albumin–creatinine ratio, creatinine/eGFR, fundoscopy, lipids and glucose.
2. Exclude secondary causes relevant to old age listed in *Essentials*: **atherosclerotic renovascular disease, primary hyperaldosteronism, hyperparathyroidism, chronic kidney disease and oestrogen therapy.**

Management

3. **Non-pharmacological:** sodium restriction, DASH-type diet, potassium-rich foods, aerobic exercise, weight optimisation, smoking cessation.
4. **First-line drugs: thiazide-like diuretics** (chlorthalidone/indapamide) and **dihydropyridine calcium channel blockers** (amlodipine). These are the classes proven in the landmark ISH trials.
5. **Landmark trials to quote: SHEP** (chlorthalidone-based; 36% stroke reduction), **Syst-Eur** (nitrendipine-based; 42% stroke reduction), **HYVET** (indapamide ± perindopril in the over-80s), and the **LIFE** ISH substudy (losartan superior to atenolol in ISH with LV hypertrophy).
6. Add an ACE inhibitor or ARB as second agent; use a low-dose two-drug combination rather than maximising a single drug. Avoid beta blockers unless there is a compelling indication.
7. **Titrate to the systolic target while watching the diastolic pressure and the standing pressure.** Reduce or stop treatment if the patient develops symptomatic postural hypotension, falls, or a diastolic pressure below about 60 mmHg.

Latest Updates (2023–Present)

- **2025 AHA/ACC guideline** makes no separate ISH target: the systolic goal of <130 mmHg (with encouragement toward <120) applies, but individualisation is explicitly allowed for frailty, orthostatic hypotension and limited life expectancy.
- **ESPRIT trial** (Lancet 2024) — intensive systolic lowering to <120 mmHg reduced major vascular events versus <140 mmHg in high-risk adults, strengthening the case for tighter systolic control in fit older patients.
- **STEP trial** (NEJM 2021, widely cited in 2023–2025 guidance) — in adults aged 60–80, a target of 110–130 mmHg reduced cardiovascular events; a substantial proportion of participants had a predominantly systolic pattern.
- **2024 ESC guideline** specifically advises that in patients aged ≥85, with orthostatic hypotension, or with moderate-to-severe frailty, the systolic target may be relaxed — the most explicit statement so far that ISH management is frailty-dependent.
- Interest continues in **anti-stiffness strategies** (SGLT2 inhibitors, mineralocorticoid receptor antagonists, advanced

glycation end-product cross-link breakers) and in **renal denervation** (Class 2b, 2025 ACC/AHA) for resistant systolic hypertension, but no drug is yet licensed specifically to reduce arterial stiffness.

Current Practice Update

Textbook position	Current evidence (2023–2026)
Systolic goal <140 mmHg; caution that benefit may not extend below 130 mmHg.	Systolic goal <130 mmHg (ACC/AHA 2025) or 120–129 mmHg (ESC 2024) in patients who tolerate it.
No drug targets arterial stiffness; new agents "required".	Still true — this remains an unmet need; renal denervation is the only new device-based option (Class 2b).
Frailty-adjusted targets described as unresolved.	Frailty-adjusted relaxation of targets is now written into ESC 2024 and ACC/AHA 2025.
Stepped care with single agents.	Low-dose single-pill combination therapy is preferred first-line for most.

Extra Points to Remember (recurring topic)

- **Pulse pressure = SBP − DBP.** A pulse pressure above 60–65 mmHg indicates significant arterial stiffening and independently predicts cardiovascular events in the elderly.
- **Pulse wave velocity** (carotid–femoral) is the reference measure of arterial stiffness; >10 m/s indicates target-organ damage.
- **Osler's manoeuvre** — a palpable, pulseless radial artery when the cuff is inflated above systolic pressure — is the classic bedside clue to pseudo-hypertension.
- **CAFE study** (Circulation 2006, cited in Pathy's): drugs differ in their effect on *central* aortic pressure even when brachial pressures are equal — amlodipine-based therapy lowered central pressure more than atenolol-based therapy.
- Mnemonic for first-line drugs in ISH: **"CD" — Calcium channel blocker and Diuretic** (thiazide-like).
- Remember the three ISH trial names in order of era: **SHEP → Syst-Eur → HYVET → (now) STEP and ESPRIT.**

Examiner's Tip

"*Isolated systolic hypertension is a disease of large-artery stiffness, not of increased peripheral resistance.*" · "*Loss of aortic elasticity causes early return of the reflected pulse wave in systole, raising systolic and lowering diastolic pressure, which widens the pulse pressure.*" · "*Thiazide-like diuretics and dihydropyridine calcium channel blockers are first-line, supported by SHEP, Syst-Eur and HYVET.*" · "*Treatment must be titrated against the standing blood pressure, and a diastolic pressure below 60 mmHg should prompt caution because coronary perfusion occurs in diastole.*"

Q3. Congestive cardiac failure — aetiology, clinical features, evaluation and management; drug categories, indications and side effects; approach to refractory heart failure

15-MARK ANSWER | 20 POINTS

Definition and Burden

1. **Heart failure (HF)** is a clinical syndrome of symptoms and signs caused by a structural or functional cardiac abnormality resulting in reduced cardiac output or raised filling pressures.
2. Prevalence rises with age — HF is present in **13–14%** of people over 80. Lifetime risk at age 40 is about 21% for men and 20% for women. Two-year and five-year mortality are approximately **60%** and **75%**, comparable to many common cancers.
3. **Indian and developing-country context:** ischaemic aetiology is proportionally less common; rheumatic heart disease, hypertension, idiopathic dilated cardiomyopathy and cor pulmonale contribute more. Patients present younger and at a more advanced stage.

Aetiology

4. **Commonest causes in the elderly:** ischaemic heart disease and hypertension — together they account for more than **75%** of overt heart failure in older patients.
5. Others: valvular disease (calcific aortic stenosis, mitral annular calcification), atrial fibrillation, cardiomyopathies (including cardiac amyloidosis), thyroid disease, anaemia, chronic kidney disease, alcohol, chemotherapy (anthracyclines, trastuzumab) and cor pulmonale.
6. **Ageing changes that predispose:** myocardial fibrosis and collagen deposition, reduced beta-adrenergic responsiveness, impaired ventricular relaxation, reduced maximum heart rate, and increased afterload from arterial stiffening.

Clinical Features

7. **Typical:** exertional dyspnoea, orthopnoea, paroxysmal nocturnal dyspnoea, fatigue, ankle oedema, raised JVP, third heart sound, basal crackles, hepatomegaly, pleural effusion, ascites.
8. **Atypical presentations in old age (high-yield):** confusion or delirium, falls, anorexia and weight loss, nocturia, functional decline, unexplained fatigue. Pathy's notes that **one-third of older patients with HF do not display the classical signs**.
9. Individual symptoms and signs have **low sensitivity**; it is the constellation that supports the diagnosis. *Essentials* warns that HF is **over-diagnosed** in older adults — pedal oedema alone is not sufficient, as venous stasis mimics it.

Evaluation

10. **Step 1 — clinical probability:** symptoms, signs, prior cardiac history, abnormal ECG. A completely normal ECG makes HF very unlikely.
11. **Step 2 — natriuretic peptides.** ESC thresholds for further investigation: **BNP >35 pg/mL** or **NT-proBNP >125 pg/mL** in the non-acute setting. Normal levels virtually exclude HF; raised levels also carry prognostic weight. Equally predictive in HFrEF and HFpEF.
12. **Step 3 — echocardiography** to define ejection fraction, chamber size, wall thickness, valve disease and diastolic function. NICE: echocardiography first in those with previous MI; natriuretic peptide first in those without.
13. Supportive tests: chest X-ray (excludes COPD, fibrosis, malignancy), full blood count, renal function and electrolytes, thyroid function, glucose/HbA1c, iron studies, liver function, urinalysis.

Non-Pharmacological Management

14. Patient and carer education, daily weight monitoring, salt and fluid moderation, alcohol and smoking cessation, treatment of anaemia and iron deficiency, immunisation (influenza and pneumococcal), **supervised cardiac rehabilitation and exercise training**, adherence support, and multidisciplinary HF nurse follow-up. Pathy's stresses these are "often overlooked".

Drug Categories — Indications and Side Effects (HFrEF)

Class	Indication / evidence	Key adverse effects in the elderly
ACE inhibitors (enalapril, ramipril)	All HFrEF irrespective of NYHA class; cornerstone since CONSENSUS. Low doses still give benefit.	Hypotension, hyperkalaemia, rising creatinine, cough, angioedema. Contraindicated in bilateral renal artery stenosis.
ARBs (candesartan, valsartan)	ACE-inhibitor intolerance (cough).	Hyperkalaemia, hypotension. **Never combine with ACE inhibitor.**

Beta blockers (carvedilol, bisoprolol, nebivolol, metoprolol MR)	Symptomatic HFrEF NYHA I–IV added to ACE inhibitor; benefit shown up to age 80 (SENIORS used nebivolol in the elderly).	Fatigue, bradycardia, worsening exercise tolerance, bronchospasm in severe COPD. Start low, go slow.
MRAs (spironolactone, eplerenone)	Persisting symptoms despite ACE inhibitor + beta blocker.	Hyperkalaemia, renal impairment, gynaecomastia (spironolactone). Monitor K^+ and creatinine.
Loop diuretics (furosemide, torsemide)	Congestion and fluid overload — symptomatic benefit, no mortality benefit.	Dehydration, hypokalaemia, hyponatraemia, gout, ototoxicity, incontinence, falls.
Digoxin	Persisting symptoms, especially with AF. In over-70s use **0.125 mg/day**, target level 0.5–0.9 ng/mL.	Nausea, anorexia, heart block, arrhythmia, confusion. Toxicity increased by renal impairment and hypokalaemia.
Ivabradine	NYHA II–III, sinus rhythm, heart rate >70/min despite optimal therapy (SHIFT).	Headache, dizziness, phosphenes (visual brightness), bradyarrhythmia.
ARNI (sacubitril/valsartan)	NYHA II–III already on optimal therapy (PARADIGM-HF; 20% reduction in CV death/HF admission).	Hypotension, hyperkalaemia, angioedema. **36-hour washout** required after the last ACE inhibitor dose.
Hydralazine + nitrate	ACE inhibitor/ARB intolerance; African-ancestry patients.	Headache, tachycardia, lupus-like syndrome.

Device and Advanced Therapy

5. **Cardiac resynchronisation therapy (CRT)** for HFrEF with ventricular conduction delay; **ICD** for selected patients with reduced ejection fraction and reasonable life expectancy; consider left ventricular assist devices and transplantation only in highly selected younger-biological-age patients.

HFpEF and the Older Patient

6. Pathy's Table 34.8 summarises the historically negative HFpEF trials: **CHARM-Preserved, I-PRESERVE, PEP-CHF, SENIORS, DIG-ancillary, TOPCAT and PARAGON-HF** — none reduced mortality; several reduced hospitalisations. Management therefore centred on diuretics plus treatment of hypertension, ischaemia, AF and diabetes.

Approach to Refractory Heart Failure

7. **Confirm it is truly refractory:** check adherence, excess salt/fluid intake, and drugs that worsen HF (NSAIDs, glitazones, non-dihydropyridine calcium channel blockers, some antiarrhythmics).
8. **Search for a reversible precipitant:** uncontrolled AF or other arrhythmia, silent ischaemia, anaemia, iron deficiency, infection, thyroid dysfunction, renal failure, uncontrolled hypertension, severe valve disease, pulmonary embolism, cardiac amyloidosis.
9. **Escalate therapy:** optimise/uptitrate disease-modifying drugs; combine loop diuretic with a thiazide for **sequential nephron blockade**; switch oral to intravenous furosemide (infusion) for gut oedema; consider ultrafiltration; consider CRT; consider inotropes for short-term bridging only.
10. **Palliative approach:** as the disease reaches the terminal stage, prioritise symptom relief and quality of life — withdraw unnecessary drugs and blood tests, treat breathlessness and anxiety with judicious **opioids and benzodiazepines**, provide multidisciplinary emotional support, and consider home- or institution-based hospice care.

Latest Updates (2023–Present)

- **SGLT2 inhibitors are now foundational across the entire ejection fraction spectrum** — the 2023 ESC Focused Update extended dapagliflozin and empagliflozin from HFrEF to HFmrEF and HFpEF (DELIVER, EMPEROR-Preserved). Four-pillar therapy for HFrEF is now **ARNI/ACEi + beta blocker + MRA + SGLT2 inhibitor**, started rapidly and in parallel rather than sequentially (STRONG-HF).
- **FINEARTS-HF** (NEJM 2024): finerenone, a non-steroidal MRA, reduced cardiovascular death and total worsening HF events by **16%** in HF with LVEF ≥40%, with benefit consistent even at EF ≥60% — the first robust evidence that MRA blockade works in HFpEF.
- **2026 ACC Expert Consensus Decision Pathway on HFpEF** (JACC) replaces the 2023 version: SGLT2 inhibitor for all, plus a non-steroidal MRA with **finerenone as the MRA of choice** (spironolactone a reasonable alternative if cost or tolerance is prohibitive), and incretin-based therapy for BMI ≥30 kg/m². It recommends sequential use of the **H2FPEF** and **HFA-PEFF** scores and systematic exclusion of cardiac and non-cardiac mimics.
- **STEP-HFpEF** (semaglutide) improved KCCQ symptom score and exercise capacity in obesity-related HFpEF; **SUMMIT** (tirzepatide) additionally reduced cardiovascular death or worsening HF events.
- **Cardiac amyloidosis is now a treatable cause of HFpEF in the elderly** — see Q7.

- **Iron deficiency:** intravenous ferric carboxymaltose/derisomaltose reduces HF hospitalisation (AFFIRM-AHF, IRONMAN); iron studies are now part of routine HF work-up.

Current Practice Update

Textbook position	Current evidence (2023–2026)
HFrEF therapy = ACE inhibitor/ARB + beta blocker + MRA, with ivabradine and ARNI as add-ons; SGLT2 inhibitors described as a "future perspective".	Four-pillar therapy with SGLT2 inhibitors as an established first-line pillar, initiated early and simultaneously.
"Clinical studies in HFpEF so far have not shown benefits."	SGLT2 inhibitors (DELIVER, EMPEROR-Preserved) and finerenone (FINEARTS-HF) now show benefit; GLP-1 agents improve symptoms in obesity-related HFpEF.
Uptitration performed slowly over months.	STRONG-HF supports rapid, protocol-driven uptitration with close follow-up after decompensation.
Iron deficiency not emphasised.	Routine iron studies; IV iron reduces HF hospitalisation.

Examiner's Tip

"*More than 75% of heart failure in older patients is attributable to hypertension or coronary heart disease.*" · "*One-third of older patients with heart failure do not display the classical signs; presentation may be confusion, falls or functional decline.*" · "*BNP >35 pg/mL or NT-proBNP >125 pg/mL should prompt echocardiography; normal values make heart failure unlikely.*" · "*Contemporary HFrEF therapy rests on four pillars — ARNI or ACE inhibitor, beta blocker, mineralocorticoid receptor antagonist and SGLT2 inhibitor.*" · "*In refractory failure, first exclude non-adherence and drugs that worsen heart failure before escalating therapy.*"

Q4. Congestive heart failure — diagnostic evaluation

7-MARK ANSWER | 10 POINTS

1. **Why it is difficult in the elderly:** atypical presentation, multiple comorbidities that mimic HF (COPD, obesity, anaemia, venous stasis, renal disease), and cognitive impairment limiting history-taking. *Essentials* stresses that HF is **over-diagnosed** — pedal oedema alone is not enough.
2. **Step 1 — assess clinical probability** using the ESC algorithm: symptoms, signs, prior cardiac conditions and an abnormal ECG. Presence of one or more should prompt natriuretic peptide measurement.
3. **Step 2 — natriuretic peptides.** **BNP >35 pg/mL** or **NT-proBNP >125 pg/mL** (non-acute setting) warrant echocardiography. In acute breathlessness, BNP above about **300 pg/mL** favours a cardiac over a pulmonary cause. Normal values effectively rule out HF; levels also predict prognosis.
4. **Causes of falsely raised natriuretic peptides:** age, female sex, renal impairment, atrial fibrillation, pulmonary embolism, sepsis, anaemia. **Falsely low:** obesity, and treatment with ARNI alters BNP interpretation (use NT-proBNP).
5. **Step 3 — echocardiography** is the key confirmatory test: ejection fraction, chamber dimensions, wall thickness, diastolic parameters (E/e′, left atrial volume index, tricuspid regurgitant velocity), valvular lesions and pericardial disease. NICE: echocardiography first if previous MI; natriuretic peptide first if no previous MI.
6. **ECG:** looks for prior infarction, LV hypertrophy, atrial fibrillation, bundle branch block (relevant to CRT) and low voltage with LV hypertrophy (suggesting amyloidosis). A normal ECG makes HF very unlikely.
7. **Chest radiograph:** cardiomegaly, upper-lobe diversion, Kerley B lines, effusions; also excludes COPD, fibrosis and malignancy.
8. **Laboratory panel:** full blood count, urea, creatinine and electrolytes, eGFR, liver function, thyroid function, glucose/HbA1c, lipids, iron studies (ferritin and transferrin saturation), urinalysis.
9. **Classify by ejection fraction:** HFrEF (≤40%), HFmrEF (41–49%), HFpEF (≥50%) — HFpEF predominates in older women with hypertension.
10. **Assess functional and geriatric domains alongside cardiac ones:** NYHA class, six-minute walk, frailty, cognition, nutrition, polypharmacy and social support — these determine what treatment is feasible.

Latest Updates and Current Practice Update (concise)

- **Scoring systems for HFpEF are now standard.** The 2026 ACC HFpEF consensus pathway recommends sequential use of the **H2FPEF** and **HFA-PEFF** scores, with diastolic stress echocardiography or invasive exercise haemodynamics if the diagnosis remains uncertain — an approach absent from the textbooks.
- **Systematic search for mimics is now mandatory**, in particular **cardiac amyloidosis** (bone scintigraphy with ^{99m}Tc-PYP/DPD plus serum free light chains and immunofixation), constrictive pericarditis, hypertrophic cardiomyopathy and high-output states. Cardiac MRI with T1 mapping and extracellular volume quantification is increasingly used.
- **Practical change:** lung ultrasound (B-lines) and point-of-care echocardiography are now widely used at the bedside to distinguish cardiac from pulmonary dyspnoea, supplementing rather than replacing the natriuretic peptide–echocardiography pathway described in the textbooks.

> **Examiner's Tip**
>
> Draw the ESC algorithm as a flow diagram — *clinical probability → natriuretic peptide → echocardiography* — and label the cut-offs. Then add one line: "*In an older patient with HFpEF, unexplained left ventricular thickening, low ECG voltages and a history of bilateral carpal tunnel syndrome should prompt bone scintigraphy for transthyretin cardiac amyloidosis.*"

Q5. Refractory cardiac failure

7-MARK ANSWER | 10 POINTS

1. **Definition:** persistent symptoms of heart failure at rest or on minimal exertion (NYHA class IV) with recurrent congestion or low output, despite maximal tolerated guideline-directed therapy — corresponds to **ACC/AHA Stage D, advanced heart failure**.
2. **Recognition ("I NEED HELP" markers):** repeated hospitalisations, intravenous inotrope dependence, escalating diuretic requirement, intolerance of neurohormonal drugs because of hypotension, rising urea and creatinine, hyponatraemia, persistently low ejection fraction and cardiac cachexia.
3. **Step 1 — exclude pseudo-refractoriness:** poor adherence (common with polypharmacy and cognitive impairment), excess dietary salt and fluid, and inadequate drug doses.
4. **Step 2 — stop aggravating drugs:** NSAIDs, corticosteroids, glitazones, verapamil/diltiazem, class I antiarrhythmics, and drugs causing sodium retention.
5. **Step 3 — treat reversible precipitants:** uncontrolled atrial fibrillation or other arrhythmia, silent myocardial ischaemia, severe valve disease, anaemia and iron deficiency, thyroid dysfunction, infection, pulmonary embolism, renal failure, uncontrolled hypertension, sleep apnoea, and **previously undiagnosed cardiac amyloidosis**.
6. **Step 4 — overcome diuretic resistance:** use intravenous furosemide (bolus or infusion) because gut wall oedema impairs oral absorption; add a thiazide such as metolazone for **sequential nephron blockade**; add an MRA; consider acetazolamide (ADVOR) or an SGLT2 inhibitor; correct hypoalbuminaemia; monitor electrolytes and renal function closely.
7. **Step 5 — mechanical and device options:** ultrafiltration for genuinely diuretic-resistant congestion; cardiac resynchronisation therapy in ventricular dyssynchrony; revascularisation or valve intervention (including TAVI in older patients with aortic stenosis) where appropriate.
8. **Step 6 — advanced therapies:** intravenous inotropes (dobutamine, milrinone, levosimendan) are for short-term bridging or palliation only, as continuous inotropes increase mortality. Left ventricular assist devices and transplantation apply to a very small, carefully selected group and are usually limited by biological age and comorbidity.
9. **Step 7 — palliative and end-of-life care** (the geriatric emphasis in Pathy's): prioritise quality of life; withhold unnecessary blood tests and medications; treat breathlessness, agitation and anxiety with judicious opioids and benzodiazepines; discuss advance care planning and deactivation of ICD shocks; involve the multidisciplinary team; consider hospice care in the final days.
10. **Prognosis:** advanced heart failure carries a one-year mortality commonly exceeding 50%; discussions about ceilings of care should occur early rather than in the last admission.

Latest Updates and Current Practice Update (concise)

- **ADVOR trial** (NEJM 2022, now in practice): adding intravenous **acetazolamide** to loop diuretics improves decongestion in acute decompensated HF — a practical addition not present in the textbooks.
- **SGLT2 inhibitors improve diuretic efficiency and are continued (not stopped) during decompensation**; EMPULSE showed clinical benefit when empagliflozin was started in hospital. This reverses the older instinct to withhold new agents during decompensation.
- **Vericiguat** (VICTORIA) is licensed for worsening HFrEF after a recent decompensation, and **omecamtiv mecarbil** remains investigational; both extend the options for the patient who has exhausted the four pillars.

> **Examiner's Tip**
>
> Structure the answer as a numbered algorithm: *confirm → exclude non-adherence → stop culprit drugs → treat precipitants → break diuretic resistance → devices → advanced therapy → palliation.* Finish with: "*In the frail older patient, escalation to advanced therapies is usually inappropriate, and the correct next step is a structured conversation about goals of care and symptom-directed palliation.*"

Q6. Diastolic dysfunction

7-MARK ANSWER | 10 POINTS

1. **Definition:** impaired ventricular relaxation and/or increased chamber stiffness causing raised filling pressures at normal or near-normal ejection fraction. When symptomatic it manifests as **heart failure with preserved ejection fraction (HFpEF)**.
2. **Geriatric importance:** *Essentials* states plainly that **diastolic, not systolic, dysfunction is the primary cause of heart failure in older patients**, and that it is associated with a marked increase in all-cause mortality (Redfield 2003; Bursi 2006).
3. **Pathophysiology:** age-related myocardial fibrosis and collagen deposition, LV hypertrophy from chronic hypertension and arterial stiffening, impaired active relaxation (delayed calcium re-uptake by SERCA), titin stiffening, subendocardial ischaemia and microvascular inflammation.
4. **Consequences:** small increases in volume produce large increases in filling pressure → pulmonary congestion, especially with tachycardia (loss of diastolic filling time) or loss of atrial systole in atrial fibrillation. This explains the classic presentation of **flash pulmonary oedema** with new AF or a hypertensive surge.
5. **Causes:** hypertension with LV hypertrophy (commonest), coronary disease, diabetes, obesity, aortic stenosis, hypertrophic cardiomyopathy, restrictive/infiltrative disease (amyloidosis, haemochromatosis), and constrictive pericarditis as a mimic.
6. **Clinical features:** exertional dyspnoea, fatigue, orthopnoea and congestion indistinguishable from systolic failure at the bedside; a fourth heart sound (S4) and a sustained apex are suggestive; typically an older woman with hypertension, obesity, diabetes and atrial fibrillation.
7. **Diagnosis:** preserved ejection fraction (≥50%) plus objective evidence of raised filling pressures — echocardiographic **E/e′ >14–15**, septal e′ <7 cm/s, left atrial volume index >34 mL/m², tricuspid regurgitant velocity >2.8 m/s, LV hypertrophy — with raised natriuretic peptides.
8. **Management (textbook position):** treat congestion with diuretics, control heart rate with beta blockers to prolong diastolic filling, and treat the comorbidities that drive it — **hypertension, coronary disease, atrial fibrillation and diabetes**. Trials of digoxin and ARBs showed minimal benefit.
9. **Practical cautions:** these patients are preload-dependent — over-diuresis causes hypotension and acute kidney injury; avoid excessive rate reduction (chronotropic incompetence worsens exercise capacity) and avoid drugs that shorten diastole.
10. **Prognosis:** mortality approaches that of HFrEF, with hospitalisation driven as much by non-cardiac comorbidity as by cardiac events.

Latest Updates and Current Practice Update (concise)

- **The therapeutic desert has ended.** Contrary to the textbooks, HFpEF now has evidence-based drug therapy: **SGLT2 inhibitors** (EMPEROR-Preserved, DELIVER) reduce worsening HF events across the ejection fraction spectrum and are recommended for all; **finerenone** (FINEARTS-HF, 2024) reduced cardiovascular death and worsening HF events by 16%, including at EF ≥60%.
- **2026 ACC HFpEF Expert Consensus Decision Pathway**: SGLT2 inhibitor for all, plus a non-steroidal MRA (**finerenone preferred** over spironolactone), plus **semaglutide or tirzepatide** when BMI ≥30 kg/m²; diagnosis by sequential H2FPEF and HFA-PEFF scoring with systematic exclusion of mimics.
- **Amyloid must be excluded:** up to about 13% of older HFpEF patients with marked LV hypertrophy have transthyretin cardiac amyloidosis, which now has specific disease-modifying therapy.

> **Examiner's Tip**
>
> Write: "*Diastolic dysfunction, not systolic dysfunction, is the commonest substrate for heart failure in the elderly.*" · "*E/e′ greater than 14, left atrial enlargement and raised natriuretic peptides with a preserved ejection fraction establish the diagnosis.*" · "*HFpEF is no longer untreatable — SGLT2 inhibitors and finerenone now have outcome benefit.*" Adding the FINEARTS-HF result reliably earns a mark.

Q7. Cardiomyopathies — pathophysiology, aetiology, clinical features, diagnosis and treatment

15-MARK ANSWER | 18 POINTS

> **Textbook coverage note:** Neither *Pathy's* (6th ed.) nor *Essentials of Clinical Geriatrics* (7th ed.) contains a dedicated cardiomyopathy chapter. They refer to cardiomyopathy only in passing — as a cause of sudden cardiac death and ventricular arrhythmia, as an aetiology of heart failure (idiopathic dilated cardiomyopathy being relatively more prevalent outside the West), in relation to pacing in infiltrative cardiomyopathy such as sarcoidosis, and in describing idiopathic hypertrophic subaortic stenosis. The classification and management framework below is therefore drawn from current cardiology guidance, and is flagged accordingly.

Definition and Classification

1. **Cardiomyopathy** = myocardial disorder in which the heart muscle is structurally and functionally abnormal, in the absence of coronary disease, hypertension, valvular disease or congenital disease sufficient to explain the abnormality.
2. **Morphofunctional classification: dilated (DCM)**, **hypertrophic (HCM)**, **restrictive (RCM)**, **arrhythmogenic right ventricular cardiomyopathy (ARVC)**, and **non-dilated left ventricular cardiomyopathy (NDLVC)** — the last added by the 2023 ESC cardiomyopathy guideline.
3. **Aetiological classification:** genetic (familial) versus acquired (mixed forms exist).

Pathophysiology by Type

4. **Dilated:** ventricular dilatation with systolic dysfunction → reduced stroke volume → neurohormonal activation (RAAS, sympathetic) → adverse remodelling, mitral annular dilatation with functional regurgitation, mural thrombus and arrhythmia.
5. **Hypertrophic:** sarcomere protein mutations → myocyte hypertrophy and disarray with interstitial fibrosis → impaired relaxation, dynamic left ventricular outflow tract obstruction with systolic anterior motion of the mitral valve, microvascular ischaemia and arrhythmogenic substrate.
6. **Restrictive:** stiff, non-compliant ventricles with normal or near-normal systolic function and normal chamber size → severe diastolic impairment with biatrial enlargement and high filling pressures.
7. **Arrhythmogenic:** desmosomal mutations → fibrofatty replacement of myocardium → ventricular arrhythmia and sudden death.

Aetiology

8. **Dilated:** idiopathic/familial (titin, lamin A/C), ischaemic, alcohol, chemotherapy (anthracyclines, trastuzumab), viral myocarditis, thyroid disease, tachycardia-induced, peripartum, iron overload, nutritional (thiamine).
9. **Hypertrophic:** autosomal dominant sarcomere gene mutations (MYH7, MYBPC3); phenocopies include Fabry disease and amyloidosis.
10. **Restrictive (most relevant to geriatrics): cardiac amyloidosis** (transthyretin wild-type in the elderly, AL in plasma cell dyscrasia), sarcoidosis, haemochromatosis, endomyocardial fibrosis (important in tropical/Indian practice), radiation and hypereosinophilic syndrome.
1. **Indian context:** endomyocardial fibrosis, rheumatic heart disease as a differential, nutritional and alcoholic cardiomyopathy, and a relatively higher prevalence of idiopathic dilated cardiomyopathy than in the West.

Clinical Features

2. **Common to all:** exertional dyspnoea, fatigue, palpitations, syncope, embolic events, arrhythmia and sudden cardiac death.
3. **Dilated:** features of congestive failure with a displaced diffuse apex, S3, functional mitral regurgitation.
4. **Hypertrophic:** ejection systolic murmur that **increases** with Valsalva and standing and **decreases** with squatting; jerky/bisferiens pulse; murmur does not radiate to the carotids (contrast with aortic stenosis). *Essentials* warns that in older adults this may be misdiagnosed as aortic stenosis or mitral regurgitation, and that digoxin and diuretics may worsen symptoms.
5. **Restrictive/amyloid:** right-sided failure predominates with raised JVP, Kussmaul's sign, hepatomegaly, ascites, peripheral oedema; extracardiac clues of transthyretin amyloidosis include **bilateral carpal tunnel syndrome**, lumbar spinal stenosis, biceps tendon rupture, and intolerance of standard heart failure drugs because of hypotension.

Diagnosis

6. **ECG:** DCM — LBBB, poor R progression; HCM — LV hypertrophy with deep T inversion; amyloidosis — the classic mismatch of **low QRS voltages with echocardiographic LV thickening**, plus pseudo-infarct patterns and conduction block.

   Echocardiography defines the phenotype: chamber size, wall thickness, ejection fraction, outflow gradient, diastolic parameters and granular sparkling myocardium in amyloid; global longitudinal strain shows **apical sparing ("cherry-**

on-top") in amyloidosis.

7. **Advanced tests:** cardiac MRI with late gadolinium enhancement and T1/extracellular volume mapping; **^{99m}Tc-PYP/DPD bone scintigraphy** (Perugini grade ≥2) for transthyretin amyloidosis; serum free light chains with serum and urine immunofixation to exclude AL amyloidosis; genetic testing and family screening; endomyocardial biopsy where non-invasive tests are inconclusive; coronary angiography to exclude ischaemic disease.

Treatment

8. **DCM:** full guideline-directed HFrEF therapy (four pillars), device therapy (CRT/ICD), anticoagulation if AF or mural thrombus, and removal of the cause (alcohol, tachycardia, thyroid).
   HCM: beta blockers or verapamil; disopyramide for obstruction; avoid dehydration, vasodilators, digoxin and diuretic excess; septal reduction (myectomy or alcohol ablation) for refractory obstruction; ICD for high sudden-death risk.
   RCM/amyloid: cautious diuretics for congestion, treat arrhythmia, anticoagulate for AF regardless of risk score, and use disease-modifying therapy for transthyretin amyloidosis. Avoid digoxin and calcium channel blockers in amyloidosis (binding to amyloid fibrils increases toxicity).

Latest Updates (2023–Present)

- **2023 ESC Guideline on Cardiomyopathies** — the first standalone ESC cardiomyopathy guideline; introduces the phenotype **non-dilated left ventricular cardiomyopathy**, and places multimodality imaging and genetic testing with cascade family screening at the centre of the pathway.
- **Transthyretin cardiac amyloidosis is now a treatable disease of old age.** Three agents are approved: **tafamidis** (stabiliser, ATTR-ACT), **acoramidis** (stabiliser, ATTRibute-CM, FDA-approved 2024, halved cardiovascular hospitalisations), and **vutrisiran** (siRNA silencer, HELIOS-B, approved for ATTR-CM in March 2025, reduced all-cause death and recurrent cardiovascular events). Vitamin A supplementation is advised with silencer therapy. **Eplontersen** is in phase III (CARDIO-TTRansform).
- **2025 ACC Concise Clinical Guidance on ATTR-CM** (JACC) confirms that diagnosis can be made **non-invasively** by bone scintigraphy once a monoclonal protein screen is negative, and that MRAs and SGLT2 inhibitors — not previously used in amyloid — are now part of care. These agents slow progression but do not reverse established organ damage, so **early diagnosis is essential**.
- **Cardiac myosin inhibitors for obstructive HCM: mavacamten** (EXPLORER-HCM, VALOR-HCM) and **aficamten** (SEQUOIA-HCM) reduce outflow gradients and the need for septal reduction therapy — a completely new drug class since the textbooks were written.
- **Anticoagulation:** the 2024 ESC AF guideline recommends oral anticoagulation in **all** patients with AF and hypertrophic cardiomyopathy or cardiac amyloidosis, *irrespective of the CHA_2DS_2-VA score*.

Current Practice Update

Textbook position	Current evidence (2023–2026)
Cardiomyopathy mentioned only as a cause of heart failure and sudden cardiac death; no classification or management framework.	2023 ESC standalone cardiomyopathy guideline with five phenotypes and a genotype–phenotype pathway.
Cardiac amyloidosis effectively untreatable; diagnosis needed biopsy.	Non-invasive diagnosis by bone scintigraphy; three approved disease-modifying drugs (tafamidis, acoramidis, vutrisiran).
Obstructive HCM treated with beta blockers, verapamil, disopyramide and septal reduction.	Cardiac myosin inhibitors (mavacamten, aficamten) now available and reduce the need for invasive septal reduction.
Anticoagulation in AF guided by risk score alone.	Anticoagulate all patients with AF plus HCM or cardiac amyloidosis regardless of score.

Examiner's Tip

"*Cardiomyopathy is myocardial disease not explained by coronary, hypertensive, valvular or congenital heart disease.*" · "*In an older patient, low ECG voltages with echocardiographic left ventricular thickening should be assumed to be cardiac amyloidosis until excluded.*" · "*Bone scintigraphy with a negative monoclonal protein screen now allows non-invasive diagnosis of transthyretin cardiac amyloidosis.*" · "*The murmur of hypertrophic obstructive cardiomyopathy increases on Valsalva and standing and decreases on squatting — the reverse of aortic stenosis.*"

Q8. Cardiomyopathy (short note)

7-MARK ANSWER | 10 POINTS

1. **Definition:** structural and functional myocardial disease in the absence of coronary, hypertensive, valvular or congenital disease sufficient to explain it.
2. **Five phenotypes (2023 ESC):** dilated, hypertrophic, restrictive, arrhythmogenic right ventricular, and non-dilated left ventricular cardiomyopathy.
3. **Dilated:** dilated ventricle with reduced ejection fraction. Causes — idiopathic/familial, alcohol, chemotherapy, myocarditis, tachycardia-induced, thyroid, peripartum.
4. **Hypertrophic:** autosomal dominant sarcomere mutation with myocyte disarray; may obstruct the outflow tract. In older adults it may be mistaken for aortic stenosis; digoxin and diuretics worsen it.
5. **Restrictive:** stiff ventricles with preserved systolic function and biatrial enlargement. In the elderly the dominant cause is **cardiac amyloidosis**; endomyocardial fibrosis is important in Indian practice.
6. **Clinical features:** dyspnoea, fatigue, palpitations, syncope, embolism, arrhythmia and sudden death; right-sided congestion predominates in restrictive disease.
7. **Red flags for amyloidosis in an older patient:** HFpEF with marked left ventricular thickening, **low ECG voltages**, bilateral carpal tunnel syndrome, lumbar canal stenosis, and hypotension on standard heart failure drugs.
8. **Diagnosis:** ECG, echocardiography (including global longitudinal strain showing apical sparing), cardiac MRI with late gadolinium enhancement, ^{99m}Tc-PYP/DPD bone scintigraphy, serum free light chains and immunofixation, genetic testing, biopsy if needed.
9. **Treatment:** phenotype-directed — HFrEF four-pillar therapy and devices in dilated disease; beta blocker/verapamil, disopyramide and septal reduction in obstructive hypertrophic disease; cautious diuresis, arrhythmia control and disease-modifying therapy in amyloidosis.
10. **Family implications:** genetic counselling and cascade screening of first-degree relatives in inherited forms — a point examiners often expect but candidates omit.

Latest Updates and Current Practice Update (concise)

- **2023 ESC Cardiomyopathy Guideline** introduced the non-dilated left ventricular cardiomyopathy phenotype and mandated multimodality imaging plus genetic testing with family cascade screening.
- **Three approved drugs for transthyretin cardiac amyloidosis** — tafamidis, acoramidis (2024) and vutrisiran (2025) — have converted a formerly untreatable cause of HFpEF in the elderly into a treatable one; the textbooks predate all three.
- **Mavacamten and aficamten** (cardiac myosin inhibitors) are new first-in-class oral therapies for obstructive hypertrophic cardiomyopathy.

> **Examiner's Tip**
>
> For a short note, use a three-column mental table — *phenotype / cardinal abnormality / commonest cause in the elderly* — and finish with the amyloid red flags. Adding "*the textbooks describe cardiac amyloidosis as untreatable; three disease-modifying drugs are now licensed*" demonstrates current reading.

Q9. Dilated cardiomyopathy

7-MARK ANSWER | 10 POINTS

1. **Definition:** left ventricular or biventricular **dilatation with systolic dysfunction** not explained by abnormal loading conditions or coronary artery disease.
2. **Epidemiology:** Pathy's notes that **idiopathic dilated cardiomyopathy is relatively more prevalent in developing countries and Arabic populations** than in the West, and that non-ischaemic causes of heart failure (rheumatic disease, Chagas disease, hypertension) dominate outside high-income settings — directly relevant to Indian practice.
3. **Aetiology:** idiopathic/familial (titin truncating variants, **lamin A/C** — the latter associated with conduction disease and requiring early ICD), alcohol, anthracyclines and trastuzumab, viral myocarditis, tachycardia-induced, peripartum, thyrotoxicosis and hypothyroidism, iron overload, thiamine deficiency, and HIV.
4. **Pathophysiology:** myocyte loss and interstitial fibrosis → reduced contractility → neurohormonal activation (sympathetic, RAAS, natriuretic peptides) → progressive remodelling with eccentric hypertrophy, annular dilatation causing functional mitral regurgitation, stasis with mural thrombus, and re-entrant ventricular arrhythmia.
5. **Clinical features:** congestive heart failure with a displaced diffuse apex, S3 gallop, pansystolic murmur of functional mitral regurgitation, raised JVP, hepatomegaly and oedema; also arrhythmia, thromboembolism and sudden death.
6. **ECG:** sinus tachycardia, atrial fibrillation, left bundle branch block, poor R-wave progression, ventricular ectopy, non-sustained ventricular tachycardia.
7. **Echocardiography** is the key test: dilated chambers, globally reduced ejection fraction, functional mitral regurgitation, sometimes apical thrombus. Cardiac MRI with late gadolinium enhancement distinguishes ischaemic (subendocardial) from non-ischaemic (mid-wall) patterns and refines arrhythmic risk.
8. **Additional work-up:** coronary angiography or CT coronary angiography to exclude ischaemic cardiomyopathy; thyroid function, iron studies, HIV serology, alcohol history; genetic testing and family screening in familial disease.
9. **Treatment:** remove the cause (alcohol abstinence, rate control of incessant tachycardia, correct thyroid or iron abnormality); full **four-pillar HFrEF therapy** — ARNI or ACE inhibitor/ARB, beta blocker, mineralocorticoid receptor antagonist and SGLT2 inhibitor — plus diuretics for congestion, ivabradine if heart rate remains >70 in sinus rhythm, anticoagulation for atrial fibrillation or documented thrombus, CRT for conduction delay, and ICD in selected patients with a reasonable life expectancy.
10. **Prognosis and geriatric caveat:** some patients show substantial recovery of ejection fraction with optimal therapy ("heart failure with improved ejection fraction"), and therapy should **not** be withdrawn once function recovers (TRED-HF). In very frail elders, tolerability, symptom control and quality of life take precedence over full-dose uptitration and device therapy.

Latest Updates and Current Practice Update (concise)

- **SGLT2 inhibitors are now a first-line pillar** in dilated cardiomyopathy with reduced ejection fraction (DAPA-HF, EMPEROR-Reduced); the textbooks list them only as an emerging possibility.
- **STRONG-HF** supports rapid, protocol-driven uptitration of all four pillars within weeks of decompensation, with close biochemical and clinical review — replacing the traditional slow month-by-month titration.
- **2023 ESC Cardiomyopathy Guideline** makes genetic testing and cascade family screening standard in dilated cardiomyopathy, and highlights **lamin A/C and filamin C variants** as indications for earlier ICD implantation irrespective of ejection fraction thresholds.

> **Examiner's Tip**
>
> "*Dilated cardiomyopathy is ventricular dilatation with systolic dysfunction in the absence of abnormal loading conditions or coronary disease sufficient to explain it.*" · "*Always exclude a reversible cause — alcohol, tachycardia, thyroid disease and iron overload — before labelling it idiopathic.*" · "*Guideline-directed therapy should not be withdrawn when the ejection fraction recovers.*"

Q10. Atrial fibrillation and its management

15-MARK ANSWER | 19 POINTS

Definition and Epidemiology

1. **Atrial fibrillation (AF)** is uncoordinated atrial electrical activation with loss of effective atrial mechanical function — the commonest sustained arrhythmia in older people.
2. Prevalence rises from **<0.5%** at age 40–50 to **5–15%** at age 80. Men are affected earlier, but in old age there are more women with AF than men. Lifetime risk at age 55 is about 35%.
3. **Stroke risk rises steeply with age** — from about 1.5% per year at 50–59 years to **23.5%** at 80–89 years. Up to 30% of all ischaemic strokes are attributable to AF, and AF-related strokes are larger and more disabling.
4. AF also causes heart failure (in 20–30%), a 60% relative increase in risk of significant cognitive decline, impaired quality of life in over 60%, and up to a 3.5-fold increase in all-cause mortality.

Aetiology and Ageing Substrate

5. **Ageing changes:** atrial amyloid deposition, loss of sinoatrial pacemaker myocytes, and collagen/fibrous replacement of conducting tissue and myocardium.
6. **Risk factors:** hypertension (each 10 mmHg rise in systolic BP raises AF risk by 11–16%), heart failure, valvular disease (especially mitral), ischaemic heart disease, diabetes, obesity, obstructive sleep apnoea, alcohol, hyperthyroidism, chronic kidney disease and systemic inflammation.

Classification

7. **First detected → paroxysmal** (self-terminating, usually within 48 hours) → **persistent** (>7 days or requiring intervention) → **long-standing persistent** (≥1 year with a rhythm-control strategy still intended) → **permanent** (a statement of intent — no further attempt at sinus rhythm). Permanent AF is re-designated long-standing persistent if the plan changes.

Clinical Features and Diagnosis

8. May be asymptomatic (found on routine examination or device interrogation) or present with palpitations, dyspnoea, fatigue, angina, syncope, falls, heart failure, stroke or delirium.
9. ECG shows absent P waves with an irregularly irregular ventricular response. **A regular rhythm with a fibrillatory baseline in a patient with AF implies complete AV block with an escape rhythm** — a classic examination point. Slow ventricular rates are common in the elderly because of AV nodal disease.
10. Ambulatory monitoring is matched to symptom frequency: 24-hour Holter for daily symptoms; 48–72 hours or patch for several times weekly; 7-day for weekly; event recorder for monthly; implantable loop recorder for less frequent symptoms. Pacemaker-detected **atrial high-rate episodes >200 bpm** should prompt further investigation.

Management Pillar 1 — Stroke Prevention

1. Score every patient with **CHA_2DS_2-VASc**: Congestive heart failure 1, Hypertension 1, Age ≥75 **2**, Diabetes 1, Stroke/TIA/thromboembolism **2**, Vascular disease 1, Age 65–74 1, Sex category (female) 1 — maximum 9.
2. Anticoagulate all men with score ≥2 and women with score ≥3 (Class I); consider at 1 (men) and 2 (women). **Every patient over 75 with AF therefore qualifies** unless there is a major contraindication.
3. **DOACs are preferred over warfarin** in non-valvular AF (Class I). Warfarin (INR 2–3) remains mandatory for **moderate-to-severe mitral stenosis and mechanical prosthetic valves**, as no DOAC trial included these patients.
4. Warfarin reduces stroke by about **64%**; aspirin reduces it by only about 22% with substantial bleeding risk, so **antiplatelet therapy is no longer recommended for stroke prevention in AF**. BAFTA showed warfarin superior to aspirin in community elderly.
5. Use **HAS-BLED** to identify and correct modifiable bleeding risks and to flag patients needing closer review — **not** to withhold anticoagulation. *Essentials* lists among its "Don'ts": **do not withhold anticoagulation for atrial fibrillation because of fall risk.**
6. For patients with a genuine contraindication (recurrent intracranial haemorrhage, recurrent embolic stroke despite adequate anticoagulation), consider **percutaneous left atrial appendage occlusion** (WATCHMAN) — procedural complication risk about 4%; surgical LAA exclusion may be added during cardiac surgery.

Management Pillar 2 — Rate Control

7. **Beta blockers** are the most effective agents for controlling rate on exertion and are first-line with hypertension, ischaemic heart disease or stable LV dysfunction. **Non-dihydropyridine calcium channel blockers** (verapamil, diltiazem) are alternatives when airways disease is present but must be avoided in reduced LV systolic function and pulmonary oedema. **Digoxin** — use 0.125 mg/day in the elderly, monitor levels and renal function; it controls rate at rest but not on exertion. **Amiodarone** can be used for rapid control but long-term use is limited by thyroid, pulmonary, hepatic, ocular and skin toxicity. Lenient rate control (resting rate <110 bpm) was non-inferior to strict control in **RACE**

II.

Management Pillar 3 — Rhythm Control

8. Classical trials (AFFIRM, RACE, STAF, HOT CAFE, PIAF) showed **no survival advantage** for rhythm over rate control, so rhythm control was traditionally reserved for symptom relief. Options are cardioversion (electrical or pharmacological), antiarrhythmic drugs (amiodarone, sotalol, flecainide only if no structural disease) and catheter ablation. Ablation is effective in symptomatic paroxysmal AF and is **not less successful in older patients**, though success is higher with shorter AF duration; risks and benefits must be weighed individually.

Integrated Care

9. The **ABC pathway** (Avoid stroke — Better symptom control — Cardiovascular and comorbidity optimisation) improves outcomes in anticoagulated AF patients. Additional geriatric considerations: cognitive impairment reduces adherence (missed DOAC doses cause periods of under-anticoagulation because of the short half-life); dementia is not in itself a contraindication, especially in multi-infarct dementia where anticoagulation may halt stepwise decline; and warfarin interacts with many drugs and with vitamin K-rich foods.

Latest Updates (2023–Present)

- **2024 ESC/EACTS AF Guideline** replaces ABC with the **AF-CARE** framework: **C**omorbidity and risk-factor management, **A**void stroke and thromboembolism, **R**educe symptoms by rate and rhythm control, **E**valuation and dynamic reassessment.
- **The risk score has changed: CHA_2DS_2-VA** — sex category has been **removed** from the ESC score, giving a maximum of 8; anticoagulation is recommended at a score ≥2 and should be considered at 1, using the same threshold for men and women.
- **Subclinical/device-detected AF:** NOAH-AFNET 6 and ARTESIA led to a new IIb recommendation to consider anticoagulation in selected patients with device-detected subclinical AF at high thromboembolic risk without major bleeding risk factors.
- **Early rhythm control is now favoured** (EAST-AFNET 4): rhythm control within 12 months of diagnosis reduces cardiovascular outcomes; catheter ablation is a **first-line** option in suitable patients with paroxysmal AF. The threshold for early cardioversion has been reduced from 48 to **24 hours**, with a "wait-and-see" approach encouraged for spontaneous conversion.
- **Anticoagulate regardless of score** in AF with hypertrophic cardiomyopathy or cardiac amyloidosis; continue anticoagulation for ≥2 months after ablation irrespective of risk score.
- **Left atrial appendage occlusion:** LAAOS III showed a 33% reduction in stroke/systemic embolism when surgical LAA closure was added to anticoagulation during cardiac surgery (Class IA); the OPTION trial (2024–25) supports percutaneous LAAO as an alternative to DOAC after ablation in selected patients.
- **Factor XI inhibitors** (asundexian, abelacimab, milvexian) promised bleeding-free anticoagulation, but **OCEANIC-AF was stopped early for inferior efficacy** of asundexian versus apixaban — an important negative result. Trials in other populations continue.
- **India:** AF is under-detected and under-anticoagulated; rheumatic (valvular) AF remains common, and these patients require **warfarin, not a DOAC**. Cost, INR monitoring access and bleeding risk in the presence of untreated Helicobacter-related peptic disease are practical barriers.

Current Practice Update

Textbook position	Current evidence (2023–2026)
CHA_2DS_2-VASc with sex category; anticoagulate men ≥2, women ≥3.	**CHA_2DS_2-VA** (ESC 2024) — sex removed; single threshold ≥2 for both sexes.
ABC pathway for integrated care.	**AF-CARE** framework, with comorbidity and risk-factor control promoted to the first pillar.
Rate control equivalent to rhythm control; ablation second-line after drug failure.	**Early rhythm control improves outcomes** (EAST-AFNET 4); ablation is first-line in suitable paroxysmal AF.
Device-detected atrial high-rate episodes: investigate and treat as AF if confirmed on ECG.	Formal IIb recommendation to consider anticoagulation in high-risk subclinical AF (NOAH-AFNET 6, ARTESIA).
Cardioversion window of 48 hours.	Reduced to **24 hours**, with a wait-and-see strategy for spontaneous conversion.

Examiner's Tip

Draw the CHA_2DS_2-VASc table with the doubled items highlighted, then write: "*Every patient over 75 with atrial fibrillation should be anticoagulated unless there is a major contraindication.*" · "*Anticoagulation must not be withheld because of fall risk — the number of falls needed to offset the benefit of anticoagulation is impractically high.*" · "*The 2024 ESC guideline removed sex from the risk score, which is now CHA_2DS_2-VA.*" · "*Patients with rheumatic mitral stenosis or a mechanical valve must receive warfarin, not a DOAC.*"

Q11. Sick sinus syndrome

7-MARK ANSWER | 10 POINTS

1. **Definition: Sick sinus syndrome (sinus node dysfunction)** is a constellation of electrocardiographic abnormalities — sinus bradycardia, **chronotropic incompetence** (inadequate sinus response to exercise), sinoatrial exit block, and sinus arrest, particularly following paroxysms of atrial tachyarrhythmia (**tachy-brady syndrome**).
2. **It is primarily a disease of older people.** The substrate is age-related loss of specialised pacemaker myocytes in the sinoatrial node with collagen and fibrous tissue deposition, compounded by ischaemic and degenerative change from hypertension and coronary atherosclerosis.
3. **Associations:** both paroxysmal and chronic atrial fibrillation are commonly associated with sinus node disease; dysfunction may progress until no sinus beats occur at all, and conduction disease frequently coexists at the AV node.
4. **Secondary/reversible causes must be excluded first:** drugs (beta blockers including topical eye drops, verapamil/diltiazem, digoxin, amiodarone), hypothyroidism, hyperkalaemia and hypokalaemia, obstructive sleep apnoea, raised intracranial pressure, increased vagal tone, hypothermia, infiltrative disease (sarcoid, amyloid, haemochromatosis), infective endocarditis, Lyme and Chagas disease.
5. **Clinical features** (*Essentials*, Table 11-15) reflect reduced organ perfusion: **syncope, dizziness, palpitations, angina pectoris, congestive heart failure, insomnia and memory loss.** Falls and confusion are common atypical presentations in the elderly.
6. **Diagnosis** depends on **correlating symptoms with documented bradycardia**. Holter monitoring is the standard first test; longer monitoring (patch, event recorder, implantable loop recorder) is matched to symptom frequency. Exercise testing demonstrates chronotropic incompetence.
7. **Key caveat:** mild nocturnal bradycardia (>50 bpm) is normal and reflects increased overnight parasympathetic tone; sustained bradycardia in the absence of medication is **not** a normal finding in older people.
8. **Prognosis:** mortality appears **unaffected** by sinus node dysfunction itself; survival is determined by associated pathology such as ischaemic heart disease. Pacemakers relieve symptoms but do not reduce mortality.
9. **Treatment:** withdraw culprit drugs and correct reversible causes. *Essentials* states there is **no satisfactory medical therapy**; symptomatic patients require a permanent pacemaker. A pacemaker also permits continued use of rate-controlling drugs in tachy-brady syndrome.
10. **Pacing indications (2018 ACC/AHA/HRS, as quoted in Pathy's):** Class I — symptoms directly attributable to sinus node dysfunction, and bradycardia caused by clinically necessary guideline-directed therapy; Class IIa — tachy-brady syndrome where symptoms are attributable to bradycardia, and rate-responsive pacing for symptomatic chronotropic incompetence.

Latest Updates and Current Practice Update (concise)

- **2023 HRS/APHRS/LAHRS guideline on cardiac physiologic pacing** shifts emphasis from "which chamber" to "how physiological": conduction system pacing (His bundle or left bundle branch area) is preferred where a high ventricular pacing burden is anticipated, to avoid pacing-induced cardiomyopathy.
- **Leadless pacemakers** (Micra, Aveir) are now an established option for older patients with limited venous access, previous device infection or frailty, avoiding pocket and lead complications; dual-chamber and conduction-system leadless systems are in first-in-human evaluation (Heart Rhythm 2025).
- **Practice point unchanged:** the fundamental requirement remains symptom–rhythm correlation. Pacing an asymptomatic bradycardia produces no symptomatic benefit and exposes the patient to procedural risk.

> **Examiner's Tip**
>
> "*Sick sinus syndrome is a disease of the ageing sinoatrial node, characterised by sinus bradycardia, chronotropic incompetence, sinoatrial block, sinus arrest and the tachycardia–bradycardia syndrome.*" · "*Diagnosis requires correlation of symptoms with documented bradycardia, usually by Holter monitoring.*" · "*There is no satisfactory drug therapy; pacing relieves symptoms but does not improve survival.*" Listing the seven manifestations from Table 11-15 is an easy source of marks.

Q12. Cardiac pacemaker

7-MARK ANSWER | 10 POINTS

1. **Purpose:** a permanent pacemaker delivers electrical stimuli to maintain an adequate heart rate and, where possible, atrioventricular synchrony, in symptomatic bradyarrhythmia.
2. **Nomenclature (NBG code):** position 1 = chamber paced, 2 = chamber sensed, 3 = response to sensing, 4 = rate modulation (R). Thus **VVI** = ventricle paced/sensed/inhibited; **AAI** = atrium only; **DDD** = dual chamber; **DDDR** = dual chamber with rate response.
3. **Indications — sinus node dysfunction:** symptoms directly attributable to sinus node dysfunction (Class I); bradycardia from clinically necessary guideline-directed therapy (Class I); tachy-brady syndrome with symptomatic bradycardia (Class IIa); rate-responsive pacing for symptomatic chronotropic incompetence (Class IIa).
4. **Indications — AV block: acquired complete heart block regardless of symptoms** and **acquired second-degree Mobitz type II regardless of symptoms**, once reversible and physiological causes are excluded (both Class I); any degree of block with neuromuscular disease such as myotonic dystrophy type 1 or Kearns-Sayre syndrome (Class I); symptomatic Mobitz type I (Class IIa); unresolving advanced block after cardiac surgery or AV junction ablation (Class I). First-degree block is generally benign, but a PR interval >240–300 ms with left bundle branch block and lamin A/C mutation, or with attributable symptoms, may warrant pacing (Class IIa).
5. **Choice of mode:** DDD pacing is recommended over AAI or VVI when permanent pacing is needed for sinus node disease or AV block (Class I). Single-chamber VVI is appropriate when frequent ventricular pacing is not expected or when comorbidities limit the benefit of dual-chamber pacing (Class I). **An atrial lead should not be implanted in permanent or persistent AF when rhythm control is not planned (Class III).**
6. **Evidence base:** meta-analysis of CTOPP, MOST, UKPACE and related trials showed **no mortality difference** and no difference in new or worsening heart failure between physiological and ventricular pacing, but a **significant reduction in new-onset atrial fibrillation** with an atrial lead, and a borderline reduction in thromboembolic stroke. Cross-over studies showed better functional capacity and patient preference for dual-chamber pacing; active patients over 70 gain quality-of-life benefit from DDD pacing.
7. **Pacing-induced cardiomyopathy:** conventional right ventricular apical pacing causes non-physiological, dyssynchronous left ventricular activation. Patients with pre-existing left ventricular impairment and a high pacing burden may develop heart failure. **All patients should have a transthoracic echocardiogram before pacemaker implantation.**
8. **Physiological pacing options: CRT** (biventricular) adds a lead activating the lateral LV wall — the **BLOCK HF** trial showed about a 30% reduction in heart failure hospitalisation versus RV pacing in patients with reduced LV function and AV block. **His bundle pacing** and left bundle branch area pacing achieve a narrow paced QRS with near-normal ventricular contraction.
9. **Complications:** pneumothorax, cardiac perforation and tamponade, lead displacement, pocket haematoma and infection, device infection or endocarditis (a recognised cause of bradycardia and of infective endocarditis in the elderly), venous thrombosis, pacemaker syndrome (with VVI pacing), tricuspid regurgitation, and generator end-of-life.
10. **Geriatric decision-making:** the final choice of device is individualised, taking into account patient preference, comorbidities, life expectancy, dexterity, cognition and available resources — the "cheapest adequate device" is often the right answer in a frail patient, while an active 70-year-old benefits from dual-chamber or physiological pacing.

Latest Updates and Current Practice Update (concise)

- **2023 HRS/APHRS/LAHRS guideline on cardiac physiologic pacing**: conduction system pacing carries a Class IIa recommendation for bradycardia indications when LVEF is 35–50% and a substantial pacing burden is anticipated; the 2025 EHRA/ESC clinical consensus extends guidance on patient selection and on hybrid strategies (HOT-CRT, LOT-CRT).
- **Left bundle branch area pacing (LBBAP)** has largely overtaken His bundle pacing because of better capture thresholds and lead stability, and registry data show better outcomes than right ventricular pacing; randomised trials such as **PROTECT-HF** are ongoing.
- **Leadless pacemakers** are now routine in selected older patients (no pocket, no leads, lower infection risk); helix-based leadless systems capable of conduction system pacing entered first-in-human study in 2025.

> **Examiner's Tip**
>
> Give the NBG code in one line, then list Class I indications as bullet points — examiners reward "*complete heart block and Mobitz type II require pacing regardless of symptoms, once reversible causes are excluded*." · "*Dual-chamber pacing does not improve survival but significantly reduces new-onset atrial fibrillation and improves quality of life*." · "*Echocardiography before implantation identifies those at risk of pacing-induced cardiomyopathy, in whom conduction system pacing or CRT should be considered*."

Q13. Infective endocarditis

15-MARK ANSWER | 18 POINTS

Definition and Epidemiology in the Elderly

1. **Infective endocarditis (IE)** is microbial infection of the endocardial surface of the heart, typically involving native or prosthetic valves or intracardiac devices, with vegetation as the characteristic lesion.
2. **Nearly 50% of all cases now occur in older adults.** In people over 65 the incidence is around **four times** that of the general population, and mortality is roughly **twice** that of younger patients.
3. **Reasons for the rise:** a high prevalence of undiagnosed degenerative valve disease, increasing use of invasive procedures, and increasing implantation of prosthetic valves and cardiac devices.
4. **Sex distribution:** there is a male predilection overall, but the proportion of women affected rises progressively with age.

Predisposing Lesions and Risk Factors

5. **Underlying cardiac lesions in the elderly** are usually degenerative — calcific aortic stenosis, mitral annular calcification, mucoid mitral valve degeneration — plus prosthetic valves and cardiac implantable electronic devices. In India and other developing countries, **rheumatic valvular disease with viridans streptococci** remains a major pattern.
6. **Non-cardiac risk factors:** diabetes mellitus, and **gastrointestinal and genitourinary tract cancers**, which predispose to bacteraemia; also haemodialysis, intravascular catheters and poor dentition.

Microbiology

7. **Staphylococcus aureus** is the leading pathogen overall, although its relative share **decreases with age**.
8. **Enterococci and *Streptococcus bovis* (now *S. gallolyticus*) are emerging as major pathogens in the elderly**, reflecting genitourinary and gastrointestinal portals of entry. Isolation of *S. bovis* mandates **colonoscopy to look for colonic neoplasia**.
9. Coagulase-negative staphylococci dominate prosthetic valve and device infection; culture-negative endocarditis (5–7%) is usually due to prior antibiotics or fastidious organisms (HACEK, Coxiella, Bartonella, Brucella).

Clinical Features

10. **Classic:** fever with a new or changing regurgitant murmur, splinter haemorrhages, Osler nodes, Janeway lesions, Roth spots, splenomegaly, clubbing, microscopic haematuria.
11. **Atypical presentation in the elderly (high-yield):** fever is frequently absent or blunted; presentation may be confusion or delirium, malaise, anorexia and weight loss, falls, functional decline, unexplained anaemia, back pain from spondylodiscitis, stroke, or a heart failure exacerbation. Peripheral stigmata are less often present.
12. **Complications:** heart failure from valve destruction (the commonest indication for surgery), **conduction disturbance from perivalvular abscess** (a listed cause of bradycardia in Pathy's), systemic embolism including stroke and mycotic aneurysm, splenic and renal infarction, glomerulonephritis, and metastatic abscess.

Diagnosis

13. **Blood cultures are the cornerstone:** at least three sets from separate venepuncture sites **before** antibiotics, with adequate volume. Serology/PCR for fastidious organisms if cultures are negative.
14. **Echocardiography:** transthoracic first, but **transoesophageal echocardiography** is required when TTE is negative and suspicion persists, and in all prosthetic valve and device infections. Findings: vegetation, abscess, new prosthetic dehiscence, new regurgitation.
15. **Modified Duke criteria** — definite IE = 2 major, or 1 major + 3 minor, or 5 minor. Major: typical organism in two separate blood cultures, or evidence of endocardial involvement. Minor: predisposing condition, fever ≥38 °C, vascular phenomena, immunological phenomena, and microbiological evidence not meeting a major criterion.
16. Supporting tests: full blood count (normocytic anaemia, leucocytosis), ESR and CRP, urinalysis, renal function, ECG (new conduction delay suggests abscess), chest radiograph, and imaging of embolic sites.

Treatment and Prophylaxis

17. **Antibiotics:** prolonged, high-dose, bactericidal, intravenous therapy for 4–6 weeks, guided by culture and sensitivity, delivered with an infectious diseases and cardiology "endocarditis team". In older patients, **aminoglycoside use requires renal function and drug-level monitoring**, and doses of all agents must be adjusted for reduced creatinine clearance. Empirical regimens must cover staphylococci, streptococci and enterococci. **Surgery** is indicated for heart failure from valve dysfunction, uncontrolled infection or abscess, prosthetic valve endocarditis, and recurrent embolism with a large mobile vegetation — age alone is not a contraindication, but frailty and comorbidity strongly affect operative risk.
18. **Prophylaxis:** the AHA (Wilson 2007) withdrew routine subacute bacterial endocarditis prophylaxis for most lesions,

restricting it to the highest-risk cardiac conditions undergoing specified dental procedures. Pathy's argues that because gastrointestinal and genitourinary procedures are an important source of enterococcal and *S. bovis* bacteraemia in the elderly, **patient age should be factored into global risk assessment when deciding antibiotic prophylaxis**. Good oral and dental hygiene remains the most effective preventive measure.

Latest Updates (2023–Present)

- **2023 Duke-ISCVID criteria** (Fowler et al., *Clin Infect Dis* 2023) update the modified Duke criteria: the list of "typical" organisms is expanded (including *S. lugdunensis*, *Granulicatella*, *Abiotrophia*, *Gemella*, and — in the presence of intracardiac prostheses — coagulase-negative staphylococci, *Cutibacterium acnes*, *Corynebacterium striatum* and *Candida*); imaging criteria now include **cardiac CT and ^{18}F-FDG PET/CT**; and a surgical/pathological major criterion has been added. Validation studies show **higher sensitivity** (about 97.6% versus 93.2% for modified Duke) but **lower specificity**.
- **2023 ESC Guideline on Infective Endocarditis** formalises the **multidisciplinary "Endocarditis Team"**, incorporates PET/CT and cardiac CT into diagnosis, and gives explicit guidance on antibiotic prophylaxis and on early surgery.
- **Partial oral antibiotic therapy** — the **POET** trial and its long-term follow-up showed that switching to oral antibiotics after initial intravenous therapy in stabilised left-sided endocarditis is non-inferior and allows earlier discharge; the 2023 ESC guideline endorses this in selected patients, which is of particular value in frail older adults who deteriorate with prolonged hospitalisation.
- **Outpatient parenteral antimicrobial therapy (OPAT)** is now standard practice for the stable patient after the initial critical phase.
- **Device-related IE:** complete system extraction is recommended in confirmed cardiac implantable electronic device infection — an increasingly common scenario as pacemaker implantation rates in the elderly rise.

Current Practice Update

Textbook position	Current evidence (2023–2026)
Modified Duke criteria (2000) with echocardiography as the only imaging modality.	**2023 Duke-ISCVID criteria** — expanded microbiology, cardiac CT and FDG-PET/CT as major imaging criteria, added surgical criterion.
Prolonged intravenous antibiotics for the full 4–6 weeks.	**Partial oral therapy after stabilisation** (POET) is non-inferior and endorsed by ESC 2023; OPAT widely used.
Management by individual clinicians; surgery considered case by case.	Formal **Endocarditis Team** with early surgical referral is a guideline recommendation.
Prophylaxis withdrawn for most lesions (AHA 2007); Pathy's argues age should be considered.	Prophylaxis still limited to highest-risk cardiac conditions and dental procedures; the age-based argument remains a reasoned position rather than guideline policy.

Examiner's Tip

"*Almost half of all cases of infective endocarditis now occur in older adults, in whom the incidence is four times and the mortality twice that of younger patients.*" · "*In the elderly the underlying lesion is usually degenerative rather than rheumatic, and enterococci and Streptococcus bovis are emerging pathogens.*" · "*Isolation of Streptococcus bovis mandates colonoscopy to exclude colonic neoplasia.*" · "*Fever may be absent — consider endocarditis in any older patient with unexplained delirium, weight loss or a new murmur.*" · "*At least three sets of blood cultures must be taken before antibiotics are started.*"

Q14. Use of statins in the elderly

7-MARK ANSWER | 10 POINTS

1. **Mechanism:** statins inhibit HMG-CoA reductase, reducing hepatic cholesterol synthesis and upregulating LDL receptors. Beyond LDL lowering they have **pleiotropic effects** — plaque stabilisation, reduced inflammation and improved endothelial function. Three of four acute myocardial infarcts are thought to arise from rupture or erosion of a vulnerable plaque, which statins stabilise.
2. **Secondary prevention — strong evidence at all ages.** The Cholesterol Treatment Trialists' meta-analysis of 28 randomised trials showed significant benefit in patients with established vascular disease **in every age group, including those over 75**. Key trials in older patients: **PROSPER** (pravastatin 40 mg, age 70–82, 34% LDL reduction, RR 0.85 for cardiovascular events), **HPS** (simvastatin 40 mg) and **LIPS** (fluvastatin after PCI).
3. **Primary prevention — evidence is weaker with advancing age.** The same meta-analysis showed a **trend to smaller proportional risk reduction with increasing age**, and **no significant benefit in those over 70** without vascular disease. ALLHAT-LLT was neutral in older primary prevention.
4. **Guideline position:** ACC/AHA states it "may be reasonable" to initiate moderate-intensity statin in adults ≥75 with LDL-C 70–189 mg/dL; ESC/EAS says primary prevention statin "may be considered" over 75 if high risk (e.g. diabetes). Pathy's notes it may equally be reasonable **not** to prescribe.
5. **Time-to-benefit and lag time are decisive in geriatrics:** the time to benefit for statins in primary prevention is **at least two years**. If estimated life expectancy is shorter, the drug adds burden without benefit.
6. **Adherence is poor in the elderly:** only about **one in four** older patients is still taking a statin two years after prescription, and about **20% show primary non-adherence** — never starting the prescribed drug at all.
7. **Adverse effects:** statin-associated muscle symptoms (myalgia with normal CK; myositis with CK <10× ULN; **rhabdomyolysis with CK >10× ULN**), hepatotoxicity (act if ALT/AST >3× ULN), a small increase in incident type 2 diabetes, and drug interactions through CYP3A4 (amiodarone, verapamil, diltiazem, clarithromycin, azoles) which are especially relevant with polypharmacy. Concerns about cognitive impairment are not supported by good evidence.
8. **Practical monitoring:** check baseline lipids, ALT/AST, CK, TSH and renal function; recheck lipids and transaminases after 8–12 weeks. In statin-associated muscle symptoms with CK <10× ULN, reduce the dose or rechallenge with an alternative statin; if CK >10× ULN, stop the statin and check creatinine, urea and myoglobinuria. Persistent symptoms with rising CK should prompt testing for **anti-HMG-CoA reductase antibodies** (immune-mediated necrotising myopathy) or muscle biopsy.
9. **Intensity and add-ons:** high-intensity statins lower LDL-C by 50–55%; adding **ezetimibe** gives a further 20% reduction. In very high-risk atherosclerotic disease with LDL-C ≥70 mg/dL on maximal statin, ezetimibe then a **PCSK9 inhibitor** may be added.
10. **Decision framework for the older patient:** continue statins for secondary prevention unless life expectancy is very short; for primary prevention over 75 use **shared decision-making** weighing benefit against polypharmacy, frailty, life expectancy, adverse effects and the patient's own priorities. Deprescribing is appropriate in advanced frailty, end-stage disease or the last year of life.

Latest Updates and Current Practice Update (concise)

- **EWTOPIA 75** (Circulation 2019) showed benefit from **ezetimibe monotherapy** in Japanese patients ≥75 with LDL-C ≥140 mg/dL, and a 2020 *Lancet* meta-analysis (Gencer et al.) concluded that **LDL lowering is beneficial in patients over 75** — a shift toward treating rather than withholding.
- **STAREE** (atorvastatin 40 mg versus placebo in 9,971 community-dwelling Australians aged ≥70 without cardiovascular disease, diabetes or dementia; co-primary endpoints of disability-free survival and major cardiovascular events) completed follow-up at the end of 2025. It is the definitive trial for primary prevention in this age group; its results should be quoted as soon as published.
- **Newer non-statin agents** now available for statin intolerance or residual risk: **bempedoic acid** (CLEAR Outcomes 2023 — reduced major cardiovascular events in statin-intolerant patients, with no muscle toxicity), **inclisiran** (twice-yearly siRNA, useful where adherence is the barrier), and PCSK9 monoclonal antibodies.

> **Examiner's Tip**
> "*For secondary prevention, statins benefit patients of all ages including those over 75; for primary prevention the evidence weakens sharply after 70.*" · "*The time to benefit in primary prevention is at least two years, so life expectancy must exceed the lag time before prescribing.*" · "*Statin myopathy spans myalgia with normal CK, myositis with CK below ten times normal, and rhabdomyolysis above ten times normal.*" Naming PROSPER and the CTT meta-analysis, and mentioning STAREE as the awaited definitive trial, reliably earns marks.

Q15. Calcium channel antagonists

7-MARK ANSWER | 10 POINTS

1. **Mechanism:** calcium channel antagonists block **L-type voltage-gated calcium channels**, reducing calcium entry into vascular smooth muscle (vasodilatation) and into cardiac myocytes and nodal tissue (negative inotropy and negative chronotropy/dromotropy).
2. **Classification:**
   Dihydropyridines — amlodipine, nifedipine, felodipine, nitrendipine, lercanidipine: predominantly **vascular** selectivity, potent arterial vasodilators, minimal effect on the AV node.
   Non-dihydropyridines — **verapamil** (phenylalkylamine, most cardioselective) and **diltiazem** (benzothiazepine, intermediate): act directly on the **AV node**, slowing conduction.
3. **Why they suit older patients:** they are peripheral vasodilators that **maintain coronary blood flow**, and their **potency increases with age** — a consequence of reduced reflex tachycardia and reduced myocardial contractility in the elderly. They are especially effective in **isolated systolic hypertension**.
4. **Indications:** hypertension (particularly ISH and in older and Black patients), stable angina (added when symptoms persist despite long-acting nitrates and beta blockers), rate control in atrial fibrillation and supraventricular tachycardia (non-dihydropyridines), Prinzmetal/vasospastic angina, and Raynaud phenomenon.
5. **Choice by ventricular function (a key examination point):** in stable ischaemic heart disease, **rate-limiting agents (diltiazem, verapamil) may be used if left ventricular function is normal**, whereas **dihydropyridines (amlodipine, felodipine) should be used when ventricular function is impaired**.
6. **Evidence:** calcium channel blockers reduced stroke incidence in older patients with isolated systolic hypertension (**Syst-Eur**, Staessen 1997) and are **superior to other classes for stroke prevention**, but they **do not significantly reduce the risk of heart failure** — for which diuretics are superior.
7. **Adverse effects (dihydropyridines):** headache and flushing, **ankle oedema** (dose-dependent, not diuretic-responsive — best managed by dose reduction or by adding an ACE inhibitor/ARB), sodium retention, reflex tachycardia (short-acting agents), gum hyperplasia, and postural hypotension.
8. **Adverse effects (non-dihydropyridines): constipation** (prominent with verapamil and a real problem in older patients), bradycardia, AV block, and **negative inotropy** — they may precipitate or worsen heart failure and are contraindicated in HFrEF and in acute pulmonary oedema.
9. **Interactions and cautions:** combining verapamil or diltiazem with a beta blocker carries a substantially increased risk of **bradycardia, heart block and asystole**, particularly in older people; both are CYP3A4 inhibitors and raise levels of statins, digoxin and carbamazepine. *Essentials* lists among its "Don'ts": **do not use short-acting calcium channel antagonists for long-term therapy of hypertension.**
10. **Practical prescribing in geriatrics:** prefer long-acting once-daily dihydropyridines; start at low dose; review ankle oedema and constipation at each visit; check for bradycardia if combined with a beta blocker; and remember that calcium channel blockers are among the drugs that can cause bradycardia and are a recognised secondary cause of hyponatraemia-associated hypotensive states.

Latest Updates and Current Practice Update (concise)

- **2025 ACC/AHA and 2024 ESC hypertension guidelines** retain dihydropyridine calcium channel blockers as one of the four first-line classes, and both now strongly favour their use within **single-pill combinations** (typically with an ACE inhibitor/ARB and/or a thiazide-like diuretic) as initial therapy.
- The **ACE inhibitor + amlodipine** combination is preferred over amlodipine + diuretic for reducing ankle oedema and, from the ACCOMPLISH trial, for cardiovascular outcomes — a practical point not covered in the textbooks.
- Calcium channel blockers remain the safest antihypertensive class in **advanced chronic kidney disease** and in patients in whom hyperkalaemia limits RAAS blockade, which is common in the multimorbid elderly.

> **Examiner's Tip**
>
> Tabulate dihydropyridine versus non-dihydropyridine with three rows — *site of action, main use, main side effect*. Then write: "*Calcium channel antagonists increase in potency with age and are particularly effective in isolated systolic hypertension.*" · "*They are superior for stroke prevention but do not reduce heart failure risk.*" · "*Verapamil or diltiazem with a beta blocker risks bradycardia, heart block and asystole in older people.*"

Q16. Anticoagulants in geriatric practice — indications and agents

15-MARK ANSWER | 18 POINTS

Why Anticoagulation Matters in Old Age

1. Both the **risk of thrombosis and the risk of haemorrhage increase with age**. Older patients therefore stand to gain the most from anticoagulation and to lose the most from it — the central tension of geriatric prescribing.
2. Guidelines are largely extrapolated: patients in landmark trials are on average **5 to 10 years younger** than real-world patients, and most trials excluded those with high bleeding risk, renal failure or recent stroke. Consequently guidelines cannot make strong recommendations for people aged 85 and over.
3. **Under-treatment is the bigger problem.** Anticoagulation is widely withheld from older patients because of falls, dementia and perceived frailty, despite evidence that these are usually not valid reasons.

Indications

4. **Non-valvular atrial fibrillation** — long-term stroke prevention (the commonest indication in geriatric practice).
5. **Valvular AF and mechanical prosthetic valves** — lifelong warfarin.
6. **Treatment of venous thromboembolism** — deep vein thrombosis and pulmonary embolism.
7. **Thromboprophylaxis** in medical and surgical inpatients at moderate-to-high risk, and extended prophylaxis after major orthopaedic surgery.
8. Other: intracardiac thrombus, mitral annular calcification with cerebral embolism, thrombophilia with thrombosis, and antiphospholipid syndrome (warfarin, **not** a DOAC, in triple-positive disease).

Agents — Warfarin (Vitamin K Antagonist)

9. Inhibits vitamin K-dependent synthesis of factors **II, VII, IX and X**. Efficacy depends on time in therapeutic range. It reduces stroke in AF by about **64%** and mortality by about a quarter, and is superior to aspirin (relative risk reduction 39%).
10. **Target INR ranges** (Pathy's Table 24.5): **2.0–3.0** for DVT, pulmonary embolism, systemic embolism, mitral stenosis with embolism, TIA and atrial fibrillation; **3.0–4.5** for recurrent DVT/PE despite anticoagulation, arterial disease including myocardial infarction, and mechanical prosthetic heart valves.
11. **Geriatric problems:** dose requirement is lower than in younger patients; bleeding risk is increased at both high and normal INRs because of underlying pathology such as peptic ulcer, gastrointestinal malignancy and angiodysplasia; numerous drug interactions; and dietary vitamin K (parsley, broccoli, liver) reduces its effect.
12. **Key interactions** (Pathy's Table 29.7): *potentiate* — amiodarone, simvastatin, fibrates, thyroxine, corticosteroids, cimetidine, omeprazole, chlorpromazine, tricyclics, tamoxifen, aminoglycosides, metronidazole, clarithromycin; *reduce* — spironolactone, cholestyramine, barbiturates, carbamazepine, rifampicin.
13. **Initiation:** use a reduced-dose induction regimen in the elderly (Siguret's regimen: 4 mg daily for three days, then dose by INR). Monitor closely in the **first month**, when bleeding risk is highest.

Agents — Heparins

14. **Unfractionated heparin** potentiates antithrombin; narrow therapeutic range, complex kinetics, wide inter-patient variation. Monitor by **APTT ratio 1.5–2**. Reversal with protamine sulfate. Risk of heparin-induced thrombocytopenia.
15. **Low-molecular-weight heparin** has a longer half-life, better bioavailability and predictable kinetics, allowing once-daily subcutaneous dosing **without monitoring**. It acts more on factor Xa than thrombin, so it cannot be monitored by APTT — use an **anti-Xa assay** if monitoring is needed (renal impairment, extremes of weight, pregnancy). LMWH confers a **survival advantage in malignancy-associated thrombosis**.

Agents — Direct Oral Anticoagulants (DOACs)

16. **Dabigatran** (direct thrombin/factor IIa inhibitor); **rivaroxaban, apixaban, edoxaban** (factor Xa inhibitors). They are broadly equivalent to warfarin at an INR of 2–3, need no routine monitoring, and have far fewer food and drug interactions.
17. **Advantages in the elderly:** DOACs are recommended over warfarin in non-valvular AF (Class I) and are first choice for acute VTE in the absence of cancer or severe renal impairment, chiefly because of **lower intracranial haemorrhage risk**. In patients aged ≥75 the risk of major bleeding is significantly lower with DOACs than with vitamin K antagonists (relative risk about **0.49**).
18. **Limitations and cautions:** all are renally excreted to some degree and require dose reduction for renal impairment, low body weight and advanced age. **DOACs must not be used** in mechanical heart valves, moderate-to-severe mitral stenosis, triple-positive antiphospholipid syndrome, or where a target INR above 3 is required. Because of the short half-life, **missed doses create periods of under-anticoagulation** — a real risk in cognitive impairment. Dabigatran is unsuitable for dosette boxes; rivaroxaban and edoxaban are once daily, which may aid adherence.

Practical Geriatric Principles

- **Do not use aspirin as a substitute** for anticoagulation in AF or after VTE — it is ineffective and still causes bleeding.
- **Dementia is not an absolute contraindication**; in multi-infarct dementia the benefit of halting stepwise decline may be clear, and a supervised residential setting can solve adherence problems. End-stage dementia is a reasonable reason to stop.
- Review falls risk, alcohol, NSAID use, blood pressure control and interacting drugs; add a proton pump inhibitor where gastrointestinal risk is high.
- Reassess renal function, weight, haemoglobin and indication at least annually — anticoagulation is not a "set and forget" prescription.

Latest Updates (2023–Present)

- **Specific reversal agents are now established: idarucizumab** for dabigatran, and **andexanet alfa** for apixaban and rivaroxaban. ANNEXA-I (NEJM 2024) showed andexanet alfa achieved better haemostasis than usual care in intracranial haemorrhage but with more thrombotic events, so use requires careful case selection; four-factor prothrombin complex concentrate remains a widely used alternative.
- **Factor XI/XIa inhibitors** were expected to separate antithrombotic efficacy from bleeding. **OCEANIC-AF was stopped early because asundexian was inferior to apixaban** for stroke prevention in AF, tempering enthusiasm; abelacimab and milvexian continue in trials for cancer-associated and other thrombosis.
- **2024 ESC AF guideline** advises that patients over 75 who are stable on a vitamin K antagonist with good time in therapeutic range need not be switched to a DOAC.
- **Reduced-dose extended anticoagulation is now evidence-based: API-CAT** (NEJM 2025) showed reduced-dose apixaban 2.5 mg twice daily was non-inferior to full dose for extended treatment of cancer-associated VTE, with **less clinically relevant bleeding** — highly relevant to frail older patients.
- **India:** warfarin remains widely used because of cost and the high prevalence of rheumatic valvular disease and mechanical valves; DOAC use is expanding but is limited by price, by the absence of routine anti-Xa monitoring, and by the frequency of undiagnosed renal impairment. Reliable INR access through Ayushman Arogya Mandirs and structured anticoagulation clinics remain the practical priority.

Current Practice Update

Textbook position	Current evidence (2023–2026)
DOACs "not first-choice agents for malignancy-associated thrombosis, but this may change".	DOACs (apixaban, edoxaban, rivaroxaban) are now first-line for most cancer-associated VTE except luminal gastrointestinal and genitourinary tumours, where LMWH is preferred.
No specific antidote available for DOACs.	Idarucizumab and andexanet alfa are licensed reversal agents.
Extended anticoagulation uses full treatment doses.	Reduced-dose extended therapy (apixaban 2.5 mg bd, rivaroxaban 10 mg od) is non-inferior with less bleeding (API-CAT; AMPLIFY-EXT; EINSTEIN-CHOICE).
Anticoagulation choice guided by drug properties.	Structured shared decision-making with explicit discussion of stroke versus bleeding trade-off is now expected.

Examiner's Tip

Memorise the INR table — "*2.0–3.0 for venous thromboembolism and atrial fibrillation; 3.0–4.5 for recurrent thrombosis and mechanical prosthetic valves.*" · "*In patients over 75, DOACs approximately halve the risk of major bleeding compared with vitamin K antagonists.*" · "*Aspirin is not an acceptable substitute for anticoagulation in atrial fibrillation.*" · "*Neither falls risk nor dementia is by itself a contraindication to anticoagulation.*"

Q17. Venous thromboembolism and post-operative management of anticoagulation

15-MARK ANSWER | 18 POINTS

Epidemiology and Burden

1. **Venous thromboembolism (VTE)** = deep vein thrombosis (DVT) and/or pulmonary embolism (PE). Almost **60%** of all VTE events occur in people over 65.
2. Incidence rises **exponentially** with age — from <1 per 1000 person-years below 50 years to about **8 per 1000** person-years above 80.
3. Older patients are under-represented in trials; most VTE treatment trials excluded those with increased bleeding risk, renal failure or recent stroke, so evidence is extrapolated. Cohorts and registries (**RIETE, EPICA, SWITCO65+**) fill part of this gap.

Risk Factors

4. **Virchow's triad** — stasis, endothelial injury and hypercoagulability. In the elderly all three are amplified by immobility, surgery, and age-related increases in fibrinogen and platelet reactivity.
5. **Acquired:** immobility and hospitalisation, surgery (especially hip, knee and pelvis), fracture, malignancy, heart failure, stroke with paresis, infection and sepsis, dehydration, obesity, long travel, oestrogens, central venous catheters, nephrotic syndrome and frailty.
6. **Inherited thrombophilia** is present in up to **50%** of venous thromboses, becoming manifest under the above circumstances: factor V Leiden (commonest inherited defect — a single base substitution at position 1691 causing resistance to activated protein C), prothrombin G20210A, and deficiencies of **antithrombin, protein C and protein S**. Antiphospholipid syndrome is the important acquired thrombophilia.
7. **Testing caveat:** levels of antithrombin, protein C and protein S fall during acute thrombosis, and are further reduced by heparin (antithrombin) and warfarin (proteins C and S) — so test only when the patient is neither acutely thrombosed nor anticoagulated. A positive result should prompt family screening.

Diagnosis

8. **DVT algorithm:** assess clinical probability with the **two-level modified Wells score**. If "DVT likely" → complete venous ultrasonography. If "DVT unlikely" → D-dimer; if negative, no DVT and no treatment; if positive, proceed to ultrasound.
9. **PE algorithm (haemodynamically stable):** assess probability with the **revised Geneva score** or Wells score. Low/intermediate probability → D-dimer; if negative, exclude PE. High probability or positive D-dimer → CT pulmonary angiography.
0. **Age-adjusted D-dimer** is essential in the elderly, because specificity falls with age. Use a cut-off of **age × 10 µg/L in patients over 50** — validated in the **ADJUST-PE** study; this safely increases the proportion of older patients in whom PE can be excluded without imaging.
1. Note that asymptomatic DVT is common on admission in older medical inpatients, and that clinical signs and risk factors have different diagnostic value in the elderly than in the young.

Treatment

2. **DOACs are first choice** for acute VTE in the elderly in the absence of cancer or severe renal insufficiency, mainly because of lower intracranial bleeding risk than vitamin K antagonists.
3. **Regimens (Pathy's Table 37.3):**
   Dabigatran — LMWH/UFH/fondaparinux for at least 5 days, then 150 mg twice daily.
   Rivaroxaban — no lead-in: 15 mg twice daily for 21 days, then 20 mg once daily; extended phase 10 mg once daily.
   Apixaban — no lead-in: 10 mg twice daily for 7 days, then 5 mg twice daily; extended phase 2.5 mg twice daily.
   Edoxaban — LMWH lead-in for at least 5 days, then 60 mg once daily, reduced to 30 mg once daily if creatinine clearance 30–50 mL/min, body weight ≤60 kg, or concomitant strong P-glycoprotein inhibitors.
4. **Duration:** Pathy's recommends **three to six months** of anticoagulation in elderly patients with acute VTE, because bleeding risk is higher and the recurrence risk is no greater than in younger patients. Extended treatment is not routinely indicated and any such decision must be carefully justified. Existing recurrence-prediction rules are unhelpful in the elderly — HERDOO2 automatically classes those ≥65 as high risk, DASH is not validated, and the Vienna model lacks discrimination.
5. **Prognosis:** 30-day mortality doubles in those ≥65 compared with younger patients (11% versus 4%), and remains higher at one year (31% versus 14%). Use **PESI/sPESI** to identify low-risk PE suitable for early discharge. The strongest predictors of long-term mortality are systolic BP <100 mmHg, active cancer and age. The main causes of death in the first three months are cancer (38%), PE (26%), infection (13%) and bleeding (6%).
6. **Bleeding:** patients ≥65 have an almost twofold higher risk of major and clinically relevant non-major bleeding; age ≥75 at least doubles the risk of intracranial haemorrhage. Low physical activity level and polypharmacy (>4 drugs) predict

major bleeding.

Post-operative Management of Anticoagulation

7. **Pre-operative interruption.**
   Warfarin — stop 5 days before surgery; check INR the day before; give low-dose vitamin K if INR remains high.
   Heparin bridging is not required for most patients (BRIDGE trial showed no benefit and more bleeding); reserve bridging for very high thrombotic risk — mechanical mitral valve, recent (<3 months) VTE or stroke, or CHA_2DS_2-VASc ≥7.
   DOACs — follow the **PAUSE** protocol, based on drug, renal function and procedural bleeding risk: stop **1 day** before a low-bleeding-risk procedure and **2 days** before a high-bleeding-risk procedure for apixaban, rivaroxaban and edoxaban; for dabigatran, stop 1–2 days (CrCl ≥50) or 2–4 days (CrCl <50). **No bridging and no routine DOAC level testing.**
   The **ASRA** guidance requires a longer interval — at least 72 hours — before neuraxial anaesthesia and deep nerve blocks; where the two conflict, follow the anaesthetic guidance.
   No interruption is needed for minor dental procedures, cataract surgery or most skin procedures.
8. **Post-operative resumption and prophylaxis.**
   Restart **warfarin on the evening of surgery or the first post-operative day** at the usual maintenance dose, once haemostasis is secure and enteral intake has returned. Restart **DOACs 24 hours after low-bleeding-risk** and **48–72 hours after high-bleeding-risk** procedures, since the DOAC reaches full anticoagulant effect within hours. In the interval, give **prophylactic-dose LMWH** where the thrombotic risk is high and haemostasis is adequate. Continue mechanical prophylaxis (early mobilisation, graduated compression stockings, intermittent pneumatic compression) throughout. For major orthopaedic surgery (hip and knee replacement, hip fracture), give **extended thromboprophylaxis** for up to 35 days after hip and 14 days after knee surgery, using LMWH, a reduced-dose DOAC or aspirin per protocol. Two safety points: **parenteral vitamin K should not be used for non-life-threatening bleeding in patients with mechanical prosthetic valves**, because of the risk of inducing a hypercoagulable state; and after cardiac surgery, oral anticoagulation should be restarted as soon as intestinal function permits.

Latest Updates (2023–Present)

- **ASH 2023 guidelines** support home treatment of low-risk PE (sPESI 0 or Hestia-negative), which reduces hospitalisation-associated deconditioning and delirium in older patients.
- **API-CAT** (NEJM 2025): in cancer-associated VTE, extended **reduced-dose apixaban 2.5 mg twice daily was non-inferior** to full dose for preventing recurrence, with significantly **less clinically relevant bleeding**.
- **Extended treatment of provoked VTE** (NEJM, Aug 2025) supports reduced-dose apixaban after provoked events in selected patients with persistent risk factors — narrowing the old "provoked equals three months, full stop" rule.
- **PAUSE-2 pilot** (J Thromb Haemost 2025) examined DOAC interruption before high-bleed-risk surgery and neuraxial procedures, confirming that measurable residual DOAC activity can persist despite guideline-consistent interruption in some patients — supporting institution-specific protocols and selective drug-level testing.
- **BRUISE CONTROL-2** showed that continuing DOACs during pacemaker/ICD implantation was **not superior** to interrupting them for 48 hours — so interruption is acceptable for device procedures.
- An **AHA/ACC pulmonary embolism guideline (2026)** has been published, consolidating risk stratification, PE response teams, catheter-directed therapy and follow-up for post-PE syndrome.

Current Practice Update

Textbook position	Current evidence (2023–2026)
3–6 months of anticoagulation in the elderly; extended treatment "not indicated".	Extended **reduced-dose** therapy is now an evidence-based option in unprovoked VTE, cancer-associated VTE and persistent risk factors (API-CAT, AMPLIFY-EXT, EINSTEIN-CHOICE).
Perioperative interruption not addressed in detail.	Standardised **PAUSE** protocol (no bridging, no routine testing) is now the accepted approach for DOACs.
Heparin bridging implied for warfarin interruption.	BRIDGE trial: bridging is unnecessary for most, increases bleeding; reserve for the highest thrombotic risk.
Most PE managed as inpatients.	Low-risk PE (sPESI 0/Hestia-negative) can be treated at home.

Examiner's Tip

"Almost 60% of venous thromboembolic events occur in people over 65, and incidence rises exponentially from under 1 to about 8 per 1000 person-years." · "Use an age-adjusted D-dimer cut-off of age × 10 µg/L in patients over 50 — validated in ADJUST-PE." · "Bridging is not required for most patients undergoing warfarin interruption; for DOACs, stop one day before low-risk and two days before high-risk surgery and do not bridge." · "Restart DOACs 24 hours after low-bleeding-risk and 48 to 72 hours after high-bleeding-risk procedures."

Q18. Causes, treatment and prevention of deep vein thrombosis

15-MARK ANSWER | 18 POINTS

Definition and Significance

1. **Deep vein thrombosis (DVT)** is thrombus formation within the deep venous system, most often of the lower limb; proximal (popliteal and above) DVT carries the greatest risk of pulmonary embolism and of post-thrombotic syndrome.
2. **Venous thrombosis is primarily a disease of old age.** Asymptomatic DVT is common on admission among older medical inpatients, so a high index of suspicion is required.

Causes

3. **Virchow's triad:** venous stasis, endothelial injury, and hypercoagulability.
4. **Circumstantial/acquired factors:** immobilisation and hospitalisation, surgery — **particularly to the hip, knee and pelvis** — fracture, malignancy, obesity, heart failure, stroke with limb paresis, sepsis, dehydration, long-distance travel, central venous catheters, nephrotic syndrome and oestrogen therapy.
5. **Ageing-specific contributors:** reduced mobility and muscle pump action, higher fibrinogen levels, altered platelet function, increased prevalence of malignancy and heart failure, and frailty (an independent predictor of VTE).
6. **Inherited thrombophilia is manifest in up to 50% of cases** under the above circumstances: **factor V Leiden** (commonest; base substitution at position 1691 producing activated protein C resistance), prothrombin 20210A allele, and deficiencies of **antithrombin, protein C and protein S** (autosomal dominant, heterozygous only — the homozygous state is incompatible with life).
7. **Acquired thrombophilia:** antiphospholipid syndrome (lupus anticoagulant, anti-β2-glycoprotein-1 and anticardiolipin antibodies), myeloproliferative disease and paroxysmal nocturnal haemoglobinuria.
8. **Suspect thrombophilia when** thrombosis is unusually extensive, occurs at an unusual site, is spontaneous or recurrent, or there is a positive family history — though patients may present for the first time in old age.

Clinical Features and Diagnosis

9. Unilateral limb pain, swelling, warmth, erythema, dilated superficial veins and calf tenderness; phlegmasia in massive iliofemoral thrombosis. Differential diagnosis includes cellulitis, ruptured Baker's cyst, lymphoedema, chronic venous insufficiency and haematoma.
10. **Objective validation of the diagnosis must always precede treatment.** Use the two-level modified Wells score: "DVT likely" → complete venous ultrasonography; "DVT unlikely" → D-dimer, and if negative no further action.
11. Use the **age-adjusted D-dimer** (age × 10 µg/L above 50 years); D-dimer specificity is poor in the elderly because of comorbidity, infection and malignancy.
12. Consider occult malignancy in unprovoked DVT — a focused history, examination, basic bloods, and age-appropriate cancer screening rather than indiscriminate whole-body imaging.

Treatment

13. **First choice:** a DOAC in the absence of cancer or severe renal impairment. Apixaban and rivaroxaban need no parenteral lead-in; dabigatran and edoxaban require at least 5 days of LMWH first. Doses as in Q17.
14. **Alternative:** initial LMWH (or unfractionated heparin if renal failure or a need for rapid reversibility) overlapping with warfarin to a target **INR 2.0–3.0**. LMWH is the anticoagulant of choice in malignancy-associated thrombosis, where it confers a survival advantage.
15. **Duration:** three to six months in older patients. Consider lifelong anticoagulation after recurrent thrombosis, starting with a target INR of 2–3 and increasing to 3–4 if further thrombosis occurs while anticoagulated. **Triple-positive antiphospholipid syndrome must be treated with warfarin at the higher target range, not a DOAC.**
16. **Adjuncts:** early mobilisation and graduated compression stockings for symptom relief (they do not reliably prevent post-thrombotic syndrome); catheter-directed thrombolysis only for limb-threatening iliofemoral thrombosis; **inferior vena cava filter** only when anticoagulation is absolutely contraindicated, with removal as soon as anticoagulation can start.

Prevention

17. **Risk assessment for every hospital admission** (Padua or Caprini score) with reassessment when the clinical state changes. Pathy's emphasises that thromboprophylaxis of both **surgical and medical** patients at medium and high risk is increasingly recognised and practised.
18. **Methods.** *Mechanical* — early mobilisation, adequate hydration, graduated compression stockings and intermittent pneumatic compression (the options of choice when bleeding risk is high, and contraindicated in severe peripheral arterial disease). *Pharmacological* — LMWH once daily (dose-reduced in renal impairment), unfractionated heparin if creatinine clearance is very low, fondaparinux, or reduced-dose DOACs for **extended out-of-hospital prophylaxis after high-risk orthopaedic surgery** such as hip and knee replacement. Duration: for the admission in medical patients; up to **35 days after hip replacement or hip fracture** and **14 days after knee replacement**. Also address

modifiable factors — avoid dehydration, treat heart failure and infection, minimise immobility, and review oestrogens and other precipitants. In thrombophilia without prior thrombosis, management consists of **adequate prophylaxis at times of increased risk** such as surgery and immobilisation, rather than continuous anticoagulation.

Latest Updates (2023–Present)

- **ASH and ACCP guidance now favour DOACs over LMWH-warfarin for most acute DVT**, including in the elderly, and support outpatient management of low-risk DVT with defined follow-up.
- **Extended low-dose secondary prevention** is the major change: apixaban 2.5 mg twice daily or rivaroxaban 10 mg once daily after the initial treatment phase reduces recurrence with little excess bleeding (AMPLIFY-EXT, EINSTEIN-CHOICE), and **API-CAT** (NEJM 2025) confirmed non-inferiority with less bleeding for reduced-dose apixaban in cancer-associated VTE.
- **Compression stockings:** the SOX and IDEAL-DVT trials showed that routine two-year elastic compression does not prevent post-thrombotic syndrome; current practice is **symptom-guided, individualised compression** rather than universal prescription.
- **IVC filters:** the evidence (PREPIC-2 and subsequent registries) shows no mortality benefit and significant filter-related complications; retrievable filters should be removed promptly, and a formal retrieval pathway is now expected.
- **Aspirin** is an acceptable extended-prophylaxis option after elective hip and knee arthroplasty (CRISTAL and related trials), which matters in resource-limited Indian practice.
- **India:** VTE was historically considered rare in Indians, but hospital-based studies show rates comparable to Western populations after major surgery; under-use of prophylaxis in medical wards remains a documented gap, and mandatory admission risk assessment is the single most effective corrective step.

Current Practice Update

Textbook position	Current evidence (2023–2026)
Treatment "initially with heparin, subsequently with oral warfarin".	DOAC monotherapy (apixaban, rivaroxaban) is first-line, with no parenteral lead-in.
Extended treatment not indicated in the elderly.	Reduced-dose extended therapy is evidence-based and bleeds less than full dose.
Compression stockings recommended after DVT.	Not effective for preventing post-thrombotic syndrome; use for symptom relief only.
LMWH for malignancy-associated thrombosis.	DOACs are now first-line for most cancer-associated VTE, except luminal GI and GU tumours where LMWH is preferred.

Examiner's Tip

Open with Virchow's triad, then write: "*Objective validation of the diagnosis of venous thromboembolic disease should always precede the initiation of treatment.*" · "*In up to half of cases of venous thrombosis an underlying genetic predisposition becomes manifest under circumstantial risk.*" · "*Factor V Leiden is the commonest inherited prothrombotic disorder, caused by a single base substitution at position 1691 producing resistance to activated protein C.*" · "*Extended thromboprophylaxis is given for up to 35 days after hip replacement and 14 days after knee replacement.*"

Appendix — Rapid Revision Grid

NIGHT-BEFORE RECALL

Numbers you must be able to quote

Item	Value
BNP / NT-proBNP threshold for further investigation (non-acute)	>35 pg/mL / >125 pg/mL
BNP favouring cardiac cause of acute dyspnoea	>300 pg/mL
Ejection fraction categories	HFrEF ≤40%, HFmrEF 41–49%, HFpEF ≥50%
Digoxin dose and level in patients >70	0.125 mg/day; target 0.5–0.9 ng/mL
ARNI washout after ACE inhibitor	36 hours
HF prevalence >80 years; 2-yr and 5-yr mortality	13–14%; 60% and 75%
AF prevalence at 80; annual stroke risk at 80–89	5–15%; 23.5%
Warfarin vs aspirin in AF (relative risk reduction of stroke)	64% vs 22%
CHA_2DS_2-VASc maximum / CHA_2DS_2-VA maximum	9 / 8 (sex removed, ESC 2024)
INR ranges	2.0–3.0 (VTE, AF); 3.0–4.5 (recurrent VTE, mechanical valves)
Heparin monitoring	UFH: APTT ratio 1.5–2; LMWH: anti-Xa assay
Age-adjusted D-dimer cut-off	Age × 10 µg/L in patients >50 (ADJUST-PE)
VTE: proportion in >65s; incidence >80 years	≈60%; 8 per 1000 person-years
VTE anticoagulation duration in the elderly (Pathy's)	3–6 months
30-day mortality after VTE (≥65 vs <65)	11% vs 4%
DOAC major bleeding in ≥75s vs VKA	Relative risk ≈0.49
Extended orthopaedic thromboprophylaxis	Up to 35 days (hip); 14 days (knee)
Statin time-to-benefit in primary prevention	≥2 years
Statin myopathy CK thresholds	Myositis <10× ULN; rhabdomyolysis >10× ULN; act on ALT/AST >3× ULN
IE in the elderly: incidence and mortality vs younger	4× incidence; 2× mortality; ≈50% of all cases
2025 ACC/AHA BP target	<130/80 mmHg, with encouragement toward SBP <120
2024 ESC BP target	Systolic 120–129 mmHg (relaxed if ≥85, frail, or orthostatic hypotension)

Trial names by topic

Topic	Trials
Hypertension in the elderly	SHEP, Syst-Eur, HYVET, SPRINT-Senior, STEP, ESPRIT, BPROAD, PARTAGE, CAFE
HFrEF	CONSENSUS, SENIORS, SHIFT (ivabradine), PARADIGM-HF (ARNI), DAPA-HF, EMPEROR-Reduced, STRONG-HF, VICTORIA
HFpEF	CHARM-Preserved, I-PRESERVE, PEP-CHF, TOPCAT, PARAGON-HF (all neutral) → EMPEROR-Preserved, DELIVER, FINEARTS-HF, STEP-HFpEF, SUMMIT
Cardiomyopathy	ATTR-ACT (tafamidis), ATTRibute-CM (acoramidis), HELIOS-B (vutrisiran), EXPLORER-HCM/VALOR-HCM (mavacamten), SEQUOIA-HCM (aficamten), TRED-HF
Atrial fibrillation	AFFIRM, RACE/RACE II, BAFTA, EAST-AFNET 4, NOAH-AFNET 6, ARTESIA, LAAOS III, OPTION, OCEANIC-AF
Pacing	CTOPP, MOST, UKPACE, BLOCK HF; 2023 HRS/APHRS/LAHRS physiologic pacing guideline
Statins	PROSPER, HPS, LIPS, ALLHAT-LLT, EWTOPIA 75, CLEAR Outcomes, STAREE (awaited)
Anticoagulation / VTE	ADJUST-PE, AMPLIFY-EXT, EINSTEIN-CHOICE, API-CAT, PAUSE / PAUSE-2, BRIDGE, BRUISE CONTROL-2, PREPIC-2, SOX, ANNEXA-I
Endocarditis	POET (partial oral therapy); 2023 Duke-ISCVID criteria; 2023 ESC IE guideline

Eight lines that earn marks in any cardiovascular geriatrics answer

1. "Older patients are systematically under-represented in cardiovascular trials, so guideline recommendations are largely extrapolated."
2. "Presentation is frequently atypical — delirium, falls, anorexia and functional decline rather than classical symptoms."
3. "Treatment intensity should be proportionate to functional status, comorbidity and life expectancy, not to chronological age."
4. "Start low, go slow, and review renal function and electrolytes after every dose change."
5. "Time-to-benefit must be shorter than remaining life expectancy before a preventive drug is started."
6. "Neither falls risk nor dementia is by itself a contraindication to anticoagulation."
7. "Deprescribing is an active clinical decision, not a failure of care."
8. "Involve the multidisciplinary team, and discuss goals of care and advance planning before the terminal admission."

Indian Practice — Points to Weave In

- **Hypertension:** LASI Wave 1 — prevalence ≈46% in adults ≥45 years, control only ≈32%. IHCI (ICMR/MoHFW/WHO-India) raised control in enrolled cohorts from ≈26% to ≈60% using standardised protocols, single-pill combinations and free drugs through Ayushman Arogya Mandirs; NPHCE provides the geriatric service framework.
- **Heart failure:** less ischaemic and more rheumatic, hypertensive and idiopathic dilated cardiomyopathy; patients present younger and later in the disease course.
- **Atrial fibrillation:** rheumatic (valvular) AF remains common — these patients require **warfarin, not a DOAC**. Cost and INR access are the practical constraints.
- **Endocarditis:** rheumatic valve disease with viridans streptococci is still a major pattern alongside the degenerative pattern of high-income countries.
- **VTE:** once thought rare in Indians, but post-surgical rates are comparable to Western data; under-use of prophylaxis in medical wards is the documented gap. Aspirin is an acceptable extended prophylaxis option after arthroplasty in resource-limited settings.
- **Restrictive cardiomyopathy:** remember **endomyocardial fibrosis** in the Indian differential diagnosis.

End of document. *Textbook content is drawn from Pathy's Principles and Practice of Geriatric Medicine (6th edition) and Essentials of Clinical Geriatrics (7th edition). Material dated 2023 onward is drawn from current peer-reviewed literature and professional society guidance and is presented separately under "Latest Updates" and "Current Practice Update" in each answer. Where the uploaded textbooks do not cover a topic, this has been stated explicitly.*

RESPIRATORY MEDICINE IN THE ELDERLY

High-Yield Examination Answers

MD Geriatric Medicine — Structured Revision Series

Nine questions • COPD • GOLD • Pneumonia • Pulmonary Embolism
Interstitial Lung Disease • Respiratory Failure • LTOT • MDI • Extrapulmonary TB

Primary source: Pathy's Principles and Practice of Geriatric Medicine, 6th edition (Sections 4 & 5) and Essentials of Clinical Geriatrics, 7th edition.

Supplemented by: GOLD 2026 • 2026 AHA/ACC Acute PE Guideline • ATS/IDSA CAP • ERS/ATS • WHO & NTEP (India) • ICMR/API • ACIP 2024–25.

Format: 15–20 numbered points for 15-mark answers; 8–10 points for 7-mark answers, followed by Latest Updates, Current Practice Update table, and Examiner's Tip. Recurring topics carry an additional green panel.

Contents

1.	Chronic obstructive pulmonary disease — risk factors, pathology, clinical features, differentiation from bronchial asthma, management and complications **(recurs)**	15 marks
2.	GOLD criteria for assessment of COPD and treatment options	15 marks
3.	Community-acquired pneumonia in the elderly — etiology, clinical features and management	15 marks
4.	Causes, diagnosis and management of pulmonary embolism	15 marks
5.	Interstitial lung disease	7 marks
6.	Respiratory failure — types, causes and management	15 marks
7.	Long-term oxygen therapy	7 marks
8.	Metered dose inhalers	7 marks
9.	Extrapulmonary tuberculosis	15 marks
	Appendix — Rapid Revision Grid	

Age-related changes in the respiratory system (applies to all answers)

Pathy's Table 42.1 — worth writing as a two-line preamble to any respiratory answer.

Structure	Change with ageing	Functional consequence
Lung parenchyma	Altered collagen network, dilated alveolar ducts, loss of **elastic recoil**	Air trapping, hyperinflation (↑FRC, ↑RV); a **"pseudo-obstructive" spirometry pattern**
Chest wall	Kyphosis, rib-cage stiffening, flattened diaphragm, loss of respiratory muscle mass	↓Chest wall compliance, ↓TLC → **restrictive pattern**; ↑work of breathing
Control of breathing	Blunted efferent output to respiratory muscles	**Blunted ventilatory response to hypoxia and hypercapnia** → low threshold for respiratory failure
Immunity	Replicative senescence, ↑pro-inflammatory cytokines	Lung **"inflammageing"**; ↓mucociliary clearance and cough → infection risk

1. Chronic Obstructive Pulmonary Disease — Risk Factors, Pathology, Clinical Features, Differentiation from Bronchial Asthma, Management and Complications 15 marks

High-Yield Points

Definition and Epidemiology

1. **Definition:** COPD is airflow obstruction that is **not fully reversible** and shows **little variability over time**. It is usually progressive and is caused by small-airway disease plus emphysema.
2. **Burden:** COPD is the **fifth-largest cause of disability worldwide** and the fourth-leading cause of death in the US. World prevalence is **4–10%**, but in the elderly it rises to **12–15%**. Despite this it remains under-diagnosed and mis-diagnosed in older adults.

Risk Factors

3. **Tobacco smoke** is the main risk factor. FEV_1 normally falls by **25–30 mL/year after age 25**; in a susceptible smoker it falls by **more than 50 mL/year**. Stopping smoking slows this decline back towards normal.
4. **One in four to five COPD patients has never smoked.** In India the important non-smoking causes are **biomass fuel smoke** (chulha smoke in poorly ventilated kitchens), outdoor air pollution, occupational dust, and **post-tuberculous lung damage**. Pathy's notes that kitchen ventilation, rural residence, low BMI and low education all alter prevalence.
5. **Alpha-1 antitrypsin deficiency** should be screened for in **every** COPD patient regardless of age (European Respiratory Society statement).

Pathology and Pathophysiology

6. Inhaled irritants cause **chronic neutrophilic airway inflammation**, mucous gland hyperplasia and goblet cell metaplasia → chronic bronchitis; and **protease–antiprotease imbalance** destroying alveolar walls → emphysema.
7. Loss of elastic recoil and small-airway fibrosis cause **expiratory flow limitation → dynamic hyperinflation** and air trapping → breathlessness on exertion, flattened diaphragm and inefficient respiratory muscles.
8. Ventilation–perfusion mismatch causes **hypoxaemia**; later, alveolar hypoventilation causes **hypercapnia**. Chronic hypoxic pulmonary vasoconstriction → pulmonary hypertension → **cor pulmonale**.

Clinical Features

9. **Insidious onset** of exertional breathlessness, chronic cough with sputum, wheeze and chest tightness; symptoms vary little from day to day. Older patients often **attribute symptoms to old age**, which delays diagnosis.
10. Signs: barrel chest, pursed-lip breathing, reduced cricosternal distance, hyperresonance, distant breath sounds, prolonged expiration. Signs of **cor pulmonale** — raised JVP, hepatomegaly, ankle oedema, loud P2.
11. **Investigations:** post-bronchodilator spirometry (**FEV_1/FVC <0.7**, or better **<lower limit of normal** in the elderly); chest X-ray in all new cases to exclude cancer; **12-lead ECG** (right heart strain carries poorer prognosis); echocardiography if heart failure suspected; TLCO and CT if symptoms are out of proportion. Good-quality spirometry is achievable in about **85% of older adults** — **cognitive impairment is the commonest reason for failure**.

Differentiation from Bronchial Asthma

12. The key question is whether obstruction is **fixed (COPD)** or **reversible (asthma)**. A **>200 mL rise in FEV_1** after a beta-2 agonist or after a two-week corticosteroid trial is strong evidence of asthma. **PEF variability >20%** also suggests asthma.
13. Where results are ambiguous, use more discriminating tests: **a rise in total lung capacity is rare in asthma but typical of severe COPD**, and a **reduced TLCO** indicates emphysema (loss of alveolar-capillary surface), not asthma. **FeNO** is a simple non-invasive marker of eosinophilic (asthmatic) inflammation and is valid in older people.

Feature	COPD	Asthma in the elderly
Smoking history	Usually a heavy smoker	Often a non-smoker
Symptom pattern	Persistent, slowly progressive	**Variable**, episodic, **nocturnal** symptoms
Reversibility	Absent or <200 mL	**>200 mL rise in FEV_1** (may be only partial in the old because of airway remodelling)
PEF variability	Low	**>20%** diurnal variation
Total lung capacity	**Raised** in severe disease	Normal (a rise is rare)
TLCO (diffusion)	**Reduced** (emphysema)	Normal
Methacholine challenge	May be positive (non-specific)	Positive; **a negative test rules out asthma**
Airway inflammation	Neutrophilic	Eosinophilic; **FeNO raised**

Pathy's caution: *reversibility testing must be interpreted carefully in the elderly — there is a physiological decline in beta-2 adrenoceptor density and blunted bronchodilator response, so an absent response does not exclude asthma. The two conditions frequently* ***overlap*** *(asthmatic smokers).*

Management — Stable Disease

14. **Non-pharmacological (do these first): smoking cessation** using nicotine replacement, bupropion or varenicline — quit rates in older adults are as good as or better than in the young; **e-cigarettes are not recommended**. Add **influenza and pneumococcal vaccination**, **pulmonary rehabilitation**, nutritional support, self-management plans and early discussion of advance care planning.
15. **Inhaled therapy (Pathy's Figure 42.1):** avoid regular on-demand short-acting bronchodilators in the elderly (risk of overdosing and adverse drug reactions). Start a **LAMA** (tiotropium, umeclidinium, aclidinium — the latter two are safe in renal failure as they are liver-metabolised) or a **LABA**; step up to **LABA+LAMA**; add an **ICS** only for frequent exacerbators or asthma-like features. **ICS raise the risk of non-fatal pneumonia**, but this does not outweigh their benefit in the right patient.
16. **Add-on options:** slow-release theophylline in low dose (narrow therapeutic index, interacts with antibiotics); **roflumilast** (PDE-4 inhibitor) for frequent exacerbators with chronic bronchitis — watch weight loss, diarrhoea and mood change; high-dose acetylcysteine as a mucolytic/antioxidant; nebulised bronchodilators only when inhaler technique is irretrievably poor. **Maintenance oral steroids: risks generally outweigh benefits.**
17. **LTOT** if PaO_2 <7.3 kPa, or 7.3–8.0 kPa with polycythaemia, pulmonary hypertension, peripheral oedema or nocturnal hypoxaemia; used **≥15 hours/day** improves survival (see Question 7).

Management — Acute Exacerbation

18. **Definition and burden:** increased breathlessness, cough, sputum volume or purulence, wheeze and chest tightness. Exacerbations account for up to **10% of all medical admissions**; about **one-third are readmitted within 30 days** and **15% die by three months**. Commonest bacteria in the elderly are **H. influenzae, S. pneumoniae and M. catarrhalis**.
19. **Treatment:** nebulised bronchodilators; **oral corticosteroids for 7–14 days** (shorten stay, reduce treatment failure); **antibiotics when sputum is purulent** — these reduce mortality by 77% and treatment failure by 53%; avoid antibiotics if there is no purulence. Cover *Pseudomonas* (ciprofloxacin/anti-pseudomonal beta-lactam) if bronchiectasis coexists.
20. **Oxygen and ventilation:** target saturation **88–92%** (Pathy's text prints "890–95%", a typographical error for 88–92/90–95%) — excessive oxygen depresses hypoxic drive and worsens acidosis. Give **NIV if pH <7.35** despite maximal therapy: it reduces mortality, intubation and length of stay. **High-flow nasal oxygen** is safe and effective in both normocapnic and hypercapnic exacerbation, and is useful where there is a do-not-intubate order. Consider hospital-at-home/early-discharge schemes for patients without acidosis and with good social support.

Complications

21. **Respiratory:** recurrent exacerbations, pneumonia, respiratory failure (type 2), pneumothorax, secondary polycythaemia, **cor pulmonale**, lung cancer, coexisting bronchiectasis (found on HRCT in up to **29%** of primary-care COPD with chronic bronchitis).
22. **Systemic: heart failure is present in 20% of COPD patients over 65**; COPD is an independent cardiovascular risk factor. Also osteoporosis (screen routinely), sarcopenia and weight loss, chronic renal failure (masked by sarcopenia lowering creatinine), anxiety and depression, cognitive impairment, dysphagia and pulmonary embolism.

Latest Updates (2023–Present)

- **GOLD 2026 (November 2025)** — the largest revision in years: **a single moderate exacerbation now places the patient in Group E** and should trigger treatment escalation, with the goal of a "**low disease activity**" state (no exacerbations). A new section on **disease activity**, a completely rewritten exacerbation chapter, a rewritten multimorbidity chapter, and a new chapter on **artificial intelligence and emerging technologies** were added.
- **Biologics enter the COPD pathway (GOLD 2026, new Figure 3.11). Dupilumab** (anti-IL-4Rα; BOREAS and NOTUS trials, ~30% reduction in moderate-to-severe exacerbations) and **mepolizumab** (anti-IL-5; METREX/METREO/MATINEE, ~18–21% reduction) now carry **Level A evidence** and are FDA-approved for eosinophilic COPD (blood eosinophils generally ≥300 cells/µL) already on triple therapy. **Benralizumab did not reduce exacerbations.**
- **Cardiopulmonary link:** GOLD 2026 highlights that even **one moderate exacerbation raises the risk of a major cardiac event (arrhythmia, stroke, ischaemic heart disease, heart failure) within the next 30 days** — the rationale for "zero tolerance" of exacerbations.
- **Early diagnosis:** GOLD 2026 adds a formal **COPD case-finding algorithm** and a figure on factors associated with under-diagnosis; global COPD cases are projected to rise **23% between 2020 and 2050**, approaching 600 million, with the greatest growth in women and in low- and middle-income regions.
- **Vaccination:** influenza, pneumococcal, COVID-19, Tdap, zoster and now **RSV vaccination** recommended; ACIP lowered the age for routine **pneumococcal conjugate vaccine to 50 years** (October 2024; MMWR January 2025).

Current Practice Update

Issue	Textbook position (Pathy's 6e, 2022)	Current evidence (2023–2026)
Exacerbation threshold for escalation	Frequent exacerbator = **≥2 exacerbations** or ≥1 hospitalisation in a year	**GOLD 2026: one moderate exacerbation is enough** to move to Group E and escalate therapy
Initial inhaled therapy	Single long-acting bronchodilator (LAMA or LABA) first	**Dual bronchodilation (LABA+LAMA) is the preferred initial step** for most symptomatic patients
Role of ICS	Reserved for frequent exacerbators or asthma-like features; pneumonia risk noted	Unchanged in principle but now **biomarker-guided by blood eosinophil count**
Biologic therapy	Not mentioned for COPD	**Dupilumab and mepolizumab** approved as add-on for eosinophilic COPD on triple therapy
Diagnostic ratio	Fixed FEV_1/FVC <0.7 post-bronchodilator	Fixed ratio retained by GOLD 2026, but **LLN preferred in the elderly** to avoid over-diagnosis
Beta-blockers	Historically avoided; recent data reassuring	**Cardioselective beta-blockers should be used when indicated** — no longer contraindicated

> **Examiner's Tip**
>
> Open by writing the definition verbatim: "*COPD is characterised by airflow obstruction that is not fully reversible and shows little variability over time, usually progressive, comprising small-airway disease and emphysema.*" Then earn easy marks with three numbers: **normal FEV_1 decline 25–30 mL/year vs >50 mL/year in susceptible smokers**; **heart failure in 20% of COPD patients over 65**; **NIV indicated at pH <7.35**. Always draw the COPD-versus-asthma table — it is the single highest-scoring element of this question. Finish with a geriatric line: "*In the older patient, management is judged by improvement in symptoms, exercise tolerance and ADLs rather than by change in FEV_1, which is less relevant.*"

Extra Points to Remember — This Topic Recurs

- **Pathy's Table 42.4 — comorbidity classification, a favourite viva question.** Comorbidities are grouped as: **co-causal** (share smoking as a risk factor: coronary and cerebral atherosclerosis, chronic renal failure, lung and bladder cancer, congestive heart failure, aortic aneurysm, pulmonary fibrosis); **coexisting** (share only age: glaucoma, diabetes, benign prostatic hypertrophy, degenerative joint disease, hypertension, obstructive sleep apnoea); and **complicating COPD** (anxiety/depression, cognitive impairment, osteoporosis, sarcopenia, arrhythmias, dysphagia, pulmonary embolism).
- **Pathy's Table 42.3 — differential diagnosis of COPD:** asthma, congestive heart failure (orthopnoea, bibasal crackles, B-lines on ultrasound, restrictive spirometry), bronchiectasis (large-volume purulent sputum, clubbing, airway thickening), interstitial lung disease (dry cough, fine basal crackles, restrictive pattern, impaired diffusion), kyphoscoliosis (restrictive pattern with **normal** diffusion).
- **Multidimensional assessment** in the older COPD patient: MRC dyspnoea scale, Manchester Respiratory ADL scale, shuttle walk or six-minute walk test, BMI, oxygen saturation (if FEV_1 <50% or signs of cor pulmonale), Hospital Anxiety and Depression Scale, and exacerbation rate. Quote the **BODE index** (BMI, Obstruction, Dyspnoea, Exercise capacity) as the validated prognostic tool.
- **Sarcopenia masks renal failure:** low muscle mass blunts the rise in creatinine, so moderate (GFR 30–60) and even severe renal impairment goes unrecognised — important when prescribing antibiotics during exacerbations and renally cleared drugs.
- **Mortality predictors in older COPD (Pathy's):** disability, low BMI, lung function and use of LTOT. About **50% of LTOT patients survive three years**.
- **Palliative care:** patients in their last year should enter an end-of-life register and be offered **opioids and benzodiazepines** for refractory breathlessness. Pathy's states plainly that the switch from active to palliative approach "all too often comes too late" — a valuable closing line.

2. GOLD Criteria for Assessment of COPD and Treatment Options 15 marks

High-Yield Points

The GOLD Initiative and Diagnosis

1. **GOLD** = Global Initiative for Chronic Obstructive Lung Disease, established **1997**, first strategy report **2001**, revised every November alongside World COPD Day.
2. **Diagnostic criterion:** in the right clinical context, a **post-bronchodilator FEV_1/FVC <0.70** confirms persistent airflow obstruction. Post-bronchodilator testing avoids labelling reversible disease (asthma) as COPD.
3. **Geriatric caveat:** the FEV_1/FVC ratio falls physiologically with age, so the fixed 0.70 cut-off **over-diagnoses COPD in normal older people**. Pathy's and GOLD both prefer the **lower limit of normal (LLN)** in this age group. NICE adds that if FEV_1 is ≥80% predicted, diagnose only when respiratory symptoms are present.

GOLD Assessment — Three Components

4. **(a) Severity of airflow limitation (GOLD grades 1–4)** — applied only once FEV_1/FVC <0.7:

GOLD grade	FEV_1 % predicted	Severity
GOLD 1	≥80%	Mild
GOLD 2	50–79%	Moderate
GOLD 3	30–49%	Severe
GOLD 4	<30%	Very severe

5. **(b) Symptom burden** — measured by the **mMRC dyspnoea scale** (cut-off ≥2) or the **COPD Assessment Test, CAT** (cut-off ≥10). CAT has 8 items scored 0–5, total 0–40.
6. **(c) Exacerbation history in the past year** — the strongest single predictor of future exacerbations.

Group	Definition — GOLD 2023–2025	Definition — GOLD 2026 (revised)
A	0–1 moderate exacerbation, no hospitalisation; mMRC 0–1 or CAT <10	**No** moderate or severe exacerbation; mMRC 0–1 or CAT <10
B	0–1 moderate exacerbation, no hospitalisation; mMRC ≥2 or CAT ≥10	**No** moderate or severe exacerbation; mMRC ≥2 or CAT ≥10
E	≥2 moderate exacerbations **or** ≥1 leading to hospitalisation	**≥1 moderate or severe exacerbation** in the past year, whatever the symptom score

7. **Remember the history:** the older **ABCD** tool used both symptoms and exacerbations for all four groups; **GOLD 2023 merged C and D into a single group "E"** (E for *exacerbations*) because exacerbation risk drives treatment more than symptoms do. **GOLD 2026 has now lowered the entry threshold to Group E to a single moderate exacerbation.**
8. **Grading of exacerbation severity: mild** — short-acting bronchodilators only; **moderate** — bronchodilators plus antibiotics and/or oral corticosteroids; **severe** — requires emergency department visit or hospitalisation.

Treatment Options — Initial Pharmacological Therapy

9. **Group A:** a bronchodilator (short- or long-acting), chosen by symptom response. **Group B: LABA + LAMA** combination. **Group E: LABA + LAMA**; add **ICS (triple therapy) if blood eosinophils ≥300 cells/μL**.
10. **Never use ICS alone in COPD.** ICS are favoured when eosinophils are ≥300/μL, when there have been ≥2 exacerbations or a hospitalisation, or when asthma coexists; they are discouraged when there is **repeated pneumonia, eosinophils <100/μL, or a history of mycobacterial infection** — a point of particular importance in India, where past tuberculosis is common.

Follow-up Therapy — the Management Cycle

11. GOLD uses a **review → assess → adjust** cycle. **Before escalating, always re-check inhaler technique, adherence, device suitability, non-pharmacological measures and comorbidities.** GOLD 2026 clearly separates **initial** treatment (treatment-naive patient) from **follow-up** treatment (patient already on therapy).
12. **If dyspnoea persists:** escalate LABA or LAMA monotherapy to LABA+LAMA; if already on dual therapy, consider switching device or molecule and investigate other causes of breathlessness.
13. **If exacerbations persist:** escalate to **LABA+LAMA+ICS** when eosinophils are ≥100/μL. If exacerbations continue on triple therapy, add **roflumilast** (FEV_1 <50% with chronic bronchitis), **azithromycin** (preferably ex-smokers;

check QTc and hearing), or a **biologic**.

Non-Pharmacological and Interventional Options

14. **Core measures for every patient:** smoking cessation, vaccination, **pulmonary rehabilitation**, physical activity, self-management education and written action plans, and post-exacerbation follow-up. GOLD 2026 elevates these to **core disease-modifying strategies**, not optional extras.
15. **LTOT** for chronic severe resting hypoxaemia; **domiciliary NIV** for persistent hypercapnia after hospitalisation for exacerbation; **lung volume reduction surgery or endobronchial valves** for selected upper-lobe emphysema; **lung transplantation** in very selected patients (rarely appropriate in the elderly).
16. **Pulmonary rehabilitation in the elderly (Pathy's):** patients are eligible from **GOLD grade 2–4**, but enrolment should be driven by **symptoms and functional limitation, not FEV_1 alone**. It improves exercise tolerance, quality of life, dyspnoea, anxiety and depression, and reduces hospital days (10.4 vs 21 days in the year after rehabilitation) — but it does **not** improve survival, lung function or oxygen requirement. **Benefits are not age-dependent**, so age alone is never a barrier.

Latest Updates (2023–Present)

- **GOLD 2026** incorporates **330 new references and 14 new figures**; a new **Chapter 6 on emerging technology and AI**, and material moved to four appendices. AI-assisted alternatives to spirometry under investigation include respiratory sound and voice analysis, oscillometry, e-nose volatile organic compounds and chest CT-based algorithms.
- **The redefinition of Group E** (≥1 moderate exacerbation) is the single most examinable change of 2026 — driven by observational data showing that even one exacerbation before starting maintenance therapy predicts further events.
- **"Disease activity, stability and control"** is introduced as a formal therapeutic target, borrowing the concept from rheumatology — treat to a state of **low disease activity**.
- **Biologics:** Figure 3.11 summarises the evidence — dupilumab and mepolizumab now have **Level A** evidence for exacerbation reduction; benralizumab failed.
- **A new physiology-based grading of exacerbation severity (the "Rome" proposal)** replaces the old treatment-based definition. Severity is now judged on **dyspnoea measured by visual analogue scale, respiratory rate, heart rate, SpO_2, CRP and arterial blood gases**, giving mild / moderate / severe categories that do not depend on where the patient was treated. The exacerbation chapter also stresses **structured discharge bundles and early follow-up**.
- **Ensifentrine**, a first-in-class inhaled **dual PDE3/PDE4 inhibitor** combining bronchodilator and anti-inflammatory action, was approved by the FDA in 2024 as maintenance therapy (ENHANCE-1 and ENHANCE-2) — a new pharmacological class that post-dates the textbook.
- **Vaping and e-cigarettes are explicitly not recommended** as cessation aids because of insufficient long-term safety data.

Current Practice Update

Item	Textbook position (Pathy's 6e, citing GOLD 2019)	GOLD 2026
Exacerbation severity	Graded by treatment given (bronchodilators only / plus steroids-antibiotics / hospitalisation)	**Rome criteria** — graded physiologically by dyspnoea VAS, respiratory rate, heart rate, SpO_2, CRP and blood gases
Assessment tool	Four-stage FEV_1 severity classification driving therapy	**ABE groups** drive therapy; FEV_1 grade used for prognosis and non-pharmacological decisions
Group E threshold	≥2 exacerbations or 1 hospitalisation (NICE: 2 in 12 months)	**≥1 moderate or severe exacerbation**
Initial therapy for symptomatic patient	Start with a single LAMA	**LABA + LAMA** dual bronchodilation preferred
Choosing ICS	Clinical phenotype (frequent exacerbator, asthma features)	**Blood eosinophil count**: favour ≥300/µL, avoid <100/µL or prior mycobacterial disease
Advanced therapy after triple	Roflumilast, theophylline, high-dose acetylcysteine	Plus **azithromycin and biologics (dupilumab, mepolizumab)**
Diagnosis strategy	Case detection by symptom-driven spirometry	Formal **case-finding algorithm**; explicit push for **earlier diagnosis** ("Think COPD")

Examiner's Tip

Draw **two tables** — the GOLD 1–4 spirometric grades and the ABE assessment box — and state clearly that **"grade determines prognosis, group determines pharmacotherapy."** The examiner is listening for the 2026 change: say aloud "*a single moderate exacerbation now places the patient in Group E*." Add the eosinophil thresholds (**≥300 favours ICS, <100 argues against**) and, for the Indian context, the caution against ICS where there is a history of tuberculosis. Close with the geriatric qualifier: "*In older adults the fixed 0.70 ratio over-diagnoses COPD; the lower limit of normal is preferred*."

3. Community-Acquired Pneumonia in the Elderly — Etiology, Clinical Features and Management 15 marks

High-Yield Points

Definition and Epidemiology

1. **Definition:** acute infection of the lung parenchyma acquired outside hospital, with new radiographic consolidation. Unlike bronchitis (which involves only the airways and has a benign course), pneumonia involves the **alveoli**.
2. **Burden:** pneumonia and influenza together are the **sixth leading cause of death in those over 65** and the leading infectious cause of death in the elderly. Incidence rises from **18 per 1000 at age 65–69 to 52 per 1000 over age 85**. Over **40% of elders with CAP require hospitalisation**; mortality is about **10%, rising to nearly 50%** where there is pre-existing cardiopulmonary disease. **Two out of three pneumonia hospitalisations are in patients over 70.**
3. **Predisposing factors (Pathy's Table 39.1):** ageing itself (declining cell-mediated immunity, reduced cough and mucociliary clearance, micro-aspiration), COPD, asthma, **sequelae of tuberculosis**, diabetes mellitus, heart failure, neurological disease, **swallowing disorders**, sedative and antipsychotic drugs, gastro-oesophageal reflux, protein-energy malnutrition, and enteral feeding or nasogastric tube. **Pneumonia is the commonest specific cause of death in Alzheimer's disease.**

Etiology

4. ***Streptococcus pneumoniae* is the single most important pathogen** — the commonest cause of CAP requiring admission, with a high mortality in the elderly.
5. Other bacteria: **Gram-negative bacilli** (after aspiration, hospitalisation or prior antibiotics), **Staphylococcus aureus**, **Haemophilus influenzae**, *Legionella pneumophila* (rarer but severe, high mortality), *Mycoplasma pneumoniae*, streptococci and Enterobacteriaceae. **Anaerobes are common in aspiration pneumonia**, and **multi-microbial pneumonia is common in the elderly**.
6. **Age shifts the spectrum:** in older patients *S. pneumoniae*, *H. influenzae* and respiratory viruses are commoner, while ***Legionella*, *Mycoplasma* and *Chlamydia* are less frequent** than in younger adults.
7. **Nursing-home residents:** aspiration pneumonia, Gram-negative enteric bacilli and multiple anaerobes predominate. In hospital-acquired pneumonia: *S. aureus*, *Pseudomonas aeruginosa* and resistant Gram-negatives.
8. **Viruses cause 10–30%** of community-acquired lung infection, often in institutional epidemics — influenza (even in vaccinated patients), **RSV**, parainfluenza, rhinovirus, adenovirus, and emerging coronaviruses (SARS 2002, MERS 2012, **SARS-CoV-2/COVID-19**, where case-fatality exceeds 30% in those over 75–80). Always consider ***M. tuberculosis*** where illness has evolved over weeks with weight loss.

Clinical Features — the Atypical Presentation

9. **The classical picture is rarely complete. Fever and cough are absent in 30–40% of cases.** Onset is often gradual with less marked signs, causing diagnostic delay.
10. **Respiratory symptoms:** cough (productive or dry), purulent sputum, dyspnoea, pleuritic chest pain, haemoptysis. **Non-respiratory symptoms:** fever, anorexia, chills and sweats.
11. **Geriatric presentations: delirium/acute confusion, falls, functional decline, incontinence, anorexia, tachypnoea alone, or unexplained decompensation of diabetes or heart failure**. **Tachypnoea is often the earliest and most reliable sign.** Hypothermia rather than fever indicates severe disease.
12. **Examination findings to record:** respiratory rate >30/min, heart rate >110/min, fever or hypothermia, bronchial breathing, crackles, dullness to percussion, and hypoxaemia on pulse oximetry.

Investigations

13. **Chest radiograph** is the gold standard for documenting parenchymal involvement; the radiographic pattern correlates poorly with the causative organism. A normal film may occur very early in pneumococcal pneumonia. **Practical strategy in the community:** the British Thoracic Society allows management without a film if pneumonia is likely and admission is not needed; otherwise repeat the film 2–3 days after starting treatment. **CT** is indicated in difficult cases and can identify very early pneumonia or pulmonary embolism.
14. **Point-of-care lung ultrasound** detects B-lines, consolidation ("pulmonary hepatisation"), effusion and pneumothorax, with excellent sensitivity and specificity compared with chest X-ray. It is particularly valuable in nursing homes and home care where imaging is hard to obtain — a technique Pathy's predicts will become a core geriatric skill.
15. **Blood tests:** neutrophil leucocytosis (may be **absent** in older patients with bacterial infection), raised CRP, electrolytes, urea, creatinine and glucose (dehydration, renal failure and hyperglycaemia are common). **Arterial**

blood gas if there is chronic respiratory failure, cyanosis, sweating or reduced consciousness. **BNP >300 pg/mL suggests cardiac rather than pulmonary dyspnoea.**

16. **Microbiology:** blood cultures and sputum examination in all hospitalised patients. **Pneumococcal and Legionella urinary antigen** only for severe pneumonia needing intensive care, or during an outbreak. **Do not send routine urine microscopy/culture** — asymptomatic bacteriuria is so common in the elderly that it misleads prescribing. Nasal swab for viral PCR during institutional epidemics.

Management

17. **Decide site of care.** Use **CURB-65** (Confusion, Urea >7 mmol/L, Respiratory rate ≥30, Blood pressure <90/60, age ≥65) or **PSI**; but Pathy's stresses that in the frail elderly the decision also depends on **severity signs and whether the patient can be cared for at home** (Figure 39.1). Note that **age ≥65 automatically scores one CURB-65 point**, so the score over-triages older patients — combine with clinical and social judgement.
18. **General measures:** oral or subcutaneous/intravenous hydration, oral nutritional supplements if malnourished, **oral care**, pressure-ulcer prevention, early mobilisation, antipyretics if fever >39.5°C or poorly tolerated, **non-pharmacological management of delirium**, and **subcutaneous heparin thromboprophylaxis** unless contraindicated.
19. **Supportive respiratory care:** nasal oxygen for dyspnoea or hypoxaemia (**with caution in hypercapnic chronic respiratory failure**); chest physiotherapy; inhaled beta-2 agonists and anticholinergics if bronchial obstruction is present; a short course of systemic corticosteroids for resistant bronchial obstruction; **emergency bronchoscopy** for large atelectasis from a mucus plug.
20. **Antibiotics (Pathy's Table 39.5, ATS/IDSA 2019) for a comorbid outpatient:** a **beta-lactam** (amoxicillin, amoxicillin-clavulanate, cefpodoxime or cefuroxime) **plus a macrolide or doxycycline**; or a **respiratory fluoroquinolone alone** (levofloxacin, moxifloxacin, gemifloxacin — moxifloxacin now restricted for adverse effects). **Nursing-home pneumonia** requires anaerobic cover: amoxicillin-clavulanate or ceftriaxone or a respiratory fluoroquinolone. **Severe or hospital-acquired pneumonia** requires broad-spectrum cover, guided by prior antibiotics in the last three months and risk factors for *Pseudomonas* (bronchiectasis).
21. **The 48-hour review is a critical step.** Response is expected within 2–4 days. If afebrile and stable, continue and switch intravenous to oral in the same class. **If not improving**, consider resistant organisms, virus or tuberculosis, empyema, a non-infectious lung disease, antibiotic adverse effects, or decompensating comorbidity — and re-image, seek urinary antigens, and take infectious disease advice.
22. **Duration and follow-up:** total course about **10 days** (5 days for azithromycin). Fever, leucocytosis and CRP settle within 2–5 days; cough persists longer. **A follow-up chest radiograph is not needed if recovery is clinically complete** — radiographic clearance can take **up to 12 weeks** in the elderly. If lung cancer is suspected, arrange CT and bronchoscopy.
23. **Prevention:** annual **influenza vaccination for everyone aged 65 and over** — it reduces influenza by about 50% and influenza mortality by about 70%; **adjuvanted or high-dose vaccine** is particularly valuable in institutionalised or chronically ill elders. Add **pneumococcal vaccination**, COVID-19 and RSV vaccination, smoking cessation, oral and dental hygiene, dysphagia screening and safe feeding practice, avoidance of sedatives and antipsychotics, treatment of reflux, and correction of malnutrition.

Latest Updates (2023–Present)

- **Pneumococcal vaccination age lowered.** ACIP (23 October 2024; MMWR January 2025) recommends a **single dose of pneumococcal conjugate vaccine for all PCV-naive adults aged ≥50 years**, replacing the previous age-65 threshold. Options are **PCV21 (Capvaxive, FDA-approved June 2024, covering eight serotypes absent from other vaccines), PCV20, or PCV15 followed by PPSV23**.
- **RSV vaccination established for older adults.** Routine RSV vaccine for all adults ≥75, and for those **50–74 years at increased risk** because of chronic heart, lung, kidney or liver disease, diabetes, frailty or residence in long-term care (ACIP expansions 2024–2025). Available products: RSVpreF (Abrysvo), RSVPreF3-adjuvanted (Arexvy) and mRNA-1345.
- **Corticosteroids in severe CAP:** the **CAPE COD trial (NEJM 2023)** showed that **intravenous hydrocortisone reduced 28-day mortality** in severe CAP requiring mechanical ventilation, high-flow oxygen or with a high pulmonary severity index — now incorporated into several society statements. Steroids are **not** advised in influenza pneumonia or where aspergillosis is suspected.
- **Diagnostics:** rapid multiplex respiratory PCR panels and **lung ultrasound** are increasingly first-line in frail and bedbound patients; procalcitonin may support antibiotic de-escalation but should not withhold antibiotics in a septic older patient.
- **India:** the ICMR and API continue to emphasise a **five-day minimum course with de-escalation** and antimicrobial stewardship given the very high burden of ESBL-producing Enterobacteriaceae; **melioidosis (*Burkholderia***

***pseudomallei*)** should be considered in diabetic patients from coastal and monsoon-affected regions presenting with severe community-acquired pneumonia.

Current Practice Update

Issue	Textbook position (Pathy's 6e)	Current evidence (2023–2026)
Pneumococcal vaccine	Recommended for older adults; PPSV23/PCV13 era	**Single PCV dose from age 50**; PCV21 or PCV20 preferred
RSV	Recognised as a cause of pneumonia; no vaccine available	**Three licensed RSV vaccines**; routine from age 75 and from 50 if at risk
Steroids in severe CAP	Reserved for resistant bronchial obstruction	**Hydrocortisone reduces mortality in severe CAP** (CAPE COD, NEJM 2023)
Duration of antibiotics	About 10 days	**5 days is sufficient** if afebrile 48 h and clinically stable, in most non-severe CAP
Healthcare-associated pneumonia (HCAP)	Nursing-home pneumonia treated as a distinct high-resistance category	The **HCAP category has been abandoned**; treat as CAP unless there are validated local risk factors for MRSA or *Pseudomonas*
Imaging	Chest X-ray, CT in difficult cases; ultrasound emerging	**Lung ultrasound now a mainstream point-of-care tool** in geriatric and nursing-home settings

Examiner's Tip

The mark-earning line for this question is the atypical presentation: "*Fever and cough are absent in 30–40% of elderly patients with pneumonia; delirium, falls, tachypnoea and functional decline may be the only presenting features*." Follow it with three numbers — **incidence 52 per 1000 over age 85**, **mortality 10% rising to 50% with cardiopulmonary disease**, and **radiographic clearance takes up to 12 weeks**. Mention the two things geriatricians do that others forget: **do not send a routine urine culture** (asymptomatic bacteriuria misleads), and **screen for dysphagia** because aspiration underlies much of this disease. End on prevention: influenza vaccine reduces influenza mortality by about 70%.

4. Causes, Diagnosis and Management of Pulmonary Embolism 15 marks

High-Yield Points

Definition and Epidemiology

1. **Definition:** obstruction of the pulmonary arterial tree, usually by thrombus embolised from the deep veins of the leg or pelvis; part of the spectrum of **venous thromboembolism (VTE)** together with DVT.
2. **Ageing is the dominant risk factor:** both the incidence of VTE **and** the haemorrhagic risk of its treatment rise with age. **Thirty-day mortality after VTE roughly trebles** in those aged ≥65 compared with the young (11% vs 4%), and at one year it is **31% vs 14%**.

Causes and Risk Factors — Virchow's Triad

3. **Venous stasis:** immobility, hospitalisation, stroke with hemiplegia, heart failure, long travel, obesity, varicose veins.
4. **Endothelial injury:** surgery (especially **hip and knee arthroplasty and hip fracture**), trauma, fracture of a lower limb, central venous catheters, previous DVT.
5. **Hypercoagulability: active malignancy** (the single most important acquired factor in the elderly), inflammatory illness, sepsis, nephrotic syndrome, oestrogen therapy, **antiphospholipid syndrome**, and inherited thrombophilias (factor V Leiden — the commonest; prothrombin 20210A; deficiencies of antithrombin, protein C and protein S). Ageing itself brings **raised fibrinogen, altered platelet function and frailty**, all independently associated with VTE.
6. **Testing for thrombophilia is rarely useful in the elderly** — levels of antithrombin, protein C and S fall during acute thrombosis and during anticoagulation, so the patient must be off treatment and thrombosis-free. First presentation in old age is usually provoked or cancer-associated rather than inherited.

Clinical Features

7. **Classic:** sudden pleuritic chest pain, dyspnoea, tachypnoea, tachycardia, haemoptysis, syncope; massive PE presents with hypotension, cardiogenic shock or cardiac arrest.
8. **In the elderly the presentation is blunted and non-specific** — isolated dyspnoea, unexplained tachypnoea, new atrial fibrillation, syncope or a fall, delirium, or simply unexplained deterioration. **Asymptomatic DVT is present on admission in a high proportion of older medical inpatients.** PE shares fever, cough, chest pain and dyspnoea with pneumonia; **clear lung fields on chest X-ray with marked hypoxaemia** should raise suspicion.

Diagnosis

9. **Step 1 — assess pre-test clinical probability** using the **Wells score** or the **revised Geneva score**. The revised Geneva score is **objective** (no subjective "PE most likely diagnosis" item), which makes it preferable in older patients:

Revised Geneva score (Pathy's Table 37.2)	Points
Age >65 years	1
Previous DVT or PE	3
Surgery under general anaesthesia or lower-limb fracture within 1 month	2
Active cancer (or cured <1 year)	2
Unilateral lower-limb pain	3
Haemoptysis	2
Pain on deep venous palpation and unilateral oedema	4
Heart rate 75–94 / ≥95 beats per minute	3 / 5
Clinical probability: Low 0–3 | Intermediate 4–10 | High >10	

10. **Step 2 — D-dimer, but only if probability is low or intermediate.** D-dimer rises physiologically with age, so the conventional 500 µg/L cut-off has poor specificity in the elderly. Use the **age-adjusted cut-off = age × 10 µg/L for patients over 50** (validated in the **ADJUST-PE study, JAMA 2014**) — this substantially increases the proportion of older patients in whom PE can be excluded without imaging, at no cost in safety.
11. **Step 3 — imaging. CT pulmonary angiography (CTPA)** is the investigation of choice. In renal impairment consider **V/Q or V/Q-SPECT scanning**, or a **compression ultrasound of the legs** first (a positive proximal DVT in a breathless patient justifies treatment without CTPA). Pathy's notes a randomised trial showing that sodium bicarbonate hydration before CTPA in chronic kidney disease was no better than no hydration.
12. **Diagnostic algorithm (Pathy's Figure 37.2):** haemodynamically stable patient → assess probability → if

low/intermediate, D-dimer (age-adjusted): negative excludes PE, positive proceeds to CTPA; if **high probability**, go **directly to CTPA**. In the unstable patient, **bedside echocardiography** showing right ventricular dilatation/dysfunction justifies immediate treatment.

13. **Supporting tests:** ECG (sinus tachycardia commonest; right axis deviation, right bundle branch block, T inversion V1–V4, the classic but uncommon **S1Q3T3**); chest X-ray usually normal or shows atelectasis, a raised hemidiaphragm or a pleural effusion; arterial blood gas shows hypoxaemia with hypocapnia; **troponin and NT-proBNP** for risk stratification.

Risk Stratification and Prognosis

14. **PESI and simplified PESI (sPESI)** are validated in the elderly and identify patients at low risk of short-term death who may be managed as outpatients. **Age is itself a PESI variable**, so few older patients score "low risk". The strongest predictors of long-term mortality are **systolic BP <100 mmHg, active cancer and age**; others are low physical activity, polypharmacy, diabetes, anaemia, hs-CRP >40 mg/L, troponin >14 pg/mL and D-dimer >3000 ng/mL at presentation.
15. **Causes of death in the first three months after VTE in the elderly: cancer 38%, PE 26%, infection 13%, bleeding 6%** — a striking set of figures worth quoting.

Management

16. **Immediate:** oxygen, analgesia, cautious fluids in right ventricular failure (over-filling worsens it), vasopressors (noradrenaline) if hypotensive, and **start anticoagulation during the diagnostic work-up** in any patient with intermediate or high clinical probability and no contraindication.
17. **Anticoagulation — DOACs are first choice** in the absence of cancer or severe renal impairment, mainly because of their **lower intracranial bleeding risk**. In patients aged ≥75, DOACs carry a **relative risk of major bleeding of 0.49** compared with vitamin K antagonists. Regimens (Pathy's Table 37.3):

Drug	Lead-in heparin	Initial and long-term (3–6 months)	Extended phase
Dabigatran	Yes, ≥5 days	150 mg twice daily	No change
Rivaroxaban	No	15 mg twice daily × 21 days, then 20 mg once daily	Consider 10 mg once daily
Apixaban	No	10 mg twice daily × 7 days, then 5 mg twice daily	Consider 2.5 mg twice daily
Edoxaban	Yes, ≥5 days	60 mg once daily (30 mg if CrCl 30–50 mL/min, weight ≤60 kg, or on strong P-gp inhibitors)	No change

18. **Reperfusion therapy: systemic thrombolysis** for high-risk (haemodynamically unstable) PE where bleeding risk is acceptable. **Catheter-directed thrombolysis, mechanical thrombectomy or surgical embolectomy** for those with contraindications to lysis or failed lysis. An **IVC filter** is reserved for absolute contraindications to anticoagulation or recurrent PE despite adequate treatment.
19. **Duration:** minimum **3 months** for all. A **provoked** PE with a major transient risk factor (surgery, fracture) stops at 3 months. **Unprovoked PE or a persistent risk factor** (cancer, thrombophilia) warrants **extended/indefinite anticoagulation** with periodic review of bleeding risk. Cancer-associated VTE: LMWH or an anti-Xa DOAC, continued while cancer is active.
20. **Bleeding risk in the elderly:** patients aged ≥65 have a **near two-fold** higher risk of major and clinically relevant non-major bleeding, and age ≥75 at least **doubles the risk of intracranial haemorrhage**. Independent predictors include **low physical activity and polypharmacy (>4 drugs)**. Pathy's states that **whether falls risk increases major bleeding remains controversial** — falls alone are not a reason to withhold anticoagulation. Existing recurrence scores are unhelpful in the old: **HERDOO2 automatically classes anyone ≥65 as high risk, DASH is not validated in the elderly, and the Vienna model lacks discriminative power.** Therefore **clinical judgement plus patient preference** must guide the decision.
21. **Prevention:** risk-assess every hospitalised older patient; use **LMWH thromboprophylaxis** unless contraindicated, mechanical prophylaxis (intermittent pneumatic compression) where bleeding risk is high, early mobilisation and adequate hydration.

Latest Updates (2023–Present)

- **The 2026 AHA/ACC Acute Pulmonary Embolism Guideline (Circulation and JACC, 19 February 2026)** — the first-ever joint de-novo American guideline on PE, written with ACCP, ACEP, CHEST, SCAI, SHM, SIR, SVM and SVN. It **retires the terms "massive" and "submassive"** and replaces binary risk classification with a **five-tier Acute PE**

Clinical Category system (A to E), plus a respiratory modifier.

- **The A–E categories in practice: Category A (subclinical, incidentally detected)** may be discharged directly from the emergency department; **Category B (symptomatic, low clinical severity)** can be discharged early; **Categories C to E** require hospitalisation. Advanced therapies are **reasonable in Category E1 (cardiopulmonary failure with shock)** and **may be considered in Categories D1–D2 (incipient cardiopulmonary failure)**; they are **not recommended in Categories A and B**.
- **Other Class 1 recommendations: PE Response Teams (PERTs)** are formally recommended; **LMWH is preferred over unfractionated heparin** when parenteral anticoagulation is required; **DOACs are preferred over vitamin K antagonists**; and anticoagulation should continue into the extended phase for a first PE without a major reversible risk factor.
- **HI-PEITHO (NEJM 2026, presented at ACC 2026)** — the first randomised evidence that **ultrasound-facilitated catheter-directed thrombolysis plus anticoagulation is superior to anticoagulation alone** in intermediate-high-risk PE, with comparable safety. **STORM-PE (Circulation 2026)** examined mechanical thrombectomy in the same population. **PEITHO-3** (reduced-dose alteplase) is ongoing.
- **Follow-up:** the 2026 guideline recommends review within the first week of discharge, a clinical visit at or before **3 months** to decide on anticoagulation duration, encouragement of **early ambulation**, and screening for **post-PE syndrome and chronic thromboembolic pulmonary hypertension (CTEPH)**.

Current Practice Update

Issue	Textbook position (Pathy's 6e, citing 2019 ESC)	Current evidence (2023–2026)
Severity classification	High / intermediate-high / intermediate-low / low risk	**Five-tier AHA/ACC Clinical Categories A–E**; "massive/submassive" abandoned
Parenteral anticoagulant	LMWH, UFH or fondaparinux as lead-in	**LMWH recommended over UFH** (Class 1)
Oral anticoagulant	DOACs first choice unless cancer or severe renal failure	Unchanged and strengthened — **DOACs over VKAs (Class 1)**
Catheter-directed therapy	Reserved for failed or contraindicated systemic lysis	**HI-PEITHO (2026) provides the first randomised support** in intermediate-high-risk PE; reasonable in Category E1
Service organisation	Not addressed	**PE Response Teams recommended (Class 1)**
D-dimer cut-off	Age-adjusted cut-off validated (ADJUST-PE)	Unchanged — remains the standard of care in the elderly

Examiner's Tip

Three things guarantee marks here. First, write the **age-adjusted D-dimer formula: age × 10 µg/L above 50 years (ADJUST-PE, JAMA 2014)** — it is the most geriatric-specific fact in the whole topic. Second, draw the diagnostic algorithm as a flow chart: *probability → D-dimer if low/intermediate → CTPA*, with **high probability going straight to CTPA**. Third, quote the causes of three-month mortality (**cancer 38%, PE 26%**) to show you understand that in the elderly the embolus is often a marker of another disease. Add the Pathy's line examiners like: "*Diagnosis and treatment are the same as in younger people, but the iatrogenic risk of treatment is higher.*" If asked about falls, say clearly that **a falls risk alone is not a contraindication to anticoagulation.**

5. Interstitial Lung Disease 7 marks

Pathy's does not carry a dedicated ILD chapter; ILD appears within the COPD differential diagnosis (Table 42.3), the muscle disorders chapter (anti-synthetase syndrome and dermatomyositis-associated ILD) and the pulmonary rehabilitation chapter. The points below combine that textbook material with current ATS/ERS/JRS/ALAT guidance, as required for a complete answer.

High-Yield Points

1. **Definition:** a heterogeneous group of diffuse parenchymal lung diseases in which inflammation and/or fibrosis affect the **alveolar interstitium**, producing a **restrictive** ventilatory defect with impaired gas transfer. ILD is predominantly a disease of older adults — **idiopathic pulmonary fibrosis (IPF) is typically diagnosed between 60 and 70 years**.
2. **Classification:** (a) **known cause** — occupational/environmental (asbestosis, silicosis, coal worker's pneumoconiosis, **hypersensitivity pneumonitis** from birds, mould, and in India **bagassosis** and **farmer's lung**), drugs (amiodarone, methotrexate, nitrofurantoin, bleomycin, immune checkpoint inhibitors) and radiation; (b) **connective tissue disease-associated** — rheumatoid arthritis, systemic sclerosis, Sjögren's, and dermatomyositis/polymyositis (the **anti-synthetase syndrome**: acute myopathy, Raynaud's, ILD, mechanic's hands and anti-Jo-1 antibody); (c) **idiopathic interstitial pneumonias** — IPF, NSIP, COP, RB-ILD, DIP; (d) **granulomatous** — sarcoidosis; (e) **others** — lymphangioleiomyomatosis, pulmonary Langerhans cell histiocytosis. In India, **post-tuberculous fibrosis** is an important mimic.
3. **Clinical features:** insidious **progressive exertional dyspnoea and a dry cough**; **fine end-inspiratory "Velcro" crackles** at the bases; **digital clubbing** (about half of IPF cases); later cyanosis and signs of **cor pulmonale**. Search deliberately for extrapulmonary clues — arthritis, sclerodactyly, Raynaud's phenomenon, Gottron's papules, heliotrope rash, dry eyes and mouth.
4. **Distinguishing ILD from COPD and heart failure (Pathy's Table 42.3):** ILD gives a **dry cough with fine basal crackles**, a **restrictive spirometry pattern** and **impaired diffusing capacity (TLCO)**, with B-line artefacts on chest ultrasound; COPD gives obstruction with hyperinflation; kyphoscoliosis gives restriction with a **normal** diffusing capacity. Always ask about **industrial exposure and drug toxicity**.
5. **Investigations: HRCT chest is central** — a **usual interstitial pneumonia (UIP) pattern** (basal and subpleural reticulation with **honeycombing** and traction bronchiectasis, minimal ground glass) in the right clinical setting confirms IPF **without biopsy**. Pulmonary function shows **reduced FVC, reduced TLC, preserved or raised FEV_1/FVC and reduced TLCO**. Add an autoimmune panel (ANA, RF, anti-CCP, myositis panel), a hypersensitivity pneumonitis exposure history and precipitins, six-minute walk test with oximetry, and echocardiography for pulmonary hypertension. Bronchoalveolar lavage and surgical or cryobiopsy are reserved for uncertain cases — and are often inappropriate in the frail elderly.
6. **The multidisciplinary discussion (MDD)** between clinician, radiologist and pathologist is the recognised diagnostic gold standard for ILD.
7. **Management — general:** remove the causative exposure or drug; **smoking cessation**; treat gastro-oesophageal reflux and obstructive sleep apnoea; **vaccination**; supplemental oxygen for resting or exertional hypoxaemia; **pulmonary rehabilitation** — Pathy's cites Dowman et al. (Thorax 2017) showing clinically important improvements in six-minute walk distance, symptoms and quality of life after exercise training in ILD; treat cough and breathlessness palliatively (opioids); consider transplantation only in highly selected younger patients.
8. **Management — disease-specific: IPF is treated with antifibrotics, not steroids** — **pirfenidone** or **nintedanib** slow the rate of FVC decline. **Immunosuppression is harmful in IPF (PANTHER-IPF)** but is the mainstay in connective tissue disease-ILD, hypersensitivity pneumonitis and sarcoidosis (corticosteroids with mycophenolate, azathioprine or rituximab). **Nintedanib is licensed for systemic sclerosis-ILD (SENSCIS) and for progressive fibrosing ILD (INBUILD)**.
9. **Prognosis:** IPF is the most aggressive form — **median survival about 3–5 years from diagnosis**, worse than many cancers. **Acute exacerbations** of IPF carry very high mortality. Death is usually from progressive respiratory failure, acute exacerbation, pulmonary hypertension or lung cancer.
10. **Geriatric considerations:** comorbidity is the rule (emphysema in the **combined pulmonary fibrosis and emphysema (CPFE)** syndrome, coronary disease, pulmonary hypertension, reflux, lung cancer); antifibrotic drugs cause diarrhoea, nausea, weight loss and deranged liver function, all poorly tolerated in frail patients; and **early advance care planning and palliative care referral** are essential given the prognosis.

Latest Updates and Current Practice (2023–Present)

- **Nerandomilast (Jascayd)** — a preferential **phosphodiesterase-4B inhibitor** with antifibrotic and immunomodulatory effects — was **FDA-approved for IPF in October 2025** (the first new IPF drug in over a decade) and **for progressive pulmonary fibrosis (PPF) in December 2025**, on the strength of the **FIBRONEER-IPF and**

FIBRONEER-ILD trials (NEJM 2025). In FIBRONEER-ILD the mean FVC decline at 52 weeks was **−86 mL (18 mg) and −69 mL (9 mg) versus −152 mL with placebo**; it worked both as monotherapy and added to nintedanib or pirfenidone, with better tolerability (mild diarrhoea) than nintedanib.

- **"Progressive pulmonary fibrosis" (PPF)** has replaced the older term "progressive fibrosing ILD" and is now a formal, treatable entity defined by worsening symptoms, physiology or radiology despite management of the underlying ILD — the 2022 ATS/ERS/JRS/ALAT criteria remain the standard.
- **Current Practice Update — three points:** (1) The textbook treats ILD only as a differential diagnosis of COPD; current practice recognises it as a **treatable, antifibrotic-responsive disease** in its own right. (2) Where Pathy's offers only pulmonary rehabilitation and supportive care, there are now **three licensed antifibrotics** — pirfenidone, nintedanib and nerandomilast. (3) **Immunosuppression, once used empirically for "pulmonary fibrosis", is now contraindicated in IPF** and reserved for autoimmune and inflammatory ILD.

> **Examiner's Tip**
>
> Structure the answer as **classification → UIP pattern on HRCT → restrictive spirometry with low TLCO → antifibrotics for IPF, immunosuppression for autoimmune ILD**. The single sentence that separates a good answer from an average one is: "*Immunosuppression improves connective tissue disease-associated ILD but is harmful in IPF, where antifibrotics are the treatment of choice*." Quote **median survival 3–5 years in IPF** and mention **nerandomilast (2025)** to show currency. For the Indian angle, add hypersensitivity pneumonitis from bird exposure and bagassosis, and always exclude **post-tuberculous fibrosis**.

6. Respiratory Failure — Types, Causes and Management 15 marks

Pathy's addresses respiratory failure within the chapters on ageing of the respiratory system, COPD and pneumonia rather than as a stand-alone chapter; the answer below builds on that material and standard ERS/ATS and ESICM guidance.

High-Yield Points

Definition and Types

1. **Definition:** failure of the respiratory system to maintain adequate gas exchange, defined on arterial blood gas as **PaO_2 <60 mmHg (8 kPa)** breathing room air, with or without **$PaCO_2$ >50 mmHg (6.7 kPa)**.

Type	Blood gas	Mechanism	Typical causes
Type 1 (hypoxaemic)	↓PaO_2, normal or ↓$PaCO_2$; **widened A-a gradient**	**V/Q mismatch** and shunt; diffusion impairment	Pneumonia, pulmonary oedema, ARDS, pulmonary embolism, ILD, pneumothorax, COVID-19
Type 2 (hypercapnic / ventilatory)	↓PaO_2 with ↑**$PaCO_2$**; A–a gradient normal if purely hypoventilation	**Alveolar hypoventilation** — won't breathe, can't breathe, or increased dead space	COPD exacerbation, opioids and sedatives, obesity hypoventilation, neuromuscular disease, kyphoscoliosis, severe asthma (late)
Type 3 (perioperative)	Mixed	Atelectasis from reduced FRC and abdominal splinting after surgery	Post-operative, especially upper abdominal and thoracic surgery in the elderly
Type 4 (shock)	Mixed	Respiratory muscle **hypoperfusion**; ventilation used to redistribute blood flow	Cardiogenic, septic or hypovolaemic shock

3. **Acute vs chronic vs acute-on-chronic. Acute** develops over minutes to hours with an **uncompensated respiratory acidosis (low pH, normal bicarbonate)**. **Chronic** develops over days to years with **renal compensation — raised bicarbonate and near-normal pH**. **Acute-on-chronic** shows a raised bicarbonate with an acidotic pH and is the commonest pattern seen in an elderly COPD patient in the emergency room.

Why the Elderly Are Predisposed

4. **Structural:** loss of elastic recoil, alveolar enlargement, air trapping and hyperinflation; kyphosis and rib-cage stiffening reduce chest wall compliance and total lung capacity; loss of diaphragm curvature and respiratory muscle mass.
5. **Physiological:** higher respiratory rate with lower tidal volume → increased work of breathing → reduced exercise tolerance; closing volume encroaches on tidal breathing, worsening V/Q mismatch; **blunted ventilatory response to hypoxia and hypercapnia** means the older patient **decompensates suddenly and without warning**.
6. **Clinical:** lung "inflammageing", impaired cough and mucociliary clearance, sarcopenia of respiratory muscles, malnutrition, polypharmacy (sedatives, opioids, antipsychotics) and multimorbidity.

Clinical Features

7. **Hypoxaemia:** dyspnoea, tachypnoea, tachycardia, restlessness, confusion, **central cyanosis** (appears only when reduced haemoglobin >5 g/dL, so it is unreliable in anaemia), arrhythmias, and in chronic hypoxaemia — polycythaemia, pulmonary hypertension and cor pulmonale.
8. **Hypercapnia:** headache (worse in the morning), warm peripheries with a **bounding pulse**, **flapping tremor (asterixis)**, papilloedema, drowsiness progressing to **CO_2 narcosis** and coma. In the elderly, **delirium may be the only sign**.
9. **Signs of exhaustion demanding urgent escalation:** inability to speak in sentences, use of accessory muscles, paradoxical abdominal movement, a **falling** respiratory rate with worsening gases, and a deteriorating conscious level.

Investigations

10. **Arterial blood gas is mandatory** — pulse oximetry cannot detect hypercapnia and is unreliable in poor perfusion, anaemia and darker skin pigmentation. Calculate the **alveolar–arterial gradient** to separate hypoventilation (normal gradient) from V/Q mismatch or shunt (widened gradient).
11. Add chest radiograph, ECG, full blood count, urea and electrolytes, blood glucose, CRP, cultures, and where relevant **D-dimer or CTPA**, **BNP**, **echocardiography**, spirometry, sleep study, and drug levels or toxicology.

Management — General Principles

12. **Treat the underlying cause** — antibiotics for pneumonia, bronchodilators and steroids for COPD, diuretics for pulmonary oedema, anticoagulation for PE, naloxone for opioid toxicity, drainage of pneumothorax.
13. **Oxygen therapy is a drug and must be prescribed with a target range.** Target **SpO_2 94–98%** in most acutely ill patients, but **88–92% in those at risk of hypercapnic respiratory failure** (COPD, obesity hypoventilation, kyphoscoliosis, neuromuscular disease). Pathy's warns of "a small but definite risk in giving too much oxygen to a patient with long-standing CO_2 retention", causing depressed hypoxic drive, increased coronary and cerebral arterial resistance, and worsened acidosis. Delivery: nasal prongs 1–6 L/min, simple face mask 6–10 L/min, **Venturi mask for controlled FiO_2**, non-rebreather reservoir mask at ≥15 L/min in emergencies.
14. **Non-invasive ventilation (NIV/BiPAP) is the treatment of choice for acute hypercapnic respiratory failure.** Start when **pH <7.35 with $PaCO_2$ >45 mmHg despite maximal medical therapy**. It reduces mortality, need for intubation and length of stay, and is strongly evidence-based in **COPD exacerbation** and **cardiogenic pulmonary oedema** (where CPAP is equally effective). Typical settings: IPAP 10–16 cmH_2O, EPAP 4–6 cmH_2O, titrated by gases at 1 and 4 hours. **Contraindications:** respiratory arrest, facial trauma or surgery, vomiting, inability to protect the airway, haemodynamic instability, undrained pneumothorax, agitation or a low GCS.
15. **High-flow nasal oxygen (HFNO)** delivers heated humidified gas up to **60 L/min** with an FiO_2 of 0.21–1.0. It washes out nasopharyngeal dead space, generates a small PEEP effect, improves secretion clearance and is far better tolerated than a mask. The **ERS 2022 guideline** suggests HFNO over conventional oxygen and over NIV in **hypoxaemic** ARF, HFNO over conventional oxygen during breaks from NIV, and — importantly — **NIV should be tried first in COPD with hypercapnic ARF**. Pathy's already notes HFNO as "safe and effective in exacerbated COPD patients with both normocapnic and hypercapnic insufficiency", and specifically valuable **where there is a do-not-intubate order or where intermediate care is needed outside the ICU**.
16. **Invasive mechanical ventilation** when non-invasive support fails or is contraindicated: for ARDS use **lung-protective ventilation — tidal volume 6 mL/kg predicted body weight, plateau pressure <30 cmH_2O, appropriate PEEP**, and **prone positioning** for severe ARDS. Permissive hypercapnia is acceptable provided pH stays above about 7.20.
17. **Adjuncts:** chest physiotherapy and secretion clearance, cautious fluid balance (restrictive in ARDS without shock), nutrition, VTE prophylaxis, treatment of anaemia, avoidance of sedatives, delirium prevention and **early mobilisation** to prevent ICU-acquired weakness.
18. **Chronic respiratory failure:** long-term oxygen therapy for chronic hypoxaemia; **domiciliary NIV** for persistent daytime hypercapnia after a COPD admission, obesity hypoventilation syndrome, kyphoscoliosis and neuromuscular disease; pulmonary rehabilitation and nutritional support.

Ethical and Geriatric Dimension

19. **Decide the ceiling of care early and explicitly.** Base it on **premorbid functional status, frailty (Clinical Frailty Scale), cognition, comorbidity and the patient's own wishes** — never on chronological age alone. Document whether NIV is being used as a **bridge to recovery** or as the **ceiling of treatment**, and record any advance directive.
20. **Prognosis and palliation:** outcomes in the elderly are driven by the underlying diagnosis and premorbid function rather than age. Where reversal is not achievable, provide **opioids and benzodiazepines for refractory breathlessness**, a fan to the face, positioning, and early palliative care involvement.

Latest Updates (2023–Present)

- **SOHO trial (NEJM, March 2026)** — the largest randomised comparison to date (1116 patients with PaO_2/FiO_2 ≤200, respiratory rate >25 and pulmonary infiltrates) found **identical 28-day mortality of 14.6% with high-flow nasal oxygen and with standard oxygen**. This tempers the enthusiasm generated by FLORALI (2015) and has prompted a re-evaluation of HFNO as an automatic first-line therapy: it remains valuable for comfort and for reducing intubation in selected patients, but a mortality benefit is not established.
- **ESICM 2023 ARDS guideline** retained the recommendation that patients with acute hypoxaemic respiratory failure **not** due to cardiogenic pulmonary oedema or COPD exacerbation should receive **HFNO in preference to conventional oxygen therapy**.
- **Oxygen targets:** current consensus is to avoid both hypoxaemia and hyperoxia — maintain **SpO_2 above 92% and below 98%** in acute hypoxaemic failure, recognising that oximeter error ranges from −3% to +1% against true arterial saturation and is greater in darker skin pigmentation, anaemia and poor perfusion.
- **Awake prone positioning**, established during COVID-19, is now used selectively in non-intubated hypoxaemic patients, though tolerance is limited in frail and cognitively impaired older adults.
- **ARDS definition:** the **new global definition (2023)** permits diagnosis using **SpO_2/FiO_2 ≤315** and includes patients on HFNO ≥30 L/min, extending the diagnosis to resource-limited settings where arterial gases and

ventilators are scarce — directly relevant to Indian practice.

Current Practice Update

Issue	Textbook position (Pathy's 6e)	Current evidence (2023–2026)
Oxygen target in COPD	Target saturations of about 88–92%	Unchanged and firmly established
NIV threshold	Consider if **pH <7.35** despite maximal therapy	Unchanged; NIV remains first-line in hypercapnic ARF and is tried **before** HFNO in COPD
High-flow nasal oxygen	Noted as safe and effective; useful with do-not-intubate orders	Preferred over conventional oxygen in **hypoxaemic** ARF, but **SOHO (2026) found no mortality benefit**
ARDS diagnosis	Berlin definition (PaO_2/FiO_2, requires arterial gas and PEEP)	**New global definition 2023** allows SpO_2/FiO_2 ≤315 and HFNO-supported patients
Decision-making	Full functional and social assessment before hospital-at-home	Formal **frailty scoring** and advance care planning now embedded in escalation decisions

> **Examiner's Tip**
>
> Open with the numerical definition (**PaO_2 <60 mmHg ± $PaCO_2$ >50 mmHg**) and immediately draw the type 1/type 2 table — that table alone carries several marks. Show you can interpret gases: "*A raised bicarbonate with an acidotic pH indicates acute-on-chronic failure*." Quote the two thresholds examiners listen for: **oxygen target 88–92% in the CO_2 retainer** and **NIV when pH <7.35**. Then add the geriatric distinction: "*The ageing lung has a blunted ventilatory response to hypoxia and hypercapnia, so the older patient decompensates abruptly and delirium may be the only sign of CO_2 retention*." Finish with the ceiling-of-care sentence — based on frailty and patient wishes, never on age.

7. Long-Term Oxygen Therapy 7 marks

High-Yield Points

1. **Definition and purpose:** LTOT is the provision of supplemental oxygen at home for **at least 15 hours per day** to patients with chronic severe resting hypoxaemia. Its aim is to **prolong survival** by reversing hypoxic pulmonary vasoconstriction and preventing cor pulmonale, not merely to relieve breathlessness.
2. **Evidence base:** Pathy's cites the **Nocturnal Oxygen Therapy Trial (NOTT, Ann Intern Med 1980)** — LTOT for ≥15 hours daily **improved survival** in severe COPD with persistent hypoxaemia, and also improved polycythaemia and neuropsychiatric function. The companion British MRC trial showed the same. **Benefit is dose-dependent: the more hours per day, the greater the survival gain.**
3. **Indications (Pathy's criteria), assessed during a period of clinical stability in a current non-smoker:**
   - **PaO_2 <7.3 kPa (55 mmHg)**, or
   - **PaO_2 7.3–8.0 kPa (55–60 mmHg) with** evidence of **secondary polycythaemia, pulmonary hypertension, peripheral oedema or nocturnal hypoxaemia**.

   Referral should also be considered in **severe COPD and in anyone with signs of cor pulmonale**.
4. **Screening in practice:** a simple pulse oximetry screen — refer for arterial blood gas assessment if **SpO_2 <93%**, or **<95% with possible cor pulmonale**. **Two arterial blood gases at least three weeks apart**, taken when the patient has been stable for 8 weeks after any exacerbation, are needed before committing to LTOT.
5. **Prerequisites and safety:** the patient **must have stopped smoking** (fire risk and loss of benefit); optimise all medical therapy first; assess for the fire hazard of open flames, cooking and heaters. In Indian homes this includes explicit counselling about kerosene stoves, agarbatti, oil lamps and beedi smoking by family members.
6. **Delivery systems:** the **oxygen concentrator** is the standard home device (economical for continuous use, needs mains electricity — an important limitation where power supply is intermittent); **compressed gas cylinders** serve as back-up and for ambulatory use; **liquid oxygen** allows longer portable use but is expensive. Delivery is usually by nasal cannula at **1–4 L/min**, titrated to achieve **PaO_2 >8 kPa (60 mmHg)** or **SpO_2 ≥90%** at rest, with the flow rate typically increased by 1 L/min during sleep and exertion.
7. **Other forms of home oxygen (distinguish these clearly): ambulatory oxygen** for exercise desaturation in patients already on LTOT or who desaturate on walking; **short-burst oxygen** for episodic breathlessness — Pathy's notes it "may improve symptoms of breathlessness if exercise desaturation is noted", but it has little evidence and is not a substitute for LTOT; and **nocturnal oxygen** for isolated sleep hypoxaemia.
8. **Outcomes and prognosis:** approximately **50% of patients receiving LTOT survive three years**. Those with a **low BMI and multiple comorbidities die earlier**. In older adults with COPD the main predictors of mortality are **disability, low BMI, lung function and the use of LTOT** — that is, the need for LTOT is itself a marker of poor prognosis.
9. **Geriatric-specific issues (a Pathy's strength — use these to score):**
   - **Falls risk** from extra machinery and long lengths of tubing; ask an engineer to fit switching mechanisms diverting supply to individual rooms, so tubing does not trail across the floor.
   - **Cognitive impairment and poor manual dexterity** compromise safe use; this area is poorly studied.
   - **Care-home logistics** — staff training and support are essential; residents using concentrators overnight may need portable cylinders with a conserver if the day room is far from the bedroom.
   - **Uncertain quality-of-life benefit:** some studies found **no improvement** in quality of life or activities of daily living; a later study found small improvements at six months. Randomised trials have **never been repeated in advanced old age**, or in patients with isolated nocturnal or exertional hypoxia — an honest limitation worth stating.
10. **Follow-up:** review at 3 months, then annually — check adherence (hours used), repeat blood gases, confirm continued non-smoking, reassess the prescription, and review whether LTOT remains appropriate as the patient approaches the end of life.

Latest Updates and Current Practice (2023–Present)

- **LOTT trial (NEJM 2016) and subsequent guidance:** in COPD with **moderate** resting desaturation (SpO_2 89–93%) or isolated exercise desaturation, oxygen gave **no benefit** in time to death or first hospitalisation. The **ATS 2020 home oxygen guideline** and GOLD accordingly **restrict LTOT to severe resting hypoxaemia only** and make no recommendation for isolated moderate or exertional desaturation — a direct challenge to the more liberal short-burst practice described in older texts.
- **GOLD 2026** reaffirms LTOT for severe chronic resting hypoxaemia and adds guidance on **adherence-enhancing**

interventions for pharmacological and oxygen therapy (Eur Respir Rev 2024), recognising that prescribed hours and used hours differ widely.

- **Current Practice Update — three points:** (1) Pathy's suggests short-burst oxygen for symptomatic exercise desaturation; **current guidelines do not support this**, since LOTT showed no benefit outside severe resting hypoxaemia. (2) Pathy's records uncertainty about quality-of-life benefit; this is now settled — **LTOT prolongs survival but does not reliably improve quality of life**, and this must be explained honestly when consenting an older patient. (3) **Domiciliary NIV, not oxygen alone**, is now the treatment for persistent daytime hypercapnia after a COPD admission.

> **Examiner's Tip**
>
> Write the two indication thresholds as a boxed pair — **PaO_2 <7.3 kPa**, or **7.3–8.0 kPa with polycythaemia, pulmonary hypertension, peripheral oedema or nocturnal hypoxaemia** — and state **"at least 15 hours per day"** and the **NOTT trial** by name. Marks are also available for the three preconditions: **clinical stability, two gases three weeks apart, and confirmed smoking cessation**. Finish with a geriatric point no one else will write: "*Long tubing and extra machinery in the home create a falls hazard; a room-switching supply and staff training in care homes are part of a safe LTOT prescription.*"

8. Metered Dose Inhalers 7 marks

High-Yield Points

1. **Definition and principle:** a pressurised metered dose inhaler (pMDI) is a hand-held device that delivers a fixed, reproducible dose of drug suspended or dissolved in a **hydrofluoroalkane (HFA) propellant** as an aerosol. Optimal **lung deposition requires particles of 1–5 µm**; larger particles impact in the oropharynx, smaller particles are exhaled.
2. **Why inhaled delivery is preferred:** drug is delivered directly to the airway, giving a rapid onset at a much lower dose with fewer systemic adverse effects — important in the elderly, where systemic beta-agonist effects (tremor, tachyarrhythmia, hypokalaemia) and anticholinergic effects are poorly tolerated. GOLD states plainly that **inhaled agents are preferred over oral agents**.
3. **Correct technique — eight steps to write out:** (1) remove cap and inspect; (2) **shake well**; (3) breathe out gently and fully, away from the device; (4) place the mouthpiece between the teeth and seal the lips; (5) **begin a slow, deep inhalation and actuate the canister simultaneously**; (6) continue inhaling slowly over 3–5 seconds; (7) **hold breath for about 10 seconds**; (8) wait **30–60 seconds** before a second dose. **Rinse the mouth after inhaled corticosteroids.**
4. **The critical step is hand–breath coordination** — actuation must coincide with the start of a slow deep breath. This is precisely the step that fails in older patients.
5. **Barriers in the elderly (Pathy's, high-yield):**
   - Most cognitively intact, physically able older patients **can** use an inhaler, but **learning and coordination difficulties are present in about 20%**.
   - **Cognitive impairment dramatically impairs inhaler use: an MMSE score below 24 is associated with a very high risk of failure.** Remarkably, the **abnormal pentagon-copying test alone** (the constructional component of the MMSE) identifies patients unable to use inhaler devices — a simple bedside screen. Board and Allen's **"simple drawing test"** makes the same point.
   - **Poor manual dexterity** — weak hand grip, tremor, arthritis, impaired coordination and poor vision.
   - **Impaired executive function** limits the acquisition of inhaling skills, particularly in hospitalised patients.
   - Inadequate **inspiratory flow and respiratory muscle strength**, and reduced awareness of bronchoconstriction.
6. **The spacer (valved holding chamber) solves most of these problems.** It removes the need for coordination, slows the aerosol, allows the propellant to evaporate so particle size falls into the respirable range, **increases lung deposition and reduces oropharyngeal deposition**. Pathy's states directly that if an MDI is prescribed, **a spacer will reduce oropharyngeal candidiasis and dysphonia**. Use tidal breathing (5 breaths per actuation) in the frail; actuate one dose at a time; wash the spacer in detergent and **air-dry without rinsing or wiping** to reduce electrostatic charge; replace every 6–12 months. Add a **face mask** for patients who cannot form a lip seal.
7. **Choosing between devices — the guiding principle is that "the perfect device does not exist"; select on an individual basis.**
   - **pMDI** — cheap, portable, dose counter available, but needs coordination; **add a spacer**.
   - **Breath-actuated MDI (Autohaler, Easi-Breathe)** — removes the coordination problem but still needs adequate inspiratory flow; cannot be used with a spacer.
   - **Dry powder inhaler (DPI)** — no coordination needed, but requires a **forceful, deep inspiration** generating adequate peak inspiratory flow, which many frail or severely obstructed older patients cannot achieve; also needs manual dexterity to load.
   - **Soft mist inhaler (Respimat)** — slow-moving fine mist, low inspiratory flow requirement, good lung deposition; useful in the elderly but the cartridge needs loading and priming.
   - **Nebuliser** — needs only tidal breathing; reserve for very advanced disease or where inhaler technique is irretrievably poor. Pathy's notes nebulised bronchodilators "may help patients with very advanced COPD... or where the inhaler technique is so poor that no other relief is possible".
8. **Adherence and device burden:** the number of devices independently reduces adherence, so **fixed-dose combination inhalers should always be considered** to reduce discontinuation and misuse. Cognitive impairment reduces adherence to both drug and non-drug therapy; the MMSE, the Mental Deterioration Battery or a secondary memory test such as **Rey's 15-word test** identify COPD patients at risk of poor compliance.
9. **Practical rules for the clinic: ask every patient to bring their inhalers to every visit and demonstrate their technique**; retrain the patient and the caregiver at each visit; check the technique **before** escalating therapy (a GOLD 2026 emphasis); use adapters fitted to the inhaler end to help weak grip; and check the dose counter. **Short-acting oral beta-2 agonists exist for the few who cannot use any inhaler, but their unfavourable therapeutic window severely limits their use.**

10. **Adverse effects of MDI therapy:** oropharyngeal candidiasis and dysphonia with inhaled corticosteroids (prevented by a spacer and mouth rinsing); local irritation and cough from the propellant; paradoxical bronchospasm; systemic effects — tremor, palpitations and hypokalaemia with beta-2 agonists, dry mouth, urinary retention and glaucoma with anticholinergics, and skin thinning, cataract and osteoporosis with high-dose inhaled corticosteroids.

Latest Updates and Current Practice (2023–Present)

- **Low-global-warming-potential propellants:** pMDIs currently use HFA-134a and HFA-227ea, which are potent greenhouse gases. Manufacturers are transitioning to **HFA-152a and HFO-1234ze**, which cut the carbon footprint by over 90%; the first reformulated salbutamol and combination inhalers began reaching markets in 2025–2026 with **unchanged dosing and technique**. Examiners increasingly expect a line on the **environmental footprint of inhalers** (a pMDI carries roughly 10–30 times the carbon footprint of a DPI), balanced against the caution that **a DPI is useless if the patient cannot generate the inspiratory flow**.
- **Smart inhalers and digital adherence monitoring** — electronic dose counters with Bluetooth-linked reminders improve adherence and technique; GOLD 2026's new chapter on emerging technology and AI covers digital adherence tools, and a 2024 network meta-analysis (Eur Respir Rev) evaluated adherence-enhancing interventions for both inhaled and oxygen therapy.
- **Current Practice Update — three points:** (1) Pathy's recommends a spacer chiefly to reduce candidiasis and dysphonia; current guidance positions the spacer as the **default for every older pMDI user**, because it also compensates for poor coordination. (2) **Device selection is now formally guided by measured peak inspiratory flow**, not by clinician preference — low inspiratory flow contraindicates a DPI. (3) GOLD 2026 requires that **inhaler technique, adherence and device suitability be re-checked before any escalation of therapy**, making the inhaler review a formal treatment step rather than an afterthought.

> **Examiner's Tip**
>
> This is an easy 7 marks if you structure it as **principle → technique steps → barriers in the elderly → spacer → device selection → adverse effects**. Write the eight technique steps as a numbered list — examiners tick them off. The two facts that mark you out as a geriatrician are: **"an MMSE below 24 predicts a very high risk of inhaler failure, and the abnormal pentagon-copying test alone identifies patients who cannot use a device"**, and **"the perfect device does not exist — choose individually, and always ask the patient to bring their inhaler and demonstrate."** Add the practical Indian point that a spacer can be improvised from a 500 mL plastic bottle where a commercial device is unaffordable.

9. Extrapulmonary Tuberculosis 15 marks

Pathy's covers tuberculosis in the elderly within the chapter on the epidemiology of respiratory infections rather than as a dedicated extrapulmonary chapter; that material is combined below with the Indian Index-TB guidelines, NTEP technical and operational guidance, and current WHO recommendations.

High-Yield Points

Definition and Epidemiology

1. **Definition:** tuberculosis involving organs other than the lung parenchyma. By NTEP convention, **tuberculous pleural effusion and intrathoracic lymphadenopathy without parenchymal involvement are classified as extrapulmonary**, while miliary TB is classified as pulmonary. If both pulmonary and extrapulmonary sites are involved, the case is **notified as pulmonary**.
2. **Burden:** extrapulmonary TB accounts for roughly **16–20% of TB cases globally** and about **20–25% of notified cases in India**, with a rising proportion. It is commoner in **children, people living with HIV, those on immunosuppressants or anti-TNF agents, patients with diabetes or chronic kidney disease, and the elderly**.
3. **Ageing and TB (Pathy's):** tuberculosis causes more deaths worldwide than any other single infectious disease. Even in low-prevalence countries, infection rates in those over 65 are almost **50% higher** than at ages 25–44. **Rates are higher in institutional and nursing-home settings** than in the general aged population, and outbreaks originate from both residents and staff. In India, **multigenerational households** mean TB is readily brought into the family and spread within it. Pathy's records that **the lungs are the site of about three-quarters of tuberculous infections in the elderly**, so roughly a quarter are extrapulmonary.

Pathogenesis

4. During **primary infection**, bacilli disseminate by the **lymphohaematogenous route** and seed distant organs, where they remain dormant. Extrapulmonary disease is usually **reactivation** of these dormant foci when cell-mediated immunity wanes — from **immunosenescence**, malnutrition, diabetes, steroids, HIV, malignancy, chronic kidney disease or anti-TNF therapy.
5. Spread may also be **contiguous** (pleura from a subpleural focus, spine from a paravertebral abscess), or by **ingestion** (intestinal TB from swallowed sputum or unpasteurised milk). **Extrapulmonary disease is characteristically paucibacillary** — the single fact that explains almost every diagnostic difficulty in this topic.
6. **Ageing alters the immune response:** the hallmark response to TB is cell-mediated immunity coordinating macrophages and monocytes with adaptive T-cell responses. Ageing brings **reduced phagocytosis, maladaptive or reduced cytokine release and reduced T-cell production**, which explains both the atypical presentation and the reduced positivity of immunological tests in older people.

Sites and Clinical Features

Site	Clinical features	Key diagnostic pointers
Lymph node (commonest, ~35–40%)	Painless, firm, **matted** cervical nodes; may become fluctuant and form a **collar-stud abscess** or discharging sinus (scrofula)	**FNAC and Xpert Ultra**; caseating granuloma on cytology/histology; excision biopsy if FNAC inconclusive
Pleural	Fever, pleuritic pain, dyspnoea; usually a **unilateral exudative effusion**	**Lymphocyte-predominant exudate; ADA >40 IU/L**; low smear yield; **pleural biopsy** gives the highest yield
Abdominal (peritoneal, intestinal, nodal)	Ascites, pain, subacute obstruction, doughy abdomen; **ileocaecal** disease mimics Crohn's and caecal carcinoma	**Ascitic ADA >39 IU/L**, SAAG <1.1; CT abdomen; colonoscopic or laparoscopic biopsy
Osteoarticular (spine = **Pott's disease**, ~50% of skeletal TB)	Back pain, **gibbus** deformity, cold abscess, paraparesis; commonly **thoracolumbar**	**MRI spine** — disc-space narrowing with anterior vertebral body destruction and paravertebral abscess; CT-guided biopsy
CNS (meningitis, tuberculoma)	Subacute headache, fever, vomiting, **cranial nerve palsies (VI, III, VII)**, altered sensorium, seizures, hydrocephalus, stroke from vasculitis	**CSF: lymphocytic pleocytosis, high protein, low glucose, ADA >10 IU/L**; **Xpert Ultra on CSF**; contrast MRI shows **basal exudates**
Genitourinary	**Sterile pyuria**, dysuria, haematuria, epididymo-orchitis, infertility, tubo-ovarian mass	Three early-morning urine samples for AFB culture and NAAT; IVU/CT showing calyceal distortion or ureteric stricture
Pericardial	Effusion, tamponade, later **constrictive pericarditis** with raised JVP, ascites and Kussmaul's sign	Echocardiography; **pericardial fluid ADA >40 IU/L**; pericardial biopsy
Miliary / disseminated	Prolonged fever, weight loss, hepatosplenomegaly, **choroid tubercles** on fundoscopy, pancytopenia	**Millet-seed nodules on chest X-ray/HRCT**; bone marrow and liver biopsy with culture; always do lumbar puncture

7. **Presentation in the elderly is blunted and non-specific (Pathy's, from Perez-Guzman's meta-analysis):** older patients are **less likely to report fever, sweating and haemoptysis**, **more likely to present with dyspnoea**, and have a **lower likelihood of radiographic cavitation**. The commonest presenting picture is **unexplained weight loss, anorexia, fatigue and functional decline** attributed to "ageing" or malignancy. **Essentials of Clinical Geriatrics states the useful rule: suspect tuberculosis when an older patient is inexplicably failing.**

Diagnosis

8. **The governing principle is "tissue is the issue".** Obtain a sample from the affected site — FNAC, biopsy, pleural, ascitic, pericardial or cerebrospinal fluid — and send it for **three things together: AFB smear, mycobacterial culture, and molecular testing**. NTEP emphasises histopathological confirmation wherever a tissue sample can be obtained.
9. **Molecular tests are the recommended initial diagnostic test. Xpert MTB/RIF Ultra** simultaneously detects *M. tuberculosis* complex and rifampicin resistance in 2 hours; it is **significantly more sensitive than Xpert MTB/RIF in paucibacillary extrapulmonary samples** (for example in tuberculous meningitis, sensitivity around **68% versus 37%**, with 100% specificity for both). India's indigenous **TrueNat MTB/MTB Plus and MTB-RIF Dx** is a chip-based, battery-operated NAAT that is deployed to peripheral NTEP laboratories. **Interpret an Ultra "trace" result in the light of the clinical setting**, as rifampicin resistance is reported as indeterminate on a trace call.
10. **Culture on liquid medium (MGIT)** remains the reference standard and the only way to obtain full drug susceptibility testing; it takes **2–6 weeks**. **Line probe assays** give rapid genotypic susceptibility for first- and second-line drugs.
11. **Histopathology: caseating epithelioid granulomas with Langhans giant cells** is the classic finding; note that granulomas alone are not specific — the differential includes sarcoidosis, fungal infection, brucellosis and Crohn's disease.
12. **Biochemical markers:** ADA is a useful **adjunct**, not a confirmatory test — **pleural >40 IU/L, ascitic >39 IU/L, pericardial >40 IU/L, CSF >10 IU/L**. Combine with lymphocyte predominance and a low glucose.
13. **Imaging is essential to define extent:** ultrasound (nodes, ascites, cold abscess), CT (abdomen, chest, sinus tracts), **MRI (spine, brain)**, echocardiography, and PET-CT in occult disease.
14. **Tests of infection, not disease:** Pathy's is explicit that the **Mantoux test suggests active tuberculosis only when it produces a large induration with blebs; simple positivity or negativity neither confirms nor excludes active disease**. **IGRA (QuantiFERON) has not been well validated for active tuberculosis in the very old**, many of whom had lifetime contact with the organism; positivity rates are lower in the elderly than in younger people with the same exposure. **Never use TST or IGRA to diagnose or exclude active extrapulmonary TB.**

15. **Mandatory ancillary work-up in every case:** HIV testing, blood glucose/HbA1c for diabetes, chest radiograph to look for concurrent pulmonary disease (which determines infectivity), baseline liver and renal function, weight, and **notification on the Nikshay portal** — notification is legally mandatory in India for both public and private practitioners.

Treatment

16. **Standard NTEP regimen for drug-sensitive extrapulmonary TB: 2 months HRZE (intensive phase) followed by 4 months HRE (continuation phase)**, given **daily as weight-band fixed-dose combinations** under the NTEP. India moved from thrice-weekly to **daily therapy** nationwide, and ethambutol is retained in the continuation phase.

Site	Total duration	Notes
Lymph node, pleural, abdominal, genitourinary, pericardial	**6 months** (2HRZE / 4HRE)	Extension only for slow or poor response, individualised
CNS TB / tuberculous meningitis	**12 months** (2HRZE / 10HRE)	Use drugs with good CSF penetration; add corticosteroids
Osteoarticular / spinal TB	**12 months** (2HRZE / 10HRE)	Surgery for instability, cord compression or failure of medical therapy
Disseminated / miliary with CNS involvement	**12 months**	Always examine CSF before deciding duration

17. **Adjunctive corticosteroids** — one of the three priority recommendations of the Indian **Index-TB guidelines** (Central TB Division and DGHS, published in the *Indian Journal of Medical Research*). Steroids are **recommended in tuberculous meningitis (dexamethasone, tapered over 6–8 weeks — reduces mortality) and in tuberculous pericarditis** (reduces constriction and the need for pericardiectomy). They are **not** routinely recommended for lymph node or pleural disease.
18. **Paradoxical reaction** — clinical or radiological worsening (enlarging nodes, new effusions, expanding tuberculomas) after an initial response, caused by immune reconstitution. It occurs in up to **25% of lymph node TB** and is **not** treatment failure: continue the same regimen, exclude drug resistance and non-adherence, and add corticosteroids or aspiration if symptoms are severe.
19. **Drug-resistant extrapulmonary TB:** managed under the **PMDT** arm of the NTEP. Since **2024, India has adopted the all-oral 6-month BPaLM regimen (bedaquiline, pretomanid, linezolid, moxifloxacin)** for eligible MDR/RR-TB and pre-XDR-TB. **BPaLM is not used for CNS, osteoarticular or disseminated TB**, where CSF and bone penetration are unproven — these need longer individualised regimens.
20. **Monitoring:** clinical response, weight gain and adherence at every visit; **baseline and monthly liver function** (older age, malnutrition, alcohol, hepatitis B or C and concurrent hepatotoxic drugs all raise the risk of drug-induced hepatitis); **monthly visual acuity and colour vision on ethambutol** (particularly hazardous in the elderly with cataract, glaucoma or renal impairment — dose must be adjusted for creatinine clearance); and **pyridoxine 10–25 mg daily with isoniazid** in all older, diabetic, malnourished, alcohol-dependent or renally impaired patients, to prevent peripheral neuropathy.
21. **Rifampicin drug interactions matter more in the elderly** because of polypharmacy — rifampicin is a potent CYP3A4 inducer and reduces the effect of **warfarin and DOACs, statins, sulfonylureas, corticosteroids, calcium channel blockers, antiepileptics, digoxin and antiretrovirals**. Review the entire drug chart at the start of therapy.
22. **Programme support in India:** treatment is free under the NTEP with **directly observed treatment and treatment adherence technologies (99DOTS, video-DOT, MERM boxes)**; case-based notification on **Nikshay**; **Ni-kshay Poshan Yojana** nutritional support by direct benefit transfer, **increased from ₹500 to ₹1,000 per month from November 2024**; and **Ni-kshay Mitra** community adoption under the Pradhan Mantri TB Mukt Bharat Abhiyan. Screen and offer **TB preventive treatment** to household contacts after excluding active disease.

Latest Updates (2023–Present)

- **WHO consolidated guidelines updated through 2025** now recommend **Xpert MTB/RIF Ultra as the initial diagnostic test for extrapulmonary specimens**, including CSF, lymph node aspirate and tissue, replacing smear microscopy as the first test. Sensitivity in extrapulmonary samples is highest in **CSF, bone and joint, and cutaneous specimens** and in **FNA tissue** (about 79%), and lowest in pleural fluid.
- **India's National Guidelines for Management of Drug-Resistant TB (27 March 2025)** and **National Guidance on Differentiated TB Care (March 2025)** are the current NTEP reference documents; the Central TB Division has also issued a dedicated **Training Module on Extrapulmonary Tuberculosis and an EPTB Standard Treatment Workflow**. Differentiated TB care formally stratifies patients by severity and risk — identifying the malnourished, elderly and comorbid for intensified support and inpatient care.

- **TB Mukt Bharat Abhiyan:** following the **100-Day TB Mukt Bharat Abhiyan (launched 7 December 2024)** across high-burden districts, a further **2026 campaign** continues intensified case-finding using portable handheld X-ray with AI-based reading, upfront NAAT, and nutritional support. India's national target for TB elimination remains **2025, five years ahead of the SDG target of 2030**.
- **Shorter regimens:** WHO conditionally recommends a **6-month intensified regimen for tuberculous meningitis** as an alternative to the standard 12 months in patients without drug resistance; **BPaLM** is now the preferred 6-month all-oral regimen for MDR/RR-TB, adopted by NTEP in 2024 — but **excluding CNS, osteoarticular and disseminated disease**.
- **Persisting quality gap:** a 2025 multicentric Indian cohort found that **76% of EPTB diagnoses were made clinically or radiologically without microbiological confirmation**, and only **29% had histopathological confirmation**, despite NTEP guidance — a direct reflection of barriers in tissue sampling and processing, and a legitimate point of criticism to raise in a viva.

Current Practice Update

Issue	Textbook position (Pathy's 6e)	Current evidence and NTEP practice (2023–2026)
Initial diagnostic test	**AFB smear and culture remain the gold standard**; no clear evidence that detection rates are lower in the elderly	Culture remains the reference standard, but **Xpert Ultra is the recommended initial test** on all extrapulmonary specimens
Value of IGRA	"Not extensively documented" for active TB in the very old	Position unchanged and now explicit: **IGRA and TST must not be used to diagnose or exclude active TB**
Drug-resistant disease	Not addressed for extrapulmonary sites	**All-oral 6-month BPaLM** for eligible MDR/RR-TB (NTEP 2024); **not for CNS, bone or disseminated disease**
Duration for CNS and bone TB	Not specified	**12 months** standard; WHO now offers a conditional **6-month intensified alternative for TB meningitis**
Adjunctive steroids	Not addressed	**Index-TB: recommended in TB meningitis and pericarditis**, not routinely elsewhere
Programme support	Not applicable	**Ni-kshay Poshan Yojana raised to ₹1,000/month (Nov 2024)**; mandatory Nikshay notification; differentiated TB care

> **Examiner's Tip**
>
> Anchor the answer on one sentence: **"Extrapulmonary TB is paucibacillary, so tissue is the issue — every accessible site must be sampled for smear, culture and Xpert Ultra together."** Then guarantee marks with numbers: **ADA cut-offs (pleural 40, ascitic 39, pericardial 40, CSF 10 IU/L)**, **durations (6 months, extended to 12 for CNS and bone)**, and the **two indications for steroids (meningitis and pericarditis)**. Add the Pathy's geriatric line — "*older patients report less fever, sweating and haemoptysis, present more often with dyspnoea, and cavitate less often*" — and the clinical rule from Essentials of Clinical Geriatrics: "*suspect tuberculosis when an older patient is inexplicably failing*." Finish with the programme detail (Nikshay notification, Ni-kshay Poshan Yojana ₹1,000/month) that shows you practise in India.

Extra Points to Remember — Common Sub-Questions

- **"Cold abscess"** — a tuberculous abscess without the classical signs of acute inflammation (no heat, no redness, minimal pain), typical of spinal and lymph node disease. It tracks along fascial planes and may present far from its origin (psoas abscess presenting in the groin).
- **Pott's spine — classic radiological triad:** disc-space narrowing, anterior vertebral body destruction with wedging (gibbus), and paravertebral soft-tissue shadow. **Relative preservation of the disc** early on distinguishes it from pyogenic spondylodiscitis; skip lesions are characteristic.
- **Tuberculous meningitis staging (British MRC): Stage I** — conscious, no neurological deficit; **Stage II** — conscious with deficit or GCS 11–14; **Stage III** — GCS <10 or dense deficit. **Outcome correlates directly with the stage at which treatment starts**, which is why empirical treatment must not await confirmation.
- **Lymph node TB vs lymphoma vs metastasis in an older patient:** TB nodes are matted with caseation on FNAC; lymphoma nodes are rubbery, discrete and require excision biopsy for architecture; metastatic nodes are hard and fixed with a primary elsewhere. **Never rely on FNAC alone to exclude lymphoma — excision biopsy is required.**
- **Drug-induced hepatitis:** stop all hepatotoxic drugs if ALT >3× upper limit with symptoms or >5× without; use a non-hepatotoxic bridging regimen (streptomycin/an injectable, ethambutol, levofloxacin) if treatment cannot be interrupted; reintroduce sequentially once ALT falls below twice normal. **Risk is higher in the elderly, the malnourished and in hepatitis B/C carriers** — a common combination in Indian practice.
- **Nursing homes and long-term care:** Pathy's and Essentials both call for **tuberculosis screening programmes in long-term care facilities**, since the older cohort lived through a high-incidence era, was rarely given isoniazid prophylaxis, and reactivates with malnutrition and comorbidity. Screen residents and staff, and remember that outbreak sources may be either.

Appendix — Rapid Revision Grid

Numbers and thresholds most likely to be asked. Read this last on the night before the examination.

Numerical thresholds

Parameter	Value
COPD diagnosis	Post-bronchodilator **FEV_1/FVC <0.70** (LLN preferred in the elderly)
GOLD spirometric grades 1–4	FEV_1 **≥80 / 50–79 / 30–49 / <30** % predicted
GOLD Group E (2026)	**≥1** moderate or severe exacerbation in the past year
Symptom cut-offs	**mMRC ≥2** or **CAT ≥10**
Eosinophil thresholds for ICS	Favour **≥300/µL**; avoid **<100/µL**
Normal vs susceptible-smoker FEV_1 decline	**25–30 mL/year** vs **>50 mL/year**
Reversibility indicating asthma	**>200 mL** rise in FEV_1; PEF variability **>20%**
NIV threshold in COPD exacerbation	**pH <7.35** despite maximal therapy
Oxygen target in CO_2 retainers	**SpO_2 88–92%** (94–98% in others)
Respiratory failure	**PaO_2 <60 mmHg (8 kPa) ± $PaCO_2$ >50 mmHg (6.7 kPa)**
LTOT indications	**PaO_2 <7.3 kPa**, or **7.3–8.0 kPa** with polycythaemia, pulmonary hypertension, oedema or nocturnal hypoxaemia
LTOT minimum daily use / survival	**≥15 hours/day**; about **50% survive 3 years**
Age-adjusted D-dimer (age >50)	**Age × 10 µg/L** (ADJUST-PE, JAMA 2014)
Revised Geneva probability bands	Low **0–3**, intermediate **4–10**, high **>10**
Minimum anticoagulation after PE	**3 months**; extended/indefinite if unprovoked or persistent risk factor
Pneumonia — absent fever/cough in elderly	**30–40%** of cases
Pneumonia — radiographic clearance	Up to **12 weeks** in older adults
ADA cut-offs in TB	Pleural **>40**, ascitic **>39**, pericardial **>40**, CSF **>10** IU/L
EPTB treatment duration	**6 months**; **12 months** for CNS and osteoarticular disease
MMSE and inhaler use	**<24** predicts a very high risk of inhaler failure
Optimal aerosol particle size	**1–5 µm**

Named trials, guidelines and eponyms

Name	What it established
NOTT (1980) and MRC	LTOT ≥15 h/day improves survival in hypoxaemic COPD
LOTT (2016)	No benefit of oxygen in moderate resting or isolated exercise desaturation
ADJUST-PE (2014)	Age-adjusted D-dimer safely excludes PE in older patients
PESI / sPESI	Validated short-term mortality prediction in acute PE, including the elderly
HI-PEITHO (NEJM 2026)	Catheter-directed thrombolysis superior to anticoagulation alone in intermediate-high-risk PE
2026 AHA/ACC PE Guideline	Five-tier Clinical Categories A–E; PERTs, LMWH over UFH, DOACs over VKA (all Class 1)
BOREAS / NOTUS	Dupilumab reduces COPD exacerbations by about 30% in eosinophilic disease
METREX / METREO / MATINEE	Mepolizumab reduces COPD exacerbations by about 18–21%
GOLD 2026	One moderate exacerbation defines Group E; disease activity as a target; biologics; AI chapter
FIBRONEER-IPF / FIBRONEER-ILD (2025)	Nerandomilast (PDE4B inhibitor) slows FVC decline in IPF and progressive pulmonary fibrosis
INBUILD / SENSCIS	Nintedanib in progressive fibrosing ILD and in systemic sclerosis-ILD
PANTHER-IPF	Triple immunosuppression is **harmful** in IPF
CAPE COD (NEJM 2023)	Hydrocortisone reduces mortality in severe community-acquired pneumonia
FLORALI (2015) / SOHO (NEJM 2026)	Early promise of HFNO not confirmed — SOHO found identical 28-day mortality (14.6%) vs standard oxygen
ERS 2022 HFNC guideline	HFNO over conventional oxygen and over NIV in hypoxaemic ARF; **try NIV first in hypercapnic COPD**
Index-TB guidelines (IJMR)	India's EPTB guidance — Xpert in diagnosis, adjunct steroids, treatment duration
BPaLM (NTEP 2024)	6-month all-oral MDR/RR-TB regimen; excludes CNS, bone and disseminated disease
ACIP October 2024 (MMWR Jan 2025)	Pneumococcal conjugate vaccine for all PCV-naive adults from **age 50**
BODE index	BMI, Obstruction, Dyspnoea, Exercise capacity — validated prognostic index in COPD
Langhans giant cell / caseating granuloma	Histological hallmark of tuberculosis
Pott's disease, gibbus, cold abscess	Spinal tuberculosis eponyms
S1Q3T3	Classic but uncommon ECG pattern in pulmonary embolism

Six lines to carry into every respiratory answer

1. The ageing lung has **reduced elastic recoil, a stiffer chest wall and a blunted ventilatory response to hypoxia and hypercapnia** — so decompensation is abrupt and warning signs are few.
2. **Presentation is atypical:** delirium, falls, functional decline and tachypnoea replace fever, cough and pleuritic pain.
3. **Spirometry is achievable in about 85% of older adults;** cognitive impairment is the commonest reason for an inadequate test.
4. **Treatment success is judged by symptoms, exercise tolerance and activities of daily living, not by change in FEV_1.**
5. **Check inhaler technique before escalating any therapy** — and check cognition before blaming the drug.
6. **Decide the ceiling of care by frailty, function and the patient's wishes — never by chronological age — and involve palliative care early.**

MD GERIATRIC MEDICINE • EXAM REVISION SERIES

Geriatric Nephrology & Acid–Base

Acute Kidney Injury • Drug-Induced Renal Impairment • Metabolic Acidosis

Q1. Causes and treatment of acute kidney injury in an elderly male *(15 marks)*
Q2. Drug-induced renal impairment in the elderly *(15 marks)*
Q3. Metabolic acidosis *(7 marks, with 15-mark expansion)*

Primary sources: Pathy's Principles and Practice of Geriatric Medicine, 6th ed. • Kane's Essentials of Clinical Geriatrics
Supplemented with guideline and trial evidence to August 2026 • Indian conventional units throughout

QUESTION 1

Causes and treatment of acute kidney injury in an elderly male

15 MARKS

High-Yield Points

A. Definition and Why the Old Kidney Is Vulnerable

1. **AKI** is an abrupt, potentially reversible fall in glomerular filtration over hours to days, with retention of nitrogenous waste. Standard criteria: rise in serum creatinine by **≥0.3 mg/dL in 48 hours**, or **≥1.5 times baseline within 7 days**, or urine output **<0.5 mL/kg/hour for 6 hours**.
2. **Tubular frailty** is the key ageing change — tubular cells in the elderly injure easily, progress readily to **acute tubular necrosis (ATN)**, and recover slowly. If the kidney has not recovered by about **3 months**, it is left as chronic kidney disease.
3. AKI is **commoner in older adults** because of frequent systemic illness (diabetes, myeloma), **polypharmacy**, and the renal ageing process itself. The cause is **usually multifactorial**, not single.
4. Ageing reduces urine concentrating power (falls about **30 mOsmol/kg per decade**), raises the thirst threshold, and lowers total body water to about **54%** of body weight (65% in the young) — so **dehydration develops very easily**.
5. **Renal dysautonomia** and loss of the autonomic vascular reflex mean the aged kidney is damaged during both hypotensive and hypertensive episodes.

B. Causes — Classification (write this as a table in the exam)

6. **Prerenal: dehydration is the main cause**; also haemorrhage and shock (cardiogenic and septic).
7. **Renal (intrinsic):** ATN from persisting prerenal causes or from **nephrotoxins** (aminoglycosides, contrast); rapidly progressive damage from primary or secondary glomerular disease; **acute interstitial nephritis**.
8. **Postrenal (obstructive):** stones, tumour, stricture and — the key one in a male — **prostatic hypertrophy**.
9. **Renal vasculitis** becomes commoner with age and is probably the commonest primary cause of renal failure in older adults; granulomatosis with polyangiitis and microscopic polyangiitis both rise with age. **Crescentic glomerulonephritis** peaks at **60–79 years** and typically presents as rapidly evolving AKI.
10. Vascular causes: renovascular atherosclerosis and ischaemic nephropathy; **reversible renal failure after an ACE inhibitor** in bilateral renal artery stenosis; **intrarenal atheroembolism**, from which the kidney rarely recovers.
11. **Urinary tract infection / pyelonephritis** is the commonest infection in the elderly, and its pathogenesis is strongly linked to obstructive uropathy; risk is raised further by urethral and vaginal atrophy, bed-rest puddling and catheterisation.
12. Add in a man specifically: **myeloma kidney**, tumour lysis syndrome, rhabdomyolysis after a long lie, and heat-related illness.

C. The Male-Specific Trap — Obstructive Uropathy

13. Prostatic hypertrophy occurs to some degree in nearly all ageing men; in a proportion it causes **slow outflow obstruction** with gradual loss of kidney function.
14. It is often recognised **too late, because the patient becomes polyuric rather than oliguric**. By the time obstruction is diagnosed, damage may be irreversible, and relief of obstruction restores function only partially.

D. Assessment

15. **Urinary indices behave differently in the elderly:** a high urinary sodium, fractional excretion of sodium or fractional excretion of urea **does not exclude prerenal AKI**, because the aged kidney normally loses sodium and urea.
16. **Serum creatinine underestimates the damage.** Renal function falls by about **50%** before creatinine rises, and reduced muscle mass plus net tubular creatinine reabsorption in the elderly delay the rise further.
17. Practical work-up: volume status and postural blood pressure, complete drug chart, bladder scan and ultrasound KUB for obstruction, urinalysis with microscopy, serum potassium and bicarbonate, creatine kinase, serum and urine protein electrophoresis, and ANCA / anti-GBM if a rapidly progressive picture is suspected.

E. Treatment

18. **Prerenal and postrenal causes matter most** — their early identification and treatment may **prevent the development of, or reverse, established AKI**. Catheterise or place a nephrostomy promptly; restore extracellular volume; treat sepsis; stop nephrotoxic drugs.
19. Correct complications: hyperkalaemia (the elderly are already predisposed because of low aldosterone and reduced tubular response, and worsen further on **NSAIDs, ACE inhibitors, beta blockers and potassium-sparing diuretics**), metabolic acidosis and fluid overload. Adjust every drug dose to the measured GFR.
20. Specific therapy: withdraw the culprit drug in interstitial nephritis, with steroids if needed; **steroids with cyclophosphamide** for vasculitis and crescentic glomerulonephritis, used as in younger adults but with closer attention to side effects.

21. **Dialysis:** temporary or permanent support may be needed. AKI is treated much the same in old and young, but older patients are **more vulnerable to dialysis-related complications — haemodynamic instability, bleeding and disequilibrium syndrome**.
22. **Prognosis:** **age by itself is not an important determinant of survival in AKI**. Failure to recover by about 3 months leaves chronic kidney disease.
23. **Prevention is paramount:** maintain an adequate extracellular volume and tailor drug dosing to GFR. Perioperatively, estimate GFR (CKD-EPI is regarded as most accurate, Cockcroft–Gault as most sensitive), stop nephrotoxic drugs before surgery, review diuretics against the planned procedure, titrate aminoglycoside dosing intervals to plasma levels, and optimise renal perfusion with haemodynamic monitoring.

Latest Updates (2023–Present)

- **KDIGO 2026 AKI/AKD Guideline (public review draft, March 2026)** — the first major revision since 2012, considering evidence to June 2025. It replaces "AKI alone" with an **AKI → AKD → CKD continuum**. **Acute kidney disease (AKD)** covers abnormality persisting up to 3 months, and AKD following AKI is separated from AKD without AKI.
- **Three-axis staging** is proposed: **C** (serum creatinine), **U** (urine output) and **B** (damage biomarker, reported as B− or B+). This lets a patient be "biomarker-positive" before creatinine moves.
- **Cystatin C is accepted as an alternative functional criterion** (≥1.5× baseline within 7 days) when creatinine is unreliable — directly relevant to **sarcopenic older adults**. **Kinetic eGFR** is suggested in hospitalised patients when a GFR estimate is needed under non-steady-state conditions.
- New content on **validated risk-prediction models, electronic AKI alerts, urine microscopy as part of evaluation, drug and nephrotoxin stewardship**, and structured **post-AKI follow-up** to reduce later CKD and cardiovascular events.
- Terminology has shifted from "contrast-induced nephropathy" to **contrast-associated AKI (CA-AKI)**, reflecting multifactorial causation rather than pure contrast toxicity; main risks are eGFR <60 mL/min/1.73 m², diabetes, volume depletion, high contrast volume and the intra-arterial route.
- **Sick-day medication guidance** (international modified-Delphi consensus, AJKD) is now standard for community-acquired AKI prevention: temporarily withhold volume-depleting and nephrotoxic drugs during acute dehydrating illness, and treat **restarting them as being as important as stopping them**.
- **Balanced crystalloids** are generally preferred to 0.9% saline for large-volume resuscitation, avoiding hyperchloraemic acidosis; low-dose dopamine, mannitol and routine loop diuretics remain of no benefit for AKI prevention or treatment.

Current Practice Update

Issue	Textbook position	Current evidence (2023–2026)
Framework	AKI described as a discrete event, with CKD if no recovery at ~3 months	Formal **AKI–AKD–CKD continuum**, with AKD as a named intermediate stage requiring its own follow-up
Diagnosis	Serum creatinine and urinary indices, with the caveat that they mislead in the elderly	Creatinine + urine output + **damage biomarkers**; **cystatin C** when muscle mass is low; kinetic eGFR in unstable patients
Detection	Clinical suspicion by the treating team	**Electronic AKI alerts** and validated risk-prediction models built into hospital systems
Contrast	Contrast listed as a nephrotoxin	**Contrast-associated AKI**; risk-stratified, lower contrast volumes; prophylaxis mainly isotonic fluid, not N-acetylcysteine
Prevention	Maintain volume, dose drugs to GFR	Same core, plus formal **nephrotoxin stewardship, pharmacist-led review and sick-day rules**
After recovery	Recovery or progression to CKD	**Structured post-AKI follow-up** is now a guideline recommendation, since AKI survivors carry excess CKD, cardiovascular and mortality risk

Exam One-Liners

- **Commonest cause of AKI in the elderly:** dehydration (prerenal).
- **Commonest intrinsic lesion:** acute tubular necrosis.
- **Commonest primary cause of renal failure in older adults:** renal vasculitis.
- **Crescentic glomerulonephritis** peaks at 60–79 years and presents as rapidly evolving AKI.
- **Obstruction in an elderly male presents with polyuria, not oliguria** — the single most examined trap.
- A raised FENa does **not** exclude prerenal AKI in the elderly.

- Age **per se** is not an important determinant of survival in AKI.
- Older patients are more prone to **intradialytic hypotension, bleeding and disequilibrium syndrome**.
- Non-recovery at 3 months = chronic kidney disease.

Extra Recall Points — Recurring Topic

Mnemonics

- **Causes — "3 P's": P**re-renal (dehydration), **P**arenchymal (ATN, AIN, vasculitis), **P**rostate (obstruction). In an elderly male, always finish with the prostate.
- **Reversible drivers — "DAMN": D**iuretics, **A**CE inhibitors/ARBs, **M**etformin, **N**SAIDs — the four to stop at the bedside on day one.
- **Bedside actions — "STOP-AKI": S**epsis treated, **T**oxins stopped, **O**ptimise perfusion and volume, **P**revent obstruction (catheterise/scan).

Numerical Anchors

- Creatinine rise **≥0.3 mg/dL in 48 h** or **≥1.5× baseline in 7 days**; urine output **<0.5 mL/kg/h × 6 h**.
- Concentrating capacity falls **~30 mOsmol/kg per decade**; minimum urine osmolality **92 vs 52 mOsmol/kg** (old vs young); maximum free-water clearance **5.9 vs 16.2 mL/min**.
- Total body water **54% vs 65%** of body weight.
- Renal function must fall **~50%** before creatinine rises.
- Non-recovery window: **3 months**. Dialysis in CKD when GFR **<10 mL/min**.

Examiner Traps

1. Writing "oliguria" for obstructive uropathy in a man — it is classically **polyuric**.
2. Using FENa >1% to call it ATN in an 80-year-old — the aged kidney loses sodium anyway.
3. Saying "old age makes AKI fatal" — age itself is not an important survival determinant.
4. Forgetting that the cause is **usually multifactorial**; listing only one.
5. Quoting a normal creatinine as reassurance in a sarcopenic patient.
6. Omitting **renal vasculitis**, which is the commonest primary cause of renal failure in this age group.

15-Second Answer Skeleton

Define AKI → ageing kidney is primed (tubular frailty, dehydration-prone, dysautonomia) → causes in three columns: **prerenal / renal / postrenal** → stress dehydration and prostate → assessment pitfalls (creatinine and FENa unreliable) → treatment: volume, sepsis, stop nephrotoxins, relieve obstruction, correct potassium and acidosis, dose to GFR, dialysis if needed → prognosis and prevention → KDIGO 2026 AKI/AKD continuum with biomarkers and cystatin C.

QUESTION 2

Drug-induced renal impairment in the elderly

15 MARKS

High-Yield Points

A. Why the Elderly Are Uniquely Susceptible

1. Renal function declines predictably with age, so drugs and their active metabolites that are cleared by the kidney are **eliminated more slowly, have longer half-lives, and accumulate to toxic steady-state concentrations**.
2. **Serum creatinine is an unreliable guide.** Muscle mass and daily endogenous creatinine production both fall, so creatinine may be normal when renal function is substantially reduced. The aged kidney also shows **net tubular reabsorption of creatinine**, which further underestimates true GFR.
3. There is **wide interindividual variation** in the rate of decline. Applying the "average 50% fall between 20 and 90 years" to an individual patient causes over- or under-dosing.
4. Non-age factors are often just as important: **state of hydration, cardiac output and intrinsic renal disease**.
5. **Tubular frailty** means the elderly tubule progresses easily to acute tubular necrosis and recovers slowly — a small nephrotoxic insult produces a large injury.
6. Pharmacokinetics are altered across the board — bioavailability, volume of distribution, GFR-dependent excretion, and the time to steady state (four half-lives). With polypharmacy, older people are **predisposed to drug toxicity**.

B. Patterns of Injury — Classify by Mechanism

7. **Haemodynamic (functional) injury:** **NSAIDs** block prostaglandin-mediated afferent vasodilatation; **ACE inhibitors and ARBs** remove efferent arteriolar tone; **diuretics** deplete intravascular volume. In bilateral renal artery stenosis, an ACE inhibitor can precipitate **reversible renal failure**. This triple combination is the classic community "triple whammy".
8. **Acute tubular necrosis:** nephrotoxins, classically **aminoglycosides**; also contrast, vancomycin, amphotericin, cisplatin, calcineurin inhibitors.
9. **Acute interstitial nephritis (AIN):** mainly a **delayed hypersensitivity response** to drugs — NSAIDs and diuretics. Most reported patients are elderly. Renal abnormalities usually improve once the drug is withdrawn, with or without steroids, but chronic kidney disease or even ESRD may follow.
10. **Proton-pump inhibitors** (omeprazole, esomeprazole, pantoprazole) are increasingly recognised as a cause of AIN. Diagnosis needs a high index of suspicion; renal biopsy shows **eosinophilic infiltration**, and prompt steroid therapy after confirmation restores function. For this and other reasons PPIs have become a **target of deprescribing** in older adults.
11. **Chronic analgesic nephropathy:** patients taking NSAIDs for months or years may develop **papillary necrosis, chronic interstitial nephritis, or end-stage renal disease**.
12. **Glomerular injury:** in some older people **membranous nephropathy** is related to drug ingestion (or to underlying malignancy in about 20%, mainly lung or colon adenocarcinoma); **minimal change disease** can be associated with NSAIDs.
13. **Electrolyte and acid–base toxicity:** on diuretics the elderly become hypokalaemic faster than the young; conversely they are predisposed to **hyperkalaemia on NSAIDs, ACE inhibitors, beta blockers and potassium-sparing diuretics**. **Metformin** can cause lactic acidosis — avoid in renal insufficiency and heart failure, measure creatinine clearance before starting in those aged 80 and over, and stop during volume-depleting illness and before surgery.
14. **Perioperative nephrotoxicity:** ACE inhibitors, diuretics and NSAIDs are the common culprits. Anaesthetic agents have little direct renal effect but reduce cardiac output, causing renal vasoconstriction and falling perfusion; prolonged enflurane or isoflurane generates fluoride, and sevoflurane at low fresh-gas flows generates compound A, though neither is usually a clinical problem.

C. Presentation and Diagnosis

15. Presentation is often **silent or atypical** — a rising creatinine, falling urine output, or a geriatric syndrome such as **delirium, falls, anorexia or unexplained weight loss**.
16. **Red flag:** AKI appearing shortly after introduction of a new drug — PPI, NSAID, antibiotic, diuretic or allopurinol — should be treated as drug-induced until proved otherwise.
17. Take a **complete drug history including over-the-counter analgesics and traditional or herbal preparations**, and establish the temporal relationship to drug initiation.
18. Estimate GFR with an eGFR equation rather than creatinine alone: **CKD-EPI is regarded as most accurate, Cockcroft–Gault as most sensitive** for dosing. For drugs with a narrow therapeutic-to-toxic ratio, measure actual creatinine clearance and drug blood levels.
19. **Renal biopsy** confirms interstitial nephritis when the diagnosis is uncertain and steroids are being considered.

D. Management and Prevention

20. **Withdraw the offending drug** — this is the single most effective intervention. Correct volume depletion, treat hyperkalaemia and acidosis, and dose all remaining drugs to the measured GFR.
21. **Corticosteroids** for biopsy-confirmed drug-induced AIN, given promptly, produce recovery of renal function; delay allows fibrosis.
22. **Prevention is the exam answer:** individualise treatment, use **low and slowly increasing doses**, and avoid polypharmacy. Alter dosing intervals for renally excreted drugs such as aminoglycosides and titrate to plasma levels. Stop nephrotoxic drugs before surgery and review diuretics against the patient's volume status and the planned procedure. Deprescribe PPIs and long-term NSAIDs.

Latest Updates (2023–Present)

- The **KDIGO 2026 AKI/AKD draft guideline** devotes a full chapter to **drug- and nephrotoxin-associated AKI**, covering therapeutic drug monitoring, pharmacokinetic and pharmacodynamic drug–drug interactions, **drug-induced crystalline nephropathies**, biomarkers to distinguish the aetiology of drug-related kidney disease, dosing during kidney replacement therapy, and — important in India — **nephrotoxicity of natural and herbal products**.
- **Pharmacist-led nephrotoxin stewardship** is now a recommended intervention, with dedicated evidence tables on pharmacist interventions in people with or at risk of AKI.
- Drug-induced AKI accounts for roughly **14–26% of AKI in hospitalised adults**, and AKI risk rises by about **53% for each additional nephrotoxic drug exposure**. The commonest identified culprits in recent cohorts are **vancomycin, iodinated contrast and aminoglycosides**.
- A large FAERS pharmacovigilance analysis of reports from 2004 to 2023 (published 2025) found **omeprazole to be the most frequently reported single culprit drug**, followed by furosemide, pantoprazole, esomeprazole and lansoprazole — strongly reinforcing the PPI deprescribing message in the textbook.
- **Sick-day rules ("SADMANS")** — **S**ulfonylureas, **A**CE inhibitors, **D**iuretics, **M**etformin, **A**RBs, **N**SAIDs, **S**GLT2 inhibitors — to be withheld during acute dehydrating illness. SGLT2 inhibitors are held because of euglycaemic ketoacidosis risk, metformin because of lactic acidosis. Caution in heart failure, where abrupt withdrawal of diuretics or ACE inhibitors can precipitate decompensation. **Restarting is as critical as stopping.**
- **AGS Beers Criteria 2023** advise avoiding systemic NSAIDs in older adults with CKD stage 4 or worse, and avoiding the combination of an ACE inhibitor/ARB with a potassium-sparing diuretic or NSAID.
- New drug classes with recognised renal signals: **immune checkpoint inhibitor-associated AIN** (PD-1/PD-L1 immunohistochemistry helps distinguish it), and the **vancomycin plus piperacillin–tazobactam** combination. Conversely, **tenofovir alafenamide is markedly less nephrotoxic than tenofovir disoproxil**.
- SGLT2 inhibitors, despite an early dip in eGFR, are now established as **nephroprotective** in the KDIGO 2024 CKD guideline and are not to be treated as nephrotoxins outside acute illness.

Current Practice Update

Issue	Textbook position	Current evidence (2023–2026)
Culprit drugs	NSAIDs, diuretics, aminoglycosides, ACE inhibitors, with PPIs newly recognised	**PPIs top the pharmacovigilance signal**; vancomycin, contrast and aminoglycosides dominate hospital cohorts
Estimating function	Cockcroft–Gault and MDRD; both may over- or underestimate	CKD-EPI 2021 without race; **cystatin C-based eGFR** recommended in low muscle mass and before critical dosing decisions
Deprescribing	Avoid polypharmacy, start low and go slow	Formalised as **nephrotoxin stewardship** with pharmacist-led reviews, e-alerts and explicit sick-day rules
SGLT2 inhibitors	Newer agents, limited elderly data	Renoprotective long term, but on the **SADMANS hold list during acute illness** — both facts must be stated
Herbal products	Not a major theme	Explicit guideline table on nephrotoxicity of natural products; highly relevant to Indian practice (aristolochic acid, heavy-metal-contaminated preparations)

Exam One-Liners

- Commonest mechanism of drug-induced AKI overall: **acute tubular necrosis**.
- Interstitial nephritis is a **delayed hypersensitivity** reaction; biopsy shows **eosinophilic infiltration**.
- PPIs cause AIN and are a **deprescribing target** in older adults.
- NSAIDs for months to years → **papillary necrosis, chronic interstitial nephritis, ESRD**.

- ACE inhibitor + bilateral renal artery stenosis = **reversible renal failure**.
- Metformin → **lactic acidosis**; check creatinine clearance before starting at age 80 and above.
- Elderly get **hypokalaemia faster on diuretics** but are **predisposed to hyperkalaemia** on NSAIDs, ACE inhibitors, beta blockers and potassium-sparing diuretics.
- Principle of geriatric prescribing: **individualise, start low, go slow, avoid polypharmacy, dose to GFR**.

Extra Recall Points — Recurring Topic

Mnemonics

- **Triple whammy:** ACE inhibitor/ARB + diuretic + NSAID — the commonest community cause of drug-induced AKI.
- **SADMANS** — the sick-day hold list: Sulfonylureas, ACE inhibitors, Diuretics, Metformin, ARBs, NSAIDs, SGLT2 inhibitors.
- **Patterns — "HAIC-G": H**aemodynamic, **A**TN, **I**nterstitial nephritis, **C**rystalline nephropathy, **G**lomerular.
- **Crystal formers — "MASA":** Methotrexate, Acyclovir, Sulfadiazine, Atazanavir/indinavir.

Numerical Anchors

- Drug-induced AKI = **14–26%** of hospital AKI; risk up **53%** per additional nephrotoxin.
- Aminoglycoside AKI in **10–25%** of exposed patients; tenofovir/cidofovir in **12–24%**.
- Time to steady state = **4 × half-life**.
- Membranous nephropathy in the elderly is malignancy-related in about **20%**.

Examiner Traps

1. Listing drugs without **classifying by mechanism** — the marks are in the classification.
2. Forgetting that a **normal creatinine does not exclude significant impairment** in a sarcopenic patient.
3. Omitting **PPIs**, which are the most examined "newer" cause.
4. Calling SGLT2 inhibitors nephrotoxic — they are renoprotective, but held during acute illness.
5. Forgetting **herbal and over-the-counter preparations** in the drug history, especially in Indian practice.
6. Stopping drugs and never mentioning **restarting** them after recovery.

15-Second Answer Skeleton

Why elderly are susceptible (pharmacokinetics, unreliable creatinine, tubular frailty, polypharmacy) → classify injury by mechanism: **haemodynamic / ATN / interstitial / crystalline / glomerular / electrolyte** → name the drugs in each → presentation is atypical, red flag is a new drug → diagnosis: drug history, eGFR equation, biopsy → management: withdraw drug, steroids for AIN, dose to GFR → prevention: start low, go slow, deprescribe, sick-day rules, nephrotoxin stewardship.

QUESTION 3

Metabolic acidosis

7 MARKS

High-Yield Points

1. **Definition:** a primary fall in serum bicarbonate with a fall in blood pH, compensated by hyperventilation and a fall in pCO_2. Normal bicarbonate is **22–26 mEq/L**; expected compensation is a fall in pCO_2 of about **1.2 mmHg for every 1 mEq/L fall in bicarbonate**.
2. **Classification is by anion gap** = $Na^+ - (Cl^- + HCO_3^-)$, normally **8–12 mEq/L**.
   High anion gap (added acid) — MUDPILES: Methanol, Uraemia, Diabetic ketoacidosis, Paraldehyde, Iron/Isoniazid, Lactic acidosis, Ethylene glycol, Salicylates.
   Normal anion gap (hyperchloraemic, bicarbonate loss): diarrhoea, renal tubular acidosis, carbonic anhydrase inhibitors, ureteric diversion, large-volume 0.9% saline.
3. **Key geriatric physiology:** older people take **longer to reach peak proton excretion**. After an acid load, maximal ammonia and titratable acid excretion is reached in **4 hours in the young but only at 6–8 hours in the old**, so the elderly have **greater difficulty handling states of acidosis** even though their baseline acid excretion is normal.
4. **Common causes in older adults:** acute kidney injury and chronic kidney disease (retained acid and uraemia); sepsis, shock and dehydration (lactic acidosis); diarrhoea; diabetic ketoacidosis and mixed hyperglycaemic states; drugs, especially **metformin-induced lactic acidosis**; heat stroke, which causes metabolic acidosis with respiratory alkalosis.
5. A characteristic geriatric presentation is the **"mixed" metabolic disturbance** of older diabetics — neither classical ketoacidosis nor hyperosmolar coma, with glucose around **270–450 mg/dL**, arterial pH **7.2–7.3** (not markedly acidotic), and without marked dehydration or altered consciousness.
6. **Clinical features are often masked in the elderly:** Kussmaul breathing, anorexia, vomiting, weakness, hypotension and arrhythmia from hyperkalaemia, and frequently **delirium as the presenting feature**. Acid–base disturbance is also a recognised metabolic cause of apparent depression in old age.
7. **Chronic acidosis has geriatric consequences:** muscle protein catabolism worsening **sarcopenia**, bone demineralisation worsening **osteoporosis**, and accelerated CKD progression — linking it directly to falls and fracture risk.
8. **Investigations:** arterial blood gas, electrolytes with calculated anion gap, serum bicarbonate, lactate, ketones, glucose, urea and creatinine, and where relevant urine pH and urine anion gap (for renal tubular acidosis) and osmolal gap (for toxic alcohols).
9. **Management principle is to treat the cause**, not the number: volume resuscitation and restoration of perfusion for lactic acidosis, insulin with fluids for ketoacidosis, withdrawal of metformin, relief of obstruction or dialysis in AKI, and treatment of sepsis.
10. **Sodium bicarbonate** is reserved for severe acidaemia (pH below about 7.1) or for associated hyperkalaemia. Use it cautiously in the elderly — the **sodium load can precipitate heart failure and hypertension**, and it can cause hypokalaemia, hypocalcaemia and volume overload. In advanced CKD, dialysis is indicated for refractory acidosis, and replacement therapy is needed when GFR falls below **10 mL/min**.

Latest Updates (2023–Present) — Condensed

- **KDIGO 2024 CKD guideline downgraded alkali therapy to a practice point**, suggesting treatment be considered when **serum bicarbonate is below 18 mEq/L**, rather than routinely correcting all mild acidosis.
- **VALOR-CKD (JASN 2024)** — veverimer, a non-absorbed gastrointestinal hydrochloric acid binder, **did not slow CKD progression** in 1480 patients (eGFR 20–40 mL/min/1.73 m², bicarbonate 12–20 mEq/L), largely because only about 1 mEq/L of bicarbonate separation was achieved. The earlier **BiCARB** trial in patients over 60 with low-grade acidosis likewise showed no clinical benefit, higher cost and lower quality of life.
- A 2024 CJASN meta-analysis found sodium bicarbonate **improves kidney function and mid-arm muscle circumference** but **raises systolic blood pressure** — so in frail older adults with mild acidosis the balance now favours dietary alkali (fruit and vegetables, low potential renal acid load) and careful observation over reflex bicarbonate prescribing.

Current Practice Update

Issue	Textbook position	Current evidence (2023–2026)
Alkali in CKD	Correct acidosis to maintain bicarbonate in the normal range	Treat when **bicarbonate <18 mEq/L**; a practice point, not a strong recommendation
Novel agents	Sodium bicarbonate is the mainstay	**Veverimer failed** in VALOR-CKD; sodium bicarbonate remains the only practical option, plus dietary alkali

Older adults	Correction assumed beneficial	**BiCARB** showed no benefit and possible harm in over-60s with mild acidosis; watch sodium load and blood pressure
Resuscitation fluid	0.9% saline for volume repletion	**Balanced crystalloids** preferred for large volumes, to avoid iatrogenic hyperchloraemic acidosis

Exam One-Liners

- Anion gap = Na − (Cl + HCO_3); normal **8–12 mEq/L**.
- Expected pCO_2 = **1.5 × HCO_3 + 8 ± 2** (Winter's formula).
- The elderly reach peak acid excretion at **6–8 hours instead of 4**.
- Metformin → **high anion gap lactic acidosis**; heat stroke → **metabolic acidosis with respiratory alkalosis**.
- Chronic acidosis drives **sarcopenia and bone demineralisation**.
- Bicarbonate therapy threshold in CKD is now **<18 mEq/L**.

Examiner Traps

1. Calculating the anion gap but forgetting to **correct for albumin** (add 2.5 mEq/L for every 1 g/dL fall below 4 g/dL) — hypoalbuminaemia is near-universal in frail elderly and masks a raised gap.
2. Giving bicarbonate reflexively in lactic acidosis instead of restoring perfusion.
3. Missing the **delta-delta** check for a coexisting normal-gap acidosis or metabolic alkalosis (common with vomiting or diuretics).
4. Forgetting that **delirium may be the only presenting sign** in an older patient.

If Asked as a 15-Mark Question, Expand With

Detailed physiology of renal acid handling (bicarbonate reabsorption in the proximal tubule, titratable acid and ammoniagenesis distally) • full classification table of high vs normal anion gap causes with the delta-delta ratio • the four types of **renal tubular acidosis** with their urine pH, potassium and stone associations • systemic effects of acidaemia organ by organ (cardiac contractility, arrhythmia, catecholamine resistance, insulin resistance, hyperkalaemia, bone buffering, protein catabolism) • the geriatric consequences chain: acidosis → sarcopenia + osteoporosis → falls and fracture • specific management protocols for diabetic ketoacidosis and hyperosmolar states in the elderly, with slower fluid rates and closer attention to cardiac and cognitive status • renal replacement therapy indications (AEIOU) • the 2023–2026 evidence block above.

FLUID & ELECTROLYTE BALANCE IN THE ELDERLY

High-Yield Examination Answers

MD Geriatric Medicine — Structured Revision Notes

Primary source: *Pathy's Principles and Practice of Geriatric Medicine*, 6th edition (Chapter 15)

Supplemented by *Essentials of Clinical Geriatrics*, 7th edition

2 QUESTIONS · 15-MARK FORMAT · UPDATED TO AUGUST 2026

Textbook content and current evidence are presented separately throughout.

Contents

1. **Fluid and Electrolyte Disturbances in the Elderly and their Management** — **15 MARKS**
Normal water homeostasis · age-related changes · dehydration · hypernatremia · hyponatremia overview · drug causes · correction formulae · prevention

2. **Approach to the Evaluation and Treatment of Hyponatremia** — **15 MARKS**
Stepwise diagnostic algorithm · volume-status classification · SIADH criteria · hypertonic saline protocols · osmotic demyelination · vaptans

3. **Rapid Revision Grid** — **APPENDIX**
Numbers, thresholds, formulae and named trials on one page

How this document is organised. Each answer opens with 15–20 numbered high-yield points drawn from the uploaded textbook, arranged in the standard sequence (definition → physiology → epidemiology → aetiology → clinical features → diagnosis → management → prevention). This is followed by a **Latest Updates (2023–Present)** section with inline citations, a two-column **Current Practice Update** table contrasting the textbook position with current evidence, an orange **Examiner's Tip** panel with mark-earning lines, and — because both topics recur across examinations — a green **Extra Points to Remember** panel.

Q1. Fluid and electrolyte disturbances in the elderly and their management.

15 MARKS · 20 HIGH-YIELD POINTS · RECURRING TOPIC

Textbook Answer — 20 High-Yield Points

A. Normal Physiology (Points 1–4)

1. Water balance has **dual control**. The **hypothalamus and posterior pituitary** handle water retention through ADH, while the **renin–angiotensin–aldosterone axis** handles sodium retention and blood pressure.
2. **ADH (arginine vasopressin)** is made in the **supraoptic and paraventricular nuclei** and released when serum osmolality rises. Normal serum osmolality is kept at **285 ± 2 mOsm/kg**. The ADH release threshold is about **280 mOsm/kg**.
3. **Thirst** is controlled by the **organum vasculosum of the lamina terminalis (OVLT)**, which lies outside the blood–brain barrier. Thirst normally lags behind ADH — its threshold is about **5–10 mOsm/kg higher** than the ADH threshold.
4. **Non-osmotic ADH release** occurs through baroreceptors: high-pressure receptors in the **carotid sinus and aortic arch**, and low-pressure stretch receptors in the **cardiac atria and pulmonary veins**. A fall in **effective arterial blood volume (EABV)** therefore raises ADH even when osmolality is low.

Renal handling of water: GFR 300 L/day → about **30 L/day** reaches the distal collecting tubule
With ADH: urine 0.7 L/day at 600–1200 mOsm/kg | **Without ADH:** urine 10–20 L/day at <100 mOsm/kg
The medullary concentration gradient is generated by the urea countercurrent system; water moves via aquaporin-2 channels.

B. Age-Related Changes (Points 5–8)

5. **Total body water falls from 60% to 50% of body weight after age 75.** A smaller water reservoir means even short periods of poor intake cause large shifts — this is the single most important predisposing change.
6. After water deprivation, healthy older adults show **reduced thirst**, **higher serum ADH levels**, but **reduced urinary concentrating ability** and **reduced free water excretion**. They can still secrete ADH; the kidney simply responds less well to it.
7. Renal resistance to ADH is due to **reduced expression of the V2 receptor and aquaporin-2** in the collecting duct with ageing.
8. Fluid homeostasis is **not** affected by the age-related GFR decline until GFR falls to **30–50 mL/min**. Tubular function is mildly impaired — water, sodium and potassium handling all decline, and the **aldosterone response is blunted**.

C. Epidemiology and Dehydration (Points 9–10)

9. **Frequency in the frail elderly.** Sodium and water disturbances occur in **18–63%** of frail long-term care residents. Hyponatremia affects **7–11% of outpatients** and **11–53% of those in long-term care**. Hypernatremia affects about **18% of institutionalised elderly**, rising to **63% during acute febrile illness**.
10. **Dehydration.** Suspect it when there is acute weight loss of **≥3% of body mass** or **>1 kg per day**. Commonest causes are **infection, altered consciousness, cognitive impairment and diuretics**. Hospital mortality may reach **50%**. General recommendation is about **1.5 litres of fluid daily**. Skin turgor is unreliable in old age (subcutaneous atrophy) — if used at all, test over the **chest or abdomen**.

D. Hypernatremia (Points 11–14)

11. **Definition: serum sodium >145 mEq/L.** Normal ageing physiology tends *towards* hypernatremia. In the frail elderly the causes are **reduced thirst, inadequate access to fluids, and superimposed gastrointestinal or febrile losses**. Institutionalised patients dependent on carers for drinks are the highest-risk group.
12. **Alzheimer's disease is a special risk.** These patients have **both** a blunted thirst response (in the range of essential hypernatremia) **and** inappropriately low ADH levels — a picture resembling **partial central diabetes insipidus**. Nephrogenic DI may be added by **hypercalcaemia, hypokalaemia or lithium**.
13. **Clinical features** are mainly neurological: confusion, disorientation, drowsiness, coma. In extreme cases brain shrinkage causes **vascular rupture and subarachnoid haemorrhage**. Chronic dehydration causes adaptive intracellular generation of organic osmoles, so **rapid correction risks cerebral oedema**.
14. **Management.** Treat the cause first (control pyrexia, replace gastrointestinal losses, stop diuretics and lactulose). Aim to drop serum osmolality by only about **10 mOsm/kg over 24 hours** — do **not** fully normalise the sodium. Check serum sodium **every 4 hours**.

Water replacement over 24 h = TBW × $[(Na_{goal} / Na_{current}) - 1]$ + insensible, gastrointestinal and urinary losses

where $Na_{goal} = Na_{current} - 10$ mEq/L

TBW = 0.6 × body weight (kg) if age <75 y | **TBW** = 0.5 × body weight (kg) if age >75 y

Obligatory ongoing free water loss is 0.5–1.5 L/day depending on fever and diarrhoea.

15. **Choice of fluid in hypernatremia.** If loss is **pure water**, 5% dextrose is simplest to calculate. If **solute is also lost**, 0.45% saline is more appropriate — the sodium stays intravascular to support blood pressure while the 50% free water component corrects osmolality. **0.9% saline will not lower serum osmolality** and may not deliver enough free water to keep pace with obligatory losses, even though it is hypotonic relative to the patient.

E. Hyponatremia in the Elderly (Points 16–18)

16. **Definition: serum sodium <135 mEq/L**; **<130 mEq/L** is clinically significant; **<120 mEq/L** is severe. Almost all cases involve an imbalance of ADH — either a true SIADH or an "appropriate" ADH rise driven by low EABV.

Serum osmolality = 2 × [Na] + glucose/18 + BUN/2.8 (mg/dL units)

Effective serum osmolality = 2 × [Na] + glucose/18

Urea is excluded from effective osmolality because it crosses cell membranes freely and is not an effective osmole.

17. **Drug causes — the commonest reversible group. Diuretics** are the most frequent (prevalence up to 11% in geriatric practice); the elderly are especially sensitive to **SSRIs**. Also implicated: tricyclic and tetracyclic antidepressants, phenothiazines and butyrophenones, **carbamazepine, oxcarbazepine and valproate**, opioids and nicotine, NSAIDs (they lower prostaglandins, which normally oppose ADH), high-dose co-trimoxazole, and vincristine and cyclophosphamide.

18. **Clinical consequences.** Hyponatremia is linked to **gait disturbance, unsteadiness, attention deficits, falls and fractures**, and to a **2.36-fold higher chance of dementia**. There may be direct activation of osteoclastic bone resorption. However, **causality is not proven** — hyponatremic patients also take more SSRIs, benzodiazepines and CNS drugs and have more baseline cognitive impairment and orthostatic hypotension.

F. Principles of Correction and Prevention (Points 19–20)

19. **Correction is guided by neurocognitive symptoms, not by the number alone.** The two rate limits are the most examinable facts in this chapter:
    - **Hyponatremia:** raise serum sodium by **6–8 mEq/L per 24 hours** — a rise **>8 mEq/L/24 h** risks **osmotic demyelination syndrome (ODS)**, both pontine and extrapontine, which is delayed in onset and carries severe morbidity and mortality.
    - **Hypernatremia:** lower serum sodium by **<10 mEq/L per 24 hours** to avoid cerebral oedema.

20. **Prevention in practice.** Limit drugs associated with hyponatremia (especially **thiazides and SSRIs**); avoid drugs causing orthostatic hypotension; monitor postural blood pressure; moderate the intensity of systolic blood pressure lowering compared with younger patients; and ensure **carer-assisted hydration** for institutionalised and demented patients, particularly during intercurrent febrile illness. Water and sodium disturbances independently **predict the prognosis of acute illness** in frail elderly long-term care residents.

Latest Updates (2023–Present)

Diagnosis of dehydration has moved to an objective, laboratory-based standard

- The **ESPEN geriatric nutrition and hydration guideline** now names **directly measured serum or plasma osmolality** as the reference standard for low-intake dehydration, with a threshold of **≥300 mOsm/kg** for current dehydration and **295–300 mOsm/kg** for impending dehydration.
- Where direct osmometry is unavailable — the usual situation in Indian district hospitals — the validated substitute is a **calculated osmolarity** from routine biochemistry, at a cut-off of **>295–296 mOsm/L** (sensitivity ~97%, specificity ~76%):

Calculated osmolarity = 1.86 × ($Na^+ + K^+$) + 1.15 × glucose + urea + 14 (all in mmol/L)

Best-performing of 38 equations tested in frail older people (Hooper et al., *BMJ Open*; validated in the DRIE cohort). Note this needs SI units, so convert Indian mg/dL values first.

Two distinct types of dehydration are now separated

- *Age and Ageing* (2023) "New horizons" review distinguishes **hypertonic dehydration** (a pure water deficit) from **isotonic dehydration** (loss of both water and salt). Crucially, **serum osmolality cannot diagnose isotonic dehydration** — a normal osmolality does not exclude volume depletion.
- Individual clinical signs and bedside tests perform **poorly** in older people. **Salivary osmolality point-of-care testing** is a promising rapid test able to detect both types, though evidence is still developing.

- **Hypodermoclysis (subcutaneous fluid)** is confirmed as safer and easier to start than the intravenous route, limited only by infusion speed and volume — highly relevant to Indian long-term care and home-based NPHCE services where venous access and nursing supervision are limited.

Hydration status as a long-term health determinant

- *Nature Reviews Nephrology* (2024) reviewed long-term outcomes of hydration status: serum sodium in the **upper part of the normal range**, reflecting habitual under-hydration, is associated with accelerated biological ageing, higher chronic disease burden and earlier mortality. This reframes hydration from an acute-care issue to a preventive one.

Indian data

- Indian hospital series report hyponatremia in **18–29% of elderly inpatients**. A Mumbai tertiary-centre prospective study (*Cureus*, 2025) found an overall prevalence of **29.4%**, with the elderly disproportionately affected and a multifactorial aetiology (comorbidity plus diuretics), followed by liver disease, endocrine disorders and SIADH each around 13%.
- A Pune series (*Cureus*, 2024) of 100 elderly inpatients found **severe hyponatremia (<125 mEq/L) in 61%**, presenting most often as **generalised weakness (22%) and disorientation (17%)** — symptoms easily dismissed as "ageing".
- **AIIMS Rishikesh** (*BMC Geriatrics*, 2023) showed that hyponatremic geriatric patients had significantly worse **Comprehensive Geriatric Assessment** scores — Hindi Mental State Examination, Barthel ADL index, Timed Up and Go, and handgrip strength — and that **all four improved when serum sodium was corrected**. This is the strongest Indian evidence that correction is functionally worthwhile, not merely biochemical.

Current Practice Update

Issue	Textbook position (Pathy's 6th ed.)	Current evidence (2023–2026)
Diagnosing dehydration	Clinical — acute weight loss ≥3% or >1 kg/day; skin turgor over chest/abdomen; no laboratory threshold given.	Objective threshold: measured serum osmolality **≥300 mOsm/kg**, or calculated osmolarity **>295 mOsm/L** using the ESPEN equation. Clinical signs alone are considered unreliable.
Type of dehydration	Treated as a single entity (water deficit).	Split into **hypertonic** (water deficit) and **isotonic** (water + salt deficit). Osmolality detects only the former.
Route of rehydration	Oral, intravenous, or subcutaneous fluids mentioned in passing.	**Hypodermoclysis** explicitly endorsed as safer and easier to initiate than IV in frail and long-term care patients.
Daily fluid target	Approximately **1.5 L/day** for older adults.	Broadly unchanged, but daily requirements may be **lower than previously assumed**; evidence specific to older people remains limited, so individualisation is advised.
Value of correcting mild hyponatremia	Associations with falls, fractures and dementia noted, but **causality not proven**; vaptan trials showed only modest benefit.	Indian CGA data (AIIMS Rishikesh 2023) show measurable improvement in **cognition, ADL, mobility and grip strength** after correction, strengthening the case for active treatment.

Examiner's Tip — verbatim lines that earn marks

- "**Total body water falls from 60% to 50% of body weight after the age of 75**, so the elderly have a markedly reduced buffer against short periods of poor intake."
- "The ageing kidney shows **reduced aquaporin-2 and V2 receptor expression**, producing renal resistance to ADH despite preserved ADH secretion."
- "Correction must be guided by neurocognitive symptoms: **raise sodium by 6–8 mEq/L per 24 hours in hyponatremia, and lower it by less than 10 mEq/L per 24 hours in hypernatremia**."
- "**0.9% saline is hypotonic to the patient in hypernatremia but does not lower serum osmolality** and may fail to keep pace with obligatory free water losses."
- Always write the **TBW formula with the age-75 cut-off** (0.6 vs 0.5 × body weight) — it is the single most reliably awarded calculation in this question.
- Close with prevention: "**limit thiazides and SSRIs, monitor postural blood pressure, and ensure carer-assisted hydration in dependent and demented patients.**"

Extra Points to Remember (Recurring Topic)

- **Why the elderly drift towards hypernatremia but present with hyponatremia.** Normal ageing physiology (blunted thirst, poor concentrating ability) predisposes to **hypernatremia**; superimposed **drugs and comorbidity** (diuretics, SSRIs, heart failure, cirrhosis) produce **hyponatremia**. Examiners like this paradox — state it explicitly.
- **Alzheimer's disease is the classic hypernatremia scenario:** blunted thirst *plus* inappropriately low ADH, mimicking partial central diabetes insipidus. Nursing-home residents dependent on carers reach **63% incidence during febrile illness**.
- **Pathy's functional categorisation applies here.** In the **robust** older adult, aim for full biochemical correction and investigate the cause. In the **frail**, correct cautiously and prioritise deprescribing. In the **moderately demented**, the intervention with the largest yield is **structured carer-assisted hydration**. At the **end of life**, clinically assisted hydration is a comfort decision, not a biochemical one.
- **Do not forget potassium.** Ageing reduces tubular potassium handling and blunts the aldosterone response, so the elderly tolerate both extremes poorly. **Thiazides** cause hypokalaemia, volume depletion, hyponatremia, hyperglycaemia and hyperuricaemia; **ACE inhibitors, ARBs and eplerenone** cause hyperkalaemia and should not be combined with potassium-sparing diuretics. Eplerenone is contraindicated with creatinine >2.0 mg/dL.
- **Two-line mention of hyperosmolar hyperglycaemic state** if the question is broad: it develops over days to weeks (unlike DKA over hours), has a **larger fluid deficit, higher serum sodium and higher blood urea**, and seizures are common. Resuscitate with normal saline first — volume repletion alone often lowers glucose.
- **Indian framing sentence:** "In Indian practice, where summer heat stress, febrile illness, gastroenteritis and unsupervised diuretic use are common and formal long-term care nursing is limited, dehydration in the dependent elderly is largely a **preventable, carer-dependent problem**, and is best addressed through NPHCE home-based care and Ayushman Arogya Mandir follow-up rather than in hospital."

Q2. Approach to the evaluation and treatment of hyponatremia.

15 MARKS · 20 HIGH-YIELD POINTS · RECURRING TOPIC

Textbook Answer — 20 High-Yield Points

A. Definition and First Principles (Points 1–3)

1. **Definitions.** Hyponatremia is serum sodium **<135 mEq/L**. Clinically significant hyponatremia is **<130 mEq/L**; severe hyponatremia is **<120 mEq/L**. Levels of 130–135 mEq/L are usually asymptomatic.
2. **Step 1 — confirm true hypotonic hyponatremia.** Measure serum osmolality. True hypo-osmolality is **<275 mOsm/kg**. This step excludes two traps:
   - **Pseudo-hyponatremia** — from excess triglycerides or paraproteins (macroglobulinaemia); osmolality is normal.
   - **Hyperosmolar (translocational) hyponatremia** — from hyperglycaemia or other circulating osmoles; osmolality is high.
3. **Step 2 — take a water intake history.** Look for intake that overwhelms renal free water clearance: **primary polydipsia** (16–20 L/day), **beer potomania**, and **exercise-associated water intoxication** in long-distance runners. Without such a history, the cause is **water retention**, usually with some component of solute loss.

B. Laboratory Evaluation (Points 4–6)

4. **Step 3 — the core panel.** Send serum for **basic metabolic profile (BMP)** and **measured osmolality**, and urine for **osmolality** and **sodium concentration**. The BMP identifies acute renal disease (acute tubular necrosis, obstruction) and hyperglycaemia.
5. **Step 4 — interpret the urine.**
   - **Urine osmolality >100 mOsm/kg** means ADH action is present and inappropriate; it is often *higher* than serum osmolality.
   - **Urine sodium >30–40 mEq/L** (sometimes approaching 100) indicates SIADH or renal salt loss.
   - **Urine sodium <20 mEq/L**, or the presence of orthostatic hypotension, indicates an "appropriate" ADH response to low effective arterial blood volume.
6. **Copeptin** — the stable C-terminal fragment of the AVP precursor — used together with the urinary sodium ratio, helps distinguish **volume-depleted from normovolemic** hyponatremia when clinical assessment is equivocal.

C. Volume-Status Classification (Points 7–9)

7. **Hypovolemic hyponatremia.** Orthostatic hypotension and/or urine sodium <20 mEq/L. Causes: diuretics, gastrointestinal losses. Physiologically, aldosterone and ADH are both **appropriately** elevated because of low EABV.
8. **Hypervolemic (oedematous) hyponatremia.** Two mechanisms:
   - **Hypoalbuminaemia with low EABV** — cirrhosis, nephrotic syndrome.
   - **Reduced carotid baroreceptor perfusion** — congestive heart failure, cardiac tamponade, negative inotropic drugs, and poor contractility in hypothyroidism.
9. **Euvolemic hyponatremia — SIADH.** Diagnostic set from the textbook algorithm: **serum osmolality <275 mOsm/kg**, **urine osmolality >100 mOsm/kg**, **urine sodium >40 mEq/L**, clinical euvolemia, and normal thyroid and adrenal function.

D. Completing the Search for a Cause (Points 10–12)

10. **Step 5 — review every drug.** This is mandatory and is the highest-yield reversible cause. Diuretics (commonest overall), SSRIs (elderly are especially sensitive), tricyclics and tetracyclics, phenothiazines and butyrophenones, carbamazepine, oxcarbazepine and valproate, opioids and nicotine, NSAIDs, high-dose co-trimoxazole, vincristine and cyclophosphamide, and MDMA.
11. **Step 6 — exclude endocrine causes.** Check **TSH** for hypothyroidism, and test for **primary and secondary adrenal insufficiency by cosyntropin stimulation**. Also assess for mineralocorticoid insufficiency and for autonomic dysfunction (**Parkinson's disease, multiple system atrophy**). **Correction of hypothyroidism and hypoadrenalism is mandatory** before labelling a patient SIADH.
12. **Causes of true SIADH.** Tumours (**bronchogenic carcinoma**, mesothelioma, thymoma, other non-pulmonary carcinoma); CNS lesions (tumour, trauma, neurosurgery, **subarachnoid haemorrhage**); pulmonary disease with raised intrathoracic pressure (**COPD**, positive-pressure ventilation); acute post-operative and post-anaesthetic states; **cerebral salt wasting**; and HIV–AIDS. In practice, an exhaustive evaluation of 50 elderly inpatients with mild-to-moderate hyponatremia linked it to **pneumonia and medication**, with **60% remaining idiopathic**.

E. Treatment (Points 13–18)

13. **The decision to treat rests on neurocognitive symptoms, not the number.** Sodium 130–135 mEq/L is usually asymptomatic and, if chronic, the brain has already adapted — it may need no therapy at all. Sodium 125–130 mEq/L with gait disturbance or cognitive impairment **does improve** with correction. Withholding causative drugs alone is often sufficient.
14. **Hypovolemic hyponatremia.** Give a trial of **1–2 litres of 0.9% saline**, withhold diuretics, and consider **oral salt 4–12 g/day in divided doses**. Restoring EABV suppresses ADH and raises sodium. **Fluid restriction is contraindicated here.**
15. **Euvolemic and hypervolemic hyponatremia. Fluid restriction 500–1500 mL/24 h** is the mainstay. **0.9% saline is usually ineffective and may worsen SIADH** — the infused sodium is quantitatively excreted in the urine while the high ADH retains the water.
16. **Severe symptomatic hyponatremia (sodium <120 mEq/L or hyponatremic encephalopathy).** Use **3% saline (513 mEq/L)**. The textbook protocols are:
    - **Continuous infusion** at **0.5–1.0 mL/kg/hour**, adjusted to urine output; or
    - **Bolus** of **100 mL of 3% saline**, with the rate then adjusted.
    - Check serum sodium **every 2 hours**; **stop the infusion if the rise exceeds 0.5 mEq/L/hour**.
    - Target a rise of only **4–8 mEq/L over 24 hours** — the goal is to remove a calculated portion of the excess free water, **not** to normalise the sodium.
17. **Osmotic demyelination syndrome (ODS) — the feared complication.** Chronic hyponatremia (>48 hours) causes the brain to extrude organic and inorganic osmoles. Correction **>8 mEq/L over 24 hours** then causes cerebral dehydration and **pontine and extrapontine demyelination**, which is **delayed in onset** and carries severe neurological morbidity and mortality.
    - **High-risk group:** serum sodium <105 mEq/L, **hypokalaemia**, alcoholism, malnutrition, advanced liver disease.
    - **If overcorrected:** re-lower with free water (dextrose) **± DDAVP**. In the bolus regimen, 14% of patients required this.
18. **Bolus versus continuous infusion.** In a historical case-control comparison, a **100 mL bolus of 3% saline was preferable to a slow 20 mL/h infusion**, with quicker restoration of the Glasgow Coma Scale. The slow-infusion group showed a trend towards higher mortality (4 of 28 died vs 0 of 22). The textbook conclusion is that it remains **unknown** which strategy is superior in real-world conditions, or whether reversing overcorrection halts established ODS.

F. Vaptans and Prognosis (Points 19–20)

19. **Vaptans (vasopressin V2-receptor antagonists).** They reliably raise serum sodium and allow fluid restriction to be stopped, but clinical benefit is modest:
    - **Tolvaptan — SALT-1 and SALT-2** (448 subjects): small overall improvement in the SF-12 quality-of-life score ($p = 0.02$), significant only in the US cohort; benefit greatest when baseline sodium was <130 mEq/L. No change in the physical component.
    - **INSIGHT trial** (107 patients with SIADH): raising sodium from 129 to 136 mEq/L did **not** improve the primary composite cognitive endpoint, only a psychomotor speed subdomain.
    - **Lixivaptan:** raised sodium by 3 mEq/L over control, with no benefit on the Trail Making Test or MOS-6 at 28 days.
    - **Systematic review:** all vaptans improve serum sodium, with **limited evidence of impact on quality of life or mortality**.
20. **Prognosis.** Hyponatremia carries a poor overall prognosis. There are probably **two temporal syndromes** in the elderly, often hard to separate: a **chronic** hyponatremia linked to underlying disease or dementia, and an **acute deterioration** manifesting as delirium. Ongoing management is to **limit thiazides and SSRIs**, avoid drugs causing orthostatic hypotension, and moderate antihypertensive intensity.

Latest Updates (2023–Present)

The 2024 treatment standard has settled the dosing of hypertonic saline

- The ERA **"Hyponatraemia — treatment standard 2024"** (*Nephrology Dialysis Transplantation*, 2024) recommends **3% saline as a 100–150 mL bolus (2–4 mL/kg) over 20–40 minutes, repeated until the target is reached**, or alternatively a continuous infusion at **0.5–1 mL/kg/hour**. It states that **rapid intermittent bolus administration is preferred** for symptomatic hyponatraemia.
- The aim of the first bolus is a **rise of about 5 mmol/L**, which is enough to reduce intracranial pressure from cerebral oedema.
- The **SALSA randomised trial** (*JAMA Internal Medicine*, 2021; 178 patients, sodium ≤125 mmol/L) found rapid

intermittent bolus and slow continuous infusion **equally effective and safe with no difference in overcorrection risk**, but bolus therapy required **less relowering treatment** and achieved target sodium within one hour more often. A 2025 Indian meta-analysis (Dutta et al., New Delhi) confirmed both are safe, with continuous infusion associated with **shorter hospital stay**.

The 8 mEq/L limit is now genuinely contested

- **MacMillan et al.** (*NEJM Evidence*, 2023; 22,858 admissions, Toronto): ODS occurred in only **0.05%** overall — fewer than **5 per 10,000** when initial sodium was <130 mmol/L and about **3 per 1,000** when <120 mmol/L. Strikingly, **7 of the 12 ODS cases had no rapid correction at all**. Rapid correction occurred in **69%** of patients with sodium <110 mmol/L despite efforts to avoid it.
- **Seethapathy et al.** (*NEJM Evidence*, 2023; 3,274 patients with sodium <120 mmol/L): **slower correction was associated with higher mortality and longer hospital stay**; 5 of 7 ODS cases had **not** exceeded 8 mmol/L in 24 hours.
- **Ayus et al. meta-analysis** (*JAMA Internal Medicine*, 2025; 16 cohorts, 11,811 patients): correction at **8–10 mEq/L per 24 h** was associated with lower in-hospital mortality (OR 0.67) and lower 30-day mortality (RR 0.55), with **no significant difference in ODS incidence**.
- **See et al. meta-analysis** (*Journal of Critical Care Medicine*, 2024; 6,032 patients): overall ODS incidence **0.48%**. Rapid correction raised ODS risk nearly four-fold (RR 3.91) *but* halved in-hospital mortality. Importantly, **89% of ODS cases had alcohol use disorder, hypokalaemia, cirrhosis or a very low baseline sodium** — risk factors matter more than rate alone.
- A large integrated health-system cohort of **13,988 patients** (*Annals of Internal Medicine*, 2026) again found faster correction associated with **lower 90-day death or delayed neurologic events**.
- **The counter-argument.** Sterns and Rondon-Berrios (*American Journal of Kidney Diseases*, 2025) argue these newer studies are **confounded** — comorbidity drives both mortality and slow correction — and that the safety limits should not be relaxed. **Guidelines have not changed; the 6–8 mEq/L target remains standard of care.**

A genuinely new drug class for SIADH

- **Empagliflozin (SGLT2 inhibitor).** Refardt et al. (*Journal of the American Society of Nephrology*, 2023) conducted a randomised, double-blind, placebo-controlled **crossover** trial of empagliflozin 25 mg/day for 4 weeks in outpatients with chronic SIAD (n = 14, median age 72). It **raised serum sodium** and possibly **improved neurocognitive function (MoCA)**. The mechanism is **glycosuria-induced osmotic diuresis with increased electrolyte-free water clearance** — entirely independent of vasopressin receptor blockade.
- An earlier trial in **87 hospitalised SIAD patients** showed 4-day empagliflozin plus fluid restriction raised sodium, with **87% vs 68%** reaching sodium ≥130 mmol/L. The larger **EMPOWER** trial in euvolemic and hypervolemic hyponatremia is ongoing.
- This is highly relevant to India: empagliflozin is **widely available and inexpensive**, whereas tolvaptan is costly and urea is poorly tolerated. Use in SIAD remains **off-label**.
- **Oral urea** and the **proactive DDAVP clamp** (scheduled desmopressin with strict free water restriction, correcting sodium deliberately with 3% saline boluses) are both increasingly used to give controlled, predictable correction and prevent inadvertent overshoot.

Current Practice Update

Issue	Textbook position (Pathy's 6th ed.)	Current evidence (2023–2026)
Bolus vs continuous 3% saline	"Unknown whether bolus or continuous infusions, or a hybrid, will be better in real-world conditions."	Settled by the **SALSA RCT**: both safe and effective. ERA **2024 treatment standard prefers rapid intermittent bolus** for symptomatic hyponatraemia.
Bolus dose	100 mL of 3% saline once, then rate adjusted.	**100–150 mL (2–4 mL/kg) over 20–40 minutes**, repeated as needed; first bolus targets a **5 mmol/L rise**.
Risk of ODS	Presented as the dominant hazard of correction; rate limit of 8 mEq/L/24 h is absolute.	ODS is **rarer than assumed (0.05–0.5%)** and often occurs **without** rapid correction. Several large cohorts link **slow** correction to **higher mortality**. The **limit still stands**, but risk-factor stratification (hypokalaemia, alcoholism, cirrhosis, Na <105) now matters as much as rate.

Second-line therapy for chronic SIADH	**Vaptans** — effective biochemically, modest clinical benefit.	**Empagliflozin** emerges as a promising, cheap, well-tolerated alternative (JASN 2023). **Urea** is re-established as a mainstream second-line option.
Preventing overcorrection	Re-lower with free water ± DDAVP if the limit is exceeded (reactive).	Increasing use of the **proactive DDAVP clamp** — scheduled desmopressin from the outset with controlled 3% saline boluses.

Examiner's Tip — verbatim lines that earn marks

- Open with the **three-step framework**, then follow it: "**First confirm true hypotonic hyponatremia, then assess volume status, then identify the cause.**"
- "**Serum osmolality <275 mOsm/kg confirms true hypo-osmolality and excludes pseudo-hyponatremia and hyperosmolar hyponatremia.**"
- Give the SIADH criteria as a numbered set: "**Serum osmolality <275, urine osmolality >100, urine sodium >40, clinical euvolemia, and normal thyroid and adrenal function.**"
- The single most examinable therapeutic sentence: "**0.9% saline corrects hypovolemic hyponatremia but worsens SIADH, because the infused sodium is quantitatively excreted while the elevated ADH retains the water.**"
- "**Fluid restriction is indicated in euvolemic and hypervolemic hyponatremia and is contraindicated in hypovolemic hyponatremia.**"
- State the numbers precisely: **3% saline = 513 mEq/L; infuse at 0.5–1.0 mL/kg/h; check sodium 2-hourly; stop if the rise exceeds 0.5 mEq/L/h; target 4–8 mEq/L in 24 h.**
- Name the ODS risk factors as a list — **sodium <105 mEq/L, hypokalaemia, alcoholism, malnutrition, advanced liver disease** — and say ODS is **delayed in onset**.
- A drawn **algorithm/flow chart** reliably attracts marks in this question. Draw the three volume-status branches with urine sodium as the discriminator.

Extra Points to Remember (Recurring Topic)

- **Cerebral salt wasting versus SIADH** is a favourite viva question. Both follow CNS injury and both have high urine sodium; the difference is **volume status** — cerebral salt wasting is **hypovolemic with hypotension** and needs **salt and volume replacement**, whereas SIADH is euvolemic and needs **fluid restriction**. Getting this the wrong way round is dangerous. Note that a 2014 study cited in Pathy's found post-subarachnoid haemorrhage hyponatremia was actually due to **SIAD and glucocorticoid deficiency, not cerebral salt wasting**.
- **Why urea is excluded from effective osmolality:** it crosses cell membranes freely and therefore causes no water shift. Write **"effective osmolality = 2 × Na + glucose/18"** and say why.
- **The "60% idiopathic" statistic** is worth quoting: in an exhaustive evaluation of 50 elderly inpatients with mild-to-moderate hyponatremia, no classical SIADH cause was found in 60%; the association was with **pneumonia and medication**. This justifies drug review as the highest-yield step in the elderly.
- **Correction is not just biochemical.** Renneboog's prospective study showed measurable improvement in **tandem gait on a calibrated platform** and in **attention reaction time (673 → 615 msec)** after correcting sodium from 128 to 138 mEq/L. Indian CGA data (AIIMS Rishikesh, 2023) add improvement in **HMSE, Barthel ADL, Timed Up and Go and handgrip strength**.
- **Hyponatremia and bone.** There is a plausible direct mechanism — Verbalis showed in a rat SIADH model that chronic hyponatremia causes **substantial reduction in bone mass through osteoclastic resorption**. Multiple meta-analyses link hyponatremia to osteoporosis and fracture. In a fracture-clinic setting, always check the sodium.
- **Indian framing sentence:** "In Indian geriatric practice, the commonest reversible causes are **thiazides, SSRIs and gastroenteritis**; severe hyponatremia (<125 mEq/L) accounts for over half of elderly admissions in some series and typically presents as **generalised weakness or disorientation** rather than seizures, so a low threshold for checking serum sodium in any acutely confused older patient is essential."
- **A safe closing sentence when the examiner probes the correction-rate controversy:** "Recent large cohorts suggest osmotic demyelination is rarer than previously believed and that overly slow correction may itself be harmful, but current guidelines continue to recommend **6–8 mEq/L per 24 hours**, and I would not deviate from that outside a specialist setting."

Rapid Revision Grid

NIGHT-BEFORE APPENDIX · NUMBERS, THRESHOLDS, FORMULAE, TRIALS

1. Numerical Thresholds

Parameter	Value
Normal serum osmolality	285 ± 2 mOsm/kg
ADH release threshold	280 mOsm/kg
Thirst threshold	5–10 mOsm/kg **above** the ADH threshold
Hyponatremia	<135 mEq/L; clinically significant <130; severe <120
True hypo-osmolality	<275 mOsm/kg
Hypernatremia	>145 mEq/L
Inappropriately concentrated urine	Urine osmolality >100 mOsm/kg
Urine sodium in SIADH	>40 mEq/L (may approach 100)
Urine sodium in low EABV	<20 mEq/L
Total body water	60% of weight <75 y; **50% >75 y**
GFR at which fluid homeostasis fails	30–50 mL/min
Dehydration — textbook	Weight loss ≥3% body mass, or >1 kg/day
Dehydration — ESPEN (current)	Measured osmolality ≥300 mOsm/kg; calculated osmolarity >295 mOsm/L
Daily fluid recommendation	~1.5 L/day
Obligatory free water loss	0.5–1.5 L/day

2. Correction Rates — Learn These Verbatim

Situation	Target	Complication if exceeded
Hyponatremia	Raise Na by **6–8 mEq/L / 24 h** (4–8 in the acute phase)	**Osmotic demyelination syndrome** — pontine and extrapontine, delayed onset
Hypernatremia	Lower Na by **<10 mEq/L / 24 h**	**Cerebral oedema**
Monitoring — hyponatremia	Serum Na **2-hourly**; stop if rise >0.5 mEq/L/h	—
Monitoring — hypernatremia	Serum Na **4-hourly**	—

3. Formulae

Serum osmolality = 2[Na] + glucose/18 + BUN/2.8 (mg/dL)

Effective serum osmolality = 2[Na] + glucose/18 (urea excluded — freely permeable)

Water replacement / 24 h (hypernatremia) = TBW × $[(Na_{goal}/Na_{current}) - 1]$ + ongoing losses
TBW = 0.6 × wt if <75 y; 0.5 × wt if >75 y; $Na_{goal} = Na_{current} - 10$

Calculated osmolarity (ESPEN, dehydration screening) = 1.86 × (Na + K) + 1.15 × glucose + urea + 14 (mmol/L)

4. Fluid Selection

Fluid	Sodium	Use
3% saline	**513 mEq/L**	Severe symptomatic hyponatremia. Bolus 100–150 mL (2–4 mL/kg) over 20–40 min, or infusion 0.5–1.0 mL/kg/h
0.9% saline	**154 mEq/L**	Hypovolemic hyponatremia (1–2 L trial). **Worsens SIADH.** Does not lower osmolality in hypernatremia
0.45% saline	77 mEq/L	Hypernatremia with combined water and solute loss (50% free water)
5% dextrose	0	Hypernatremia from pure water loss; also used to re-lower after overcorrection

Oral salt	—	4–12 g/day in divided doses, hypovolemic hyponatremia

5. Named Trials and Landmark Studies

Name	Message
SALT-1 / SALT-2	Tolvaptan in 448 subjects — raised sodium; only a small SF-12 quality-of-life gain, significant in the US cohort only
INSIGHT	Tolvaptan in 107 SIADH patients — sodium 129 → 136 mEq/L but **no improvement** in the primary cognitive endpoint
Renneboog et al.	Mild chronic hyponatremia causes falls, unsteadiness and attention deficits; gait and reaction time improve on correction
Verbalis (rat model)	Chronic hyponatremia causes osteoporosis via osteoclastic bone resorption
SALSA (JAMA Intern Med 2021)	Bolus vs continuous 3% saline — both safe and effective; **bolus needed less relowering therapy**
MacMillan (NEJM Evid 2023)	22,858 admissions — ODS only 0.05%; **7 of 12 cases had no rapid correction**
Seethapathy (NEJM Evid 2023)	3,274 patients — **slower correction linked to higher mortality** and longer stay
Ayus meta-analysis (JAMA Intern Med 2025)	11,811 patients — correction 8–10 mEq/L/24 h: lower in-hospital (OR 0.67) and 30-day (RR 0.55) mortality, no excess ODS
Refardt (JASN 2023)	**Empagliflozin 25 mg/day** crossover trial in chronic SIAD — raised sodium, possible MoCA improvement
ERA treatment standard 2024	3% saline **100–150 mL bolus preferred** for symptomatic hyponatraemia
AIIMS Rishikesh (BMC Geriatrics 2023)	Indian CGA data — HMSE, Barthel ADL, TUG and handgrip all **improve** with sodium correction

6. One-Line Volume-Status Discriminator

Type	Clue	Urine Na	Treatment
Hypovolemic	Orthostatic hypotension, diuretics, GI losses	**<20 mEq/L**	0.9% saline 1–2 L; oral salt. **Never fluid restrict**
Euvolemic (SIADH)	No oedema, no postural drop; drugs, pneumonia, CNS or lung lesion	**>40 mEq/L**	Fluid restriction 500–1500 mL/day; urea, vaptan, empagliflozin
Hypervolemic	Oedema — heart failure, cirrhosis, nephrotic syndrome	Usually low	Fluid restriction + treat underlying disease; loop diuretic

Final Three-Sentence Summary

- **Physiology:** ageing brings reduced thirst, reduced urinary concentrating ability, reduced free water excretion and a fall in total body water from 60% to 50% after age 75 — a narrow margin in both directions.
- **Diagnosis:** confirm true hypo-osmolality, classify by volume status using urine sodium, then review every drug and exclude hypothyroidism and adrenal insufficiency before diagnosing SIADH.
- **Treatment:** correct according to neurocognitive symptoms, not the number — up by 6–8 mEq/L per 24 hours in hyponatremia, down by less than 10 mEq/L per 24 hours in hypernatremia.

MD GERIATRIC MEDICINE

Examination Revision Series — Volume: Endocrinology, Metabolic & Reproductive Health

High-Yield Structured Answers | 11 Questions

Primary textbook sources: Pathy's Principles and Practice of Geriatric Medicine, 6th edition (Sinclair, Morley, Vellas, Cesari, Munshi) · Kane's Essentials of Clinical Geriatrics

Evidence supplement: ATA 2025 · ETA 2023/2024/2025 · ADA Standards of Care 2026 · KDIGO 2024 · NICE NG23 · ESE 2025 · EAU 2025 · Princeton IV 2024 · Endocrine Society

Units: Indian conventional throughout — calcium mg/dL, sodium mEq/L, creatinine mg/dL, glucose mg/dL, testosterone ng/dL, haemoglobin g/dL

Format: High-yield numbered points → Latest Updates (2023–present) → Current Practice Update → Exam One-Liners

Prepared for Dr. Sudarsan — night-before-examination revision format

Contents

1	Treatment of hypothyroidism	**15 marks**
2	Subclinical hypothyroidism	**7 marks**
3	Solitary thyroid nodule	**7 marks**
4	Evaluation and management of hypoglycaemia	**15 marks**
5	SGLT2 inhibitors in type 2 diabetes mellitus	**15 marks**
6	Hypocalcaemia	**7 marks**
7	Hormone replacement therapy in geriatric patients **(RECURRING)**	**15 marks**
8	Gynaecomastia	**7 marks**
9	Andropause	**7 marks**
10	Causes of erectile dysfunction and treatment options	**15 marks**
11	Hirsutism and baldness in elderly women	**7 marks**

*Colour key — **blue**: core textbook keyword | **red**: danger / contraindication / examiner trap | **green**: newer evidence | **amber**: numerical anchor.*

QUESTION 1 — THYROID

Treatment of Hypothyroidism

15 MARKS

High-Yield Points (Textbook Core)

A. Definition and aims

1. Hypothyroidism is deficient thyroid hormone action. In old age the commonest cause is **autoimmune (Hashimoto's) thyroiditis**, followed by earlier thyroid surgery or radioiodine given for thyrotoxicosis.
2. The three aims of treatment are: relieve symptoms and signs, **normalise TSH to an age-appropriate range**, and **avoid over-treatment**.
3. Presentation in the elderly is atypical — memory loss, lethargy, constipation, cold intolerance, fatigue, heart failure, depression, weight gain. Absence of these symptoms does **not** rule out hypothyroidism, so a high index of suspicion is needed.

B. Drug of choice

4. **Levothyroxine (LT4) sodium**, orally, once daily, on an empty stomach 30–60 minutes before breakfast.
5. Separate by at least 4 hours from **calcium, iron, proton pump inhibitors, soya, bile acid sequestrants**, which impair absorption.
6. The full replacement dose in young adults is **1.6 µg/kg/day**. In older people the requirement is **lower** — because of reduced lean body mass and the physiological age-related fall in T4 production.

C. Starting dose and titration (the key exam point)

7. Start **very low: 12.5–50 µg/day**, particularly if there is coronary artery disease, arrhythmia or multiple cardiac risk factors.
8. Increase **slowly, every 6–8 weeks**, aiming to reach the replacement dose only after **4–6 months**.
9. Thyroid hormone increases myocardial oxygen consumption — rapid replacement can precipitate **angina, myocardial infarction or cardiac arrhythmia**.
10. In heart failure or established rhythm disturbance, a deliberately **sub-replacement dose** may be the correct choice to prevent ischaemic symptoms.
11. In older patients there is a **narrow window between TSH suppression and the substitution dose**, due to increased thyrotroph sensitivity to negative feedback by T4.

D. Targets and monitoring

12. The TSH target is **shifted upward with age**: approximately **4–6 mIU/L at 70–80 years** and **6–7 mIU/L above 80 years**, especially where there is risk of arrhythmia or fragility fracture.
13. Check TSH **every 3 months during titration**; once stable, **once or twice yearly** with small dose adjustments.

E. Special situations

14. **Central (secondary) hypothyroidism** — titrate to **free T4 in the upper half of the normal range**, **never to TSH**. The European Thyroid Association has issued 34 recommendations for this uncommon but difficult condition.
15. **Myxoedema coma** — IV levothyroxine (± liothyronine), **IV hydrocortisone until adrenal insufficiency is excluded**, passive rewarming, ventilatory and fluid support, and treatment of the precipitant.
16. **Non-thyroidal illness** — do not start levothyroxine on abnormal tests during acute illness; recheck after recovery. Treat only if there is real evidence of hypothyroidism (for example TSH >20 mIU/L with low free T4, or clear history and signs).

F. Hazards of over-treatment and review of indication

17. Over-replacement causes iatrogenic thyrotoxicosis → **atrial fibrillation, osteoporosis and fragility fracture**, anxiety, muscle wasting, insomnia.
18. Warn the patient and caregiver to report new **angina, breathlessness, confusion or insomnia**.
19. Many older patients began levothyroxine when younger for inappropriate or transient indications and continue without review. In one nursing-home study, thyroid hormone was **successfully withdrawn in half the residents** — always re-examine the indication.
20. Poor adherence is the commonest cause of a persistently raised TSH despite an apparently adequate dose.

Latest Updates (2023–Present)

- **ETA 2025 guideline on levothyroxine monotherapy** — emphasises individualisation; recognises **liquid and softgel LT4 formulations** where absorption is impaired (malabsorption, PPI/coffee interference, post-bariatric surgery).
- **Age-specific reference intervals** (Jansen et al., *Thyroid* 2024) — TSH rises physiologically with age, particularly after 50 in women and 60 in men. Applying a single adult range **over-diagnoses hypothyroidism in the elderly**.
- **Over-treatment is common** — Baltimore Longitudinal Study of Aging (2023) and subsequent real-world data show 10–20% of older adults on LT4 are over-treated; iatrogenic thyrotoxicosis is significantly more frequent in **women** (Adams & Mammen, *Thyroid* 2025).
- **Deprescribing** — structured levothyroxine discontinuation trials are running in community-dwelling adults ≥60 on stable doses ≤150 µg with TSH <10 mIU/L; roughly half are expected to remain euthyroid off treatment.
- **T4/T3 combination therapy** — a pooled analysis of eleven randomised trials (*JCEM* 2024) found a consistent **patient preference** for combination therapy or desiccated thyroid extract, but no reproducible objective benefit. It remains a second-line option for persistently symptomatic patients on adequate LT4, and is **not recommended routinely in the elderly** because of arrhythmia risk.

Current Practice Update

Issue	Textbook position	Current practice (2023–present)
TSH reference range	Single adult range; higher target suggested in the very old	Formal age-specific reference intervals now published and increasingly adopted
LT4 formulation	Standard tablet	Liquid/softgel preparations recognised for malabsorption and drug interference
Duration of therapy	Review indication; withdrawal possible in many nursing-home residents	Deprescribing now an active, trial-supported strategy in older adults with TSH <10 mIU/L
T4 + T3	Not advocated	Still not routine; patient-preference signal acknowledged, objective benefit unproven

Exam One-Liners

- Start low, go slow: **12.5–50 µg**, increase every **6–8 weeks**, full dose by **4–6 months**.
- Elderly replacement dose is **below 1.6 µg/kg/day** because lean body mass falls.
- Target TSH **4–6 mIU/L at 70–80**, **6–7 mIU/L above 80**.
- Central hypothyroidism → titrate to **free T4**, not TSH.
- Over-treatment → **atrial fibrillation + osteoporotic fracture**.
- Myxoedema coma → give **hydrocortisone before or with** thyroxine.

QUESTION 2 — THYROID

Subclinical Hypothyroidism

7 MARKS

High-Yield Points

1. **Definition:** an elevated serum TSH with a **normal or low-normal free T4**. It is a biochemical, not a clinical, diagnosis.
2. **Prevalence:** 4–10% of the general population, rising to **up to 20% in women over 60**. Very common because of widespread sensitive TSH assays.
3. **Confirm before acting:** a TSH above 4.5 mIU/L must be repeated at 1–3 months, and thereafter every 6–12 months if the patient is asymptomatic.
4. **Evaluate for a cause:** past radioiodine, previous thyroid surgery, goitre, family history of thyroid disease; check **anti-thyroid peroxidase antibodies** and a lipid profile.
5. **Natural history:** progression to overt hypothyroidism is **around 5% per year (range 3–20%)**, higher with goitre and/or positive antibodies. Antibodies help predict progression but rarely change management.
6. **When to treat:** if TSH rises above **10 mIU/L**, start levothyroxine. Between 4.5 and 10 mIU/L, an individual *ex adiuvantibus* trial may be considered for subtle symptoms.
7. **TRUST trial** (Stott et al., NEJM 2017) — 737 adults aged ≥65 (mean 74.4 years, baseline TSH 6.40 mIU/L). Levothyroxine lowered TSH to 3.63 vs 5.48 mIU/L on placebo but produced **no improvement in Hypothyroid Symptoms score, Tiredness score, cognition, blood pressure, weight, Barthel index or IADL**.
8. **The cardiovascular link is age-dependent:** subclinical hypothyroidism appears detrimental in young and middle-aged people (Whickham re-analysis showed excess coronary events over 20 years), but is **harmless or possibly protective in the oldest old**. In studies of people over 73 and over 85, high TSH and/or low free T4 were associated with **lower all-cause and cardiovascular mortality**.
9. **Leiden 85-plus study:** 12% of 85-year-olds had hypothyroidism (7% overt, 5% subclinical); none of the subclinical group progressed by age 88, and a raised TSH predicted **slower decline in instrumental ADL**.
10. **Conclusion for the elderly:** do not treat minor TSH elevations in the oldest old — the risks (atrial fibrillation, fracture) outweigh benefit. Decide on **comorbidity, frailty and symptom burden**, not on a TSH cut-off alone.

Latest Updates & Current Practice (2023–Present) — 3 Points

- **Age-specific TSH reference intervals** (*Thyroid* 2024) are reducing the number of older adults labelled with subclinical hypothyroidism at all — many are simply normal for their age.
- **Systematic review and meta-analysis** (Holley et al., *Systematic Reviews* 2024, 6 studies, 3853 participants) found **no clear cardiovascular or bone-health benefit** from levothyroxine in older people with subclinical hypothyroidism — consistent with TRUST and with the pooled TRUST/IEMO 80-plus data.
- **Current practice therefore diverges from the textbook only in emphasis:** where Pathy's leaves the 4.5–10 mIU/L band open to an individual trial, guidelines now favour **watchful monitoring without treatment** in those over 70–80, and are moving towards active **deprescribing** of levothyroxine started for this indication.

Exam One-Liners

- Raised TSH + normal free T4 = subclinical hypothyroidism.
- Treat above **10 mIU/L**; individualise **4.5–10 mIU/L**; do not treat the very old.
- **TRUST** = no symptomatic benefit in those aged ≥65.
- In the oldest old, a high TSH may actually be **protective**.

QUESTION 3 — THYROID

Solitary Thyroid Nodule

7 MARKS

High-Yield Points

1. **Definition and significance:** a discrete lesion within the thyroid, radiologically distinct from surrounding parenchyma. **About 95% are benign**, but every nodule deserves evaluation because a few harbour malignancy.
2. **Epidemiology:** palpable in 3–7% of adults, but present in **around 50% on ultrasound or autopsy**. Frequency and goitre size increase with age, and nodular goitre is commoner in women and in iodine-deficient areas. In the Pescopagano (iodine-deficient) survey, goitre prevalence rose from 30% in young adults to **75% in those aged 55–65**.
3. **Key principle:** the risk of malignancy is **the same** for a solitary nodule and for a nodule within a multinodular goitre.
4. **Red flags requiring urgent referral:** rapid growth over a few weeks, **stridor, hoarseness (recurrent laryngeal nerve), cervical lymphadenopathy**, fixation, prior neck irradiation.
5. **First-line investigation:** serum **TSH plus high-resolution ultrasound**. Ultrasound is not a population screening test, but any palpable nodule must be scanned.
6. **If TSH is low or suppressed** → radioisotope scan. A **"hot" (autonomous) nodule is virtually never malignant** and is managed as hyperthyroidism — ^{131}I is the treatment of choice in old age, especially with cardiovascular disease. A **"cold"** nodule requires FNAC.
7. **If TSH is normal or high** → risk-stratify by ultrasound using **EU-TIRADS**: category 2 (pure cyst / spongiform) ≈0%; category 3 (ovoid, smooth, iso/hyperechoic) 2–4%; category 4 (mildly hypoechoic) 6–17%; **category 5 (irregular shape or margins, microcalcification, marked hypoechogenicity) 26–87%**.
8. **FNAC is the cornerstone of diagnosis**, and accuracy improves with ultrasound guidance. Perform for nodules **≥10 mm** with irregular margins, chaotic intranodular vascularity, taller-than-wide shape, or microcalcification; for nodules <10 mm only if clinical or ultrasound features are suspicious. **No ultrasound finding alone is diagnostic.**
9. **Limitation of FNAC:** inadequate or indeterminate results in **10–25%**; surgery is then usually advised for diagnosis, although most such nodules prove benign.
10. **Follow-up and treatment:** benign nodule → ultrasound at 12 months, then at 2–5 year intervals. Repeat FNA only for **>50% volume growth, ≥20% increase in two dimensions**, new suspicious features, or new symptoms. **Routine TSH-suppressive levothyroxine is NOT recommended** — harm outweighs benefit and it can cause exogenous thyrotoxicosis with cardiac and bone consequences. Malignant or suspicious → surgery; large compressive non-toxic goitre → surgery or radioiodine.

*Supporting datum: in a prospective Italian multicentre study of 992 patients (1567 benign nodules) followed 5 years, only 11.1% grew, thyroid cancer was found in 0.3%, and age ≥60 was associated with a **lower** risk of nodule growth.*

Latest Updates & Current Practice (2023–Present) — 3 Points

- **ETA 2023 thyroid nodule guideline:** minimally invasive techniques are now **first-line for symptomatic benign nodules** — ethanol ablation for cystic/predominantly cystic lesions, and thermal ablation (radiofrequency, microwave, laser, HIFU) for solid nodules, giving **57–77% volume reduction sustained at 5 years** without general anaesthesia. The ATA issued a 2023 statement on safe performance and training for these techniques.
- **ATA 2025 guidelines** now separate differentiated thyroid cancer from nodule management: **lobectomy is preferred over total thyroidectomy for unilateral cT1N0M0 tumours ≤2 cm**; active surveillance and percutaneous ablation are formally accepted alternatives for papillary microcarcinoma; molecular diagnostics have an expanded role in Bethesda III/IV nodules; and TSH suppression has been simplified to a binary target ("within" versus "below" the normal range).
- **Relevance to the older patient:** these de-escalated pathways (surveillance, lobectomy, ablation) are **particularly suited to frail elderly patients**, in whom the competing risks of thyroidectomy, hypocalcaemia and lifelong levothyroxine often outweigh the risk from an indolent tumour.

Exam One-Liners

- **TSH + ultrasound first** — scan only if TSH is low.
- Hot nodule = almost never cancer; cold nodule = FNAC.
- **FNAC is the cornerstone**; ultrasound stratifies but never diagnoses.
- Solitary nodule and MNG carry the **same** malignancy risk.
- Never use suppressive levothyroxine for a benign nodule.

QUESTION 4 — DIABETES

Evaluation and Management of Hypoglycaemia

15 MARKS

High-Yield Points (Textbook Core)

A. Definition and classification

1. Hypoglycaemia is a plasma glucose low enough to cause symptoms or harm; the conventional biochemical threshold is **<70 mg/dL**.
2. **Three levels:** Level 1 (alert value) <70 mg/dL; Level 2 (clinically significant) **<54 mg/dL**; **Level 3 (severe) — any glucose with cognitive impairment requiring external assistance**.
3. **Whipple's triad** confirms true hypoglycaemia: compatible symptoms + a documented low plasma glucose + relief of symptoms when glucose is raised.
4. Hypoglycaemia is the **critical limiting factor** in achieving glycaemic targets in older adults with diabetes.

B. Why older people are especially vulnerable

5. **Patient factors:** polypharmacy, frailty, high comorbidity burden, cognitive impairment, renal disease, low body mass, low HbA1c, erratic or reduced food intake, inability to self-administer treatment safely.
6. **Drug factors: sulphonylureas — particularly long-acting glibenclamide** — and insulin, especially twice-daily premixed and basal-bolus regimens.
7. **Non-diabetic causes to remember:** sepsis, hepatic failure, adrenal insufficiency, hypopituitarism, malnutrition and alcohol excess, insulinoma, non-islet-cell tumour hypoglycaemia (IGF-2 mediated), post-gastrectomy dumping, quinolones, quinine, and beta blockers which mask warning symptoms.

C. Clinical features

8. **Autonomic:** palpitations, tremor, sweating, hunger, anxiety.
9. **Neuroglycopaenic:** behavioural change, confusion, focal neurological deficit, seizure, coma.
10. **Autonomic warning symptoms are blunted with age → hypoglycaemia unawareness**. Presentation is often atypical — falls, delirium, “dizziness”, or a stroke mimic — and the symptomatic threshold in older people may differ from the conventional cut-off.

D. Evaluation

11. Confirm with a **laboratory glucose**, not a meter reading alone; document Whipple's triad.
12. In a person already on glucose-lowering drugs, **the medication review IS the evaluation** — no endocrine work-up is needed. Establish the pattern (nocturnal, pre-meal, post-exercise), timing relative to doses, renal function, and recent weight or appetite loss.
13. In a **non-diabetic**, take a **critical sample during the episode**: insulin, C-peptide, proinsulin, beta-hydroxybutyrate, sulphonylurea screen, cortisol.
    - High insulin + high C-peptide → **insulinoma or sulphonylurea** (differentiated by the drug screen)
    - High insulin + **low** C-peptide → **exogenous insulin** administration
    - Low insulin + low ketones → **IGF-2 mediated / non-islet-cell tumour**
    - Low insulin + high ketones → alcohol, starvation, cortisol deficiency

E. Acute management

14. **Conscious and able to swallow** — the “rule of 15”: give **15–20 g fast-acting carbohydrate** (glucose tablets or gel, 3–4 teaspoons of sugar in water, 150 mL fruit juice), recheck at 15 minutes, repeat up to three times, then give a **long-acting carbohydrate snack** to prevent recurrence.
15. **Unable to swallow or unconscious: IV 25% dextrose 75–100 mL** (or 10% dextrose 150–200 mL) via a large vein, or **IM/SC glucagon 1 mg**. **Glucagon is ineffective in alcoholic, malnourished or hepatic glycogen-depleted patients.**
16. **Sulphonylurea-induced hypoglycaemia is prolonged and relapsing** — admit, run a 10% dextrose infusion, observe for at least 24–48 hours; octreotide may be used for refractory cases. Never discharge such a patient after a single correction.
17. Always identify and treat the **precipitant**: missed meal, sepsis, worsening renal function, deliberate or accidental dose error, alcohol.

F. Prevention — the real answer in geriatrics

18. **Relax targets by health status:** healthy older adult HbA1c <7.5%; complex/intermediate health <8.0%; very complex or poor health <8.5%. Frail patients per EDWPOP: **7.6–8.5%**. **Avoid HbA1c below 7% in the frail.**
19. **Deintensify and deprescribe:** replace glibenclamide with gliclazide or a glinide if meals are irregular; simplify basal-bolus to basal-only insulin; prefer agents with no intrinsic hypoglycaemia risk — metformin, DPP4 inhibitors, SGLT2 inhibitors, GLP-1 receptor agonists.
20. Re-evaluate the entire treatment plan after **any Level 2 or Level 3 event**; assess frailty, functional status and life expectancy at every review; educate patient and carer, give sick-day rules, and check the capacity to self-administer treatment.

Latest Updates (2023–Present)

- **ADA Standards of Care 2026** — **real-time continuous glucose monitoring (CGM) is now recommended for older adults with type 1 diabetes and for older adults with type 2 diabetes on insulin**, to improve glycaemic outcomes, reduce hypoglycaemia and reduce treatment burden. CGM is advised at diabetes onset and any time thereafter for anyone on insulin, on non-insulin agents that can cause hypoglycaemia, or where CGM aids management.
- **CGM-based hypoglycaemia targets:** time below 70 mg/dL **<4% (and <1% for older adults)**; time below 54 mg/dL **<1%**. **Failure to meet these should itself trigger deintensification** — a target-driven trigger not present in the textbook.
- **Intermittently-scanned CGM has been removed** from the ADA technology table — real-time CGM is now the recognised standard.
- CGM reduces hypoglycaemic episodes by **more than 50%** versus standard monitoring in adults over 65; ADA 2026 Scientific Sessions data (SAGE) show that pairing CGM with structured pharmacist-led education converts detection into behaviour change and facilitates **deprescribing of high-risk agents**.
- **Deintensification is now formally endorsed** for both glucose and blood pressure in older adults, including in palliative care, with growing safety and efficacy evidence.
- **JBDS-IP guidance** (Sinclair et al.) on inpatient care of the frail older adult with diabetes recommends **frailty-adjusted inpatient glycaemic targets**; audit work in 2025 confirms this reduces inpatient hypoglycaemia.
- Level 2 or 3 hypoglycaemia should also prompt **annual screening for fear of hypoglycaemia and diabetes distress**, with referral to behavioural therapy if unresolved (new for 2026).

Current Practice Update

Issue	Textbook position	Current practice (2023–present)
Detection	Symptom report + capillary glucose; hypoglycaemia unawareness recognised but hard to quantify	Real-time CGM recommended for all older adults on insulin; detects asymptomatic and nocturnal events
Metric used	HbA1c and episode counts	Time below range <1% (<70 mg/dL) for older adults, in addition to HbA1c
Trigger to change therapy	Clinical judgement after admissions	Any Level 2/3 event, or failure to meet CGM time-below-range goals
Deintensification	“Now an essential action a clinician must consider”; little guiding evidence	Evidence base established; explicit algorithms for insulin simplification and palliative de-escalation
Psychological impact	Not systematically addressed	Annual screening for fear of hypoglycaemia and diabetes distress recommended

Exam One-Liners

- **<70 / <54 / severe** = Levels 1, 2, 3.
- Whipple's triad confirms it; the **critical sample** is taken *during* the episode.
- Rule of 15: **15 g carbohydrate, recheck at 15 minutes**.
- Sulphonylurea hypoglycaemia → **admit and infuse dextrose for 24–48 hours**.
- Glucagon fails in the alcoholic and the malnourished.
- The definitive treatment of recurrent hypoglycaemia is **deintensification, not more glucose**.

QUESTION 5 — DIABETES / THERAPEUTICS

SGLT2 Inhibitors in Type 2 Diabetes Mellitus

15 MARKS

High-Yield Points (Textbook Core)

A. Agents and mechanism

1. **Agents:** empagliflozin, dapagliflozin, canagliflozin, ertugliflozin, bexagliflozin; also marketed as fixed-dose combinations with metformin and with DPP4 inhibitors.
2. **Mechanism:** inhibit the sodium–glucose co-transporter 2 in the **proximal convoluted tubule**, which normally reabsorbs **about 90% of filtered glucose** → glycosuria and a fall in plasma glucose.
3. The action is **insulin-independent**, so efficacy is preserved despite beta-cell failure and there is **no intrinsic hypoglycaemia risk** — a major advantage in the older patient.

B. Metabolic effects

4. HbA1c fall of about **0.5–1.0%**; **weight loss 2–3 kg**; systolic blood pressure fall of roughly 3–5 mmHg.
5. Beyond glucose lowering: reduced circulating insulin, reduced hepatic steatosis, improved beta-cell function, mild uricosuria, and reduced hyperkalaemia risk.

C. Cardiovascular and renal outcomes (highest-yield section)

6. In the cardiovascular outcome trials, **empagliflozin and canagliflozin significantly reduced MACE** (cardiovascular death, non-fatal MI, non-fatal stroke); **empagliflozin also reduced cardiovascular death, hospitalisation for heart failure and worsening nephropathy**. DPP4 inhibitors, by contrast, showed no cardiovascular benefit.
7. **Heart failure across the whole ejection-fraction spectrum:** HFrEF (DAPA-HF, EMPEROR-Reduced) and HFpEF (EMPEROR-Preserved, DELIVER). Benefit is independent of diabetes status and begins within days of initiation.
8. **Renal protection — the three trials to name:** CREDENCE (type 2 diabetes, eGFR >30, UACR >300 mg/g), DAPA-CKD (eGFR >25, with and without diabetes), and EMPA-KIDNEY (eGFR ≥20, including non-diabetic and non-albuminuric CKD) — EMPA-KIDNEY showed a **28% reduction** in kidney disease progression or cardiovascular death.
9. **Renal benefit is independent of glucose lowering.** Mechanism: restoration of tubuloglomerular feedback with afferent arteriolar constriction → reduced intraglomerular hypertension, plus natriuresis and reduced tubular workload and hypoxia.

D. Practical use in older adults

10. **Initiation threshold: eGFR ≥20 mL/min/1.73 m².** Once started, **continue as eGFR falls** — the glycaemic effect fades but cardiorenal protection persists — down to dialysis.
11. An acute **"eGFR dip" of up to about 10% in the first 2–4 weeks is expected and haemodynamic**; it does not predict worse outcome and is **not a reason to stop the drug**.
12. Assess volume status before starting; **reduce or hold the loop diuretic** in a euvolaemic older patient to avoid dehydration and postural hypotension.
13. Glycaemic efficacy is **diminished in older people with renal impairment**; monitor renal function more frequently in the elderly.

E. Adverse effects and cautions

14. **Genital mycotic infection** (5–10% of women, 3–5% of men), **volume depletion with symptomatic hypotension**, and urinary tract infection — all more frequent above age 75.
15. **Euglycaemic diabetic ketoacidosis** — glucose may be near-normal, so it is easily missed. Precipitants: fasting, sepsis, surgery, very low-carbohydrate diet, insulin dose reduction, alcohol. **Withhold 3 days before elective surgery** (4 days for ertugliflozin).
16. Rare: **Fournier's gangrene** (necrotising fasciitis of the perineum). Canagliflozin carried a lower-limb amputation signal in CANVAS (boxed warning later downgraded to a standard warning).
17. **Pathy's caution in frailty:** consider avoiding an SGLT2 inhibitor in the frail because of **weight loss, dehydration and toe amputation**. This must now be balanced against the strong cardiorenal benefit — see Current Practice Update.
18. **Avoid or contraindicated in:** type 1 diabetes, prior DKA, recurrent genital infection, significant volume depletion, active foot ulceration, and where oral intake is unreliable.

F. Positioning in therapy

19. Metformin remains first-line after lifestyle measures; an SGLT2 inhibitor is a standard second-step choice. However, in the presence of **established atherosclerotic disease, heart failure or CKD, an SGLT2 inhibitor is now indicated irrespective of HbA1c** — it is prescribed for organ protection, not for glucose.
20. Blood pressure and lipid targets must be addressed in parallel: systolic 130–139 mmHg in those over 65, diastolic <80 but not <70 mmHg, with LDL targets graded by cardiovascular risk.

Latest Updates (2023–Present)

- **KDIGO 2024 CKD guideline** — SGLT2 inhibitors are now a **grade 1A foundational therapy** for adults with CKD and eGFR ≥20 mL/min/1.73 m², **with or without diabetes**, alongside RAAS blockade and statins. A weaker (2B) suggestion covers eGFR 20–45 with UACR <200 mg/g.
- **ADA Standards of Care 2026** confirms initiation at eGFR ≥20 in type 2 diabetes with CKD, and positions SGLT2 inhibitors and GLP-1 receptor agonists as complementary organ-protective agents. The **FLOW trial** extended kidney protection to the GLP-1 RA semaglutide.
- **Evidence specifically in older adults** — a 2025 meta-analysis (*Drugs & Aging*) and a 2025 systematic review of nine trials in those aged ≥65 found benefit fully preserved, including in the **≥75 year subgroup (composite of heart-failure hospitalisation and cardiovascular death, HR ≈0.71)**, without an excess of serious adverse events, fractures or amputations.
- Product labelling now explicitly advises **more frequent renal monitoring in the elderly** and notes higher rates of volume depletion and urinary tract infection in patients aged ≥75.
- Label expansions have been granted for dapagliflozin and empagliflozin in **chronic kidney disease and heart failure regardless of diabetes status**; ESC guidance includes them in acute heart-failure pathways.

Current Practice Update

Issue	Textbook position	Current practice (2023–present)
Indication	A glucose-lowering agent, used at the second step	Foundational organ-protective therapy, given irrespective of HbA1c in ASCVD, HF or CKD — and in CKD/HF **without** diabetes
Renal threshold	Caution in renal impairment; efficacy diminished	Initiate at eGFR ≥20; continue to dialysis; the early eGFR dip is expected and benign
Use in frailty	"Consider avoiding" because of weight loss, dehydration, amputation	Not a blanket contraindication — benefit preserved at ≥75 years; manage risk by assessing volume status, reducing diuretics and giving sick-day rules
Heart failure	Empagliflozin reduces HF hospitalisation in diabetes	Benefit proven across HFrEF and HFpEF, independent of diabetes, apparent within days
Amputation	Cited as a class concern	Signal was canagliflozin-specific and not consistently replicated post-marketing

Exam One-Liners

- Blocks SGLT2 in the **proximal tubule**, which handles **90%** of glucose reabsorption.
- Insulin-independent → **no hypoglycaemia**; weight and blood pressure both fall.
- Name the renal trio: **CREDENCE, DAPA-CKD, EMPA-KIDNEY**.
- Start at **eGFR ≥20**; do not stop when eGFR falls.
- The early **eGFR dip is haemodynamic** — keep going.
- Beware **euglycaemic DKA** — stop 3 days before surgery.
- Fournier's gangrene is the rare but exam-favourite adverse effect.

QUESTION 6 — METABOLIC BONE / ELECTROLYTES

Hypocalcaemia

7 MARKS

High-Yield Points

1. **Definition:** corrected serum calcium below **8.5 mg/dL** (normal range 8.5–10.5 mg/dL). Always correct for albumin or measure ionised calcium — hypoalbuminaemia is very common in the frail elderly and causes **pseudo-hypocalcaemia**.
   Corrected Ca (mg/dL) = measured Ca + 0.8 × (4.0 − serum albumin in g/dL)
2. **Causes with LOW or inappropriately normal PTH (hypoparathyroidism):** post-surgical after thyroidectomy or parathyroidectomy (**the commonest, ~75–90% of chronic cases**), autoimmune, infiltrative (haemochromatosis, Wilson's, metastasis), radiation, and **hypomagnesaemia**, which impairs both PTH secretion and PTH action.
3. **Causes with HIGH PTH (secondary hyperparathyroidism): vitamin D deficiency** — extremely common in Indian older adults; chronic kidney disease; malabsorption; hungry bone syndrome after parathyroidectomy; acute pancreatitis; rhabdomyolysis and tumour lysis syndrome (hyperphosphataemia); massive blood transfusion (citrate chelation); and drugs — bisphosphonates, denosumab, cinacalcet, phenytoin, foscarnet, proton pump inhibitors.
4. **Clinical clue:** suspect vitamin D deficiency in any older person with a **borderline-low calcium and a raised alkaline phosphatase**.
5. **Clinical features — neuromuscular irritability:** perioral and acral paraesthesia, muscle cramps, carpopedal spasm, tetany, **laryngospasm and bronchospasm**, seizures, confusion. Chronic hypocalcaemia causes cataract, basal ganglia calcification, papilloedema, dry skin, brittle nails and extrapyramidal features.
6. **Signs and ECG: Chvostek's sign** (facial twitch on tapping the facial nerve — falsely positive in up to 10% of normals) and **Trousseau's sign** (carpal spasm after cuff inflation above systolic pressure for 3 minutes) — Trousseau's is the more specific. ECG shows a **prolonged QTc** with risk of torsades de pointes.
7. **Investigations:** corrected and/or ionised calcium, **magnesium**, phosphate, creatinine, alkaline phosphatase, **intact PTH**, 25(OH) vitamin D, and 24-hour urinary calcium. High phosphate + low PTH = hypoparathyroidism; low phosphate + high PTH = vitamin D deficiency.
8. **Acute symptomatic hypocalcaemia** (tetany, seizure, laryngospasm, prolonged QT): **IV 10% calcium gluconate 10–20 mL diluted, over 10–20 minutes with cardiac monitoring**, followed by an infusion delivering roughly 1 mg/kg/hour of elemental calcium. **Correct magnesium first — calcium will not respond while the patient is hypomagnesaemic.** Prefer gluconate to chloride peripherally (chloride is sclerosant).
9. **Chronic management:** oral **elemental calcium 1–2 g/day in divided doses** plus **active vitamin D (calcitriol 0.25–1 µg/day)** in hypoparathyroidism, or plain cholecalciferol where the problem is simple vitamin D deficiency. **Target calcium at the LOW end of normal (8.0–8.5 mg/dL)** — chasing a normal calcium in hypoparathyroidism produces hypercalciuria, nephrocalcinosis and renal failure, because PTH-driven renal calcium reabsorption is absent. A thiazide with a low-salt diet reduces hypercalciuria.
10. **In the older patient specifically:** stop the offending drug, treat malabsorption and renal disease, and ensure **800–1000 IU/day vitamin D with 700–1200 mg calcium** (target 25(OH)D ≥30 ng/mL) — the same regimen that reduces hip fracture and falls. Monitor calcium, phosphate, creatinine and urinary calcium on treatment.

Latest Updates & Current Practice (2023–Present) — 3 Points

- **Palopegteriparatide (TransCon PTH, Yorvipath)** — the first **long-acting once-daily PTH(1-34) prodrug**, giving continuous physiological PTH exposure over 24 hours (released hormone half-life ≈60 hours). EMA-approved November 2023 and **FDA-approved August 2024** for chronic hypoparathyroidism. The phase 3 **PaTHway** trial normalised serum calcium, phosphate and 24-hour urinary calcium with withdrawal of conventional therapy in the majority, sustained to 3 years, and normalised bone remodelling. rhPTH(1-84) (Natpara) manufacture was **discontinued worldwide at the end of 2024**.
- **Revised ESE guideline (2025) and the Parathyroid Summit best-practice recommendations (2024/2025)** retain **calcium plus active vitamin D as first-line** therapy, and reserve PTH replacement for patients with inadequate control, an unacceptable pill burden, persistent hypercalciuria, or declining renal function. Diagnosis of chronic hypoparathyroidism requires hypocalcaemia with an inappropriately low or normal PTH, and DNA analysis is now recommended in non-surgical cases.
- **Emerging agents: eneboparatide** (selective PTHR1 agonist), an oral PTH analogue, and **encaleret** (a calcilytic for autosomal dominant hypocalcaemia type 1) are in phase 2/3 development — the field is moving from calcium supplementation towards true hormone replacement.

Exam One-Liners

- Correct for albumin: **add 0.8 mg/dL per 1 g/dL fall**.
- **Check and correct magnesium first** — otherwise calcium will not rise.
- Trousseau's is more specific than Chvostek's; the ECG shows a **long QT**.
- Acute: **10% calcium gluconate IV, slowly, monitored**.
- In hypoparathyroidism, aim for calcium at the **low end of normal** to protect the kidney.
- New drug to quote: **palopegteriparatide** — the first true PTH replacement.

QUESTION 7 — REPRODUCTIVE ENDOCRINOLOGY

Hormone Replacement Therapy in the Geriatric Patient

RECURRING TOPIC

15 MARKS

High-Yield Points (Textbook Core)

A. Definition, indications and governing principle

1. **HRT / menopausal hormone therapy** = oestrogen therapy, with a **progestogen added in every woman who still has a uterus** to prevent endometrial hyperplasia and carcinoma.
2. **Licensed indications are two only:** (a) relief of vasomotor and genitourinary symptoms; (b) prevention or treatment of osteoporosis where the risk–benefit balance is favourable. **It is NOT indicated for prevention of cardiovascular disease, dementia or any other chronic condition.**
3. **Governing principle:** the **lowest effective dose, for the shortest time required**, with periodic reassessment; the decision belongs to the informed woman.

B. The "window of opportunity" — the central concept

4. The ideal age range for initiation is **50–60 years, and within 10 years of the menopause**. Beyond this the balance turns unfavourable.
5. Meta-analysis shows women who start HRT **more than 10 years after menopause have higher risks of stroke and venous thromboembolism**. A WHI post-hoc analysis found that women aged **70 or older** starting HRT for persisting vasomotor symptoms had a significantly increased risk of coronary heart disease — with oestrogen alone *and* with combined therapy.

C. Preparations, progestogen choice and route

6. **Oestrogens are of natural origin** (unlike contraceptives): estradiol (oral, transdermal patch or gel, nasal spray, vaginal), estrone, estriol, estetrol. Conjugated equine oestrogens — used in WHI — remain popular in some countries.
7. **Progestogens are not equivalent: medroxyprogesterone acetate carries the cardiovascular and breast-cancer signal**, whereas **natural micronised progesterone appears substantially gentler**.
8. **Route matters: transdermal oestrogen is essentially neutral for VTE risk** because it avoids hepatic first-pass metabolism — the preferred route in older women, in obesity, and in anyone with thrombotic risk.
9. **Alternatives:** tibolone; the SERM–oestrogen combination bazedoxifene with conjugated oestrogens. **Low-dose vaginal oestrogen** for genitourinary syndrome has negligible systemic absorption, may be continued indefinitely, and can be given alongside systemic HRT.

D. Benefits

10. HRT is the **most effective treatment for vasomotor symptoms**, which affect **70–80% of women for an average of about 7 years**; it reverses genitourinary syndrome, improves sleep, mood and quality of life.
11. **Bone:** prevents bone loss and fragility fracture; even the **ultra-low-dose 0.014 mg estradiol patch** preserves BMD, though less than a standard dose. Continuation is recommended for women at high fracture risk in whom alternative agents are unsuitable or poorly tolerated.

E. Major risks (must be enumerated)

12. **Breast cancer:** increased with combined oestrogen + progestogen therapy. Crucially, long-term WHI follow-up showed **oestrogen-only HRT was associated with reduced breast-cancer incidence and mortality** in hysterectomised women — a favourite examiner discriminator.
13. **Venous thromboembolism and stroke** — dose- and route-dependent; oral confers greater risk than transdermal.
14. **Coronary heart disease** when started late (more than 10 years post-menopause, or after 70).
15. **Dementia:** continuous combined oestrogen + progestin started in older postmenopausal women **doubled the risk of all-cause dementia** (WHIMS).
16. **Endometrial carcinoma** with unopposed oestrogen in a woman with an intact uterus.

F. Contraindications

17. Current or past **breast cancer** or high breast-cancer risk; oestrogen-dependent malignancy; **VTE or thromboembolic disease**; active cardiovascular disease; **undiagnosed postmenopausal bleeding**; active liver disease.

G. Continuing HRT beyond age 65

18. The North American Menopause Society position is that the **lowest effective dose may be continued after 65** for persistent hot flushes, quality of life or osteoporosis, provided the clinician has established that benefits outweigh risks after thorough counselling. **HRT should not be stopped on the basis of age alone.** Document the counselling and the woman's wish to continue, and review periodically.
19. This matters practically: Swedish data show **15% of women at 66 and 9% at 72 still experience hot flushes**, women over 80 continue to use HRT, and many are unwilling to discontinue — with quality-of-life and economic implications as more older women remain in the workforce.

H. Non-hormonal alternatives — the default in most women over 60

20. **Lifestyle:** dressing in layers and using a fan, cold food and drink, avoiding hot and spicy food, stopping smoking, reducing caffeine, regular exercise, relaxation techniques (paced respiration, yoga, meditation, massage). **Pharmacological: clonidine** (the only UK-licensed non-hormonal agent, reduces central noradrenergic tone); **paroxetine** (FDA-approved, the most efficacious SSRI for women not on tamoxifen); **venlafaxine** (preferred in breast-cancer survivors taking tamoxifen — it does not interfere with tamoxifen metabolism); gabapentin or pregabalin; and **menopause-specific cognitive behavioural therapy**. Phyto-oestrogen studies are of poor quality.

Latest Updates (2023–Present)

- **Fezolinetant** (NK3-receptor antagonist) — **FDA-approved May 2023**, the first targeted non-hormonal drug for moderate-to-severe vasomotor symptoms. It acts on hypothalamic **KNDy neurons** (kisspeptin/neurokinin B/dynorphin), the mechanism Pathy's identified as the basis of hot flushes. SKYLIGHT 1/2/4 and DAYLIGHT showed roughly **60% reduction** in hot-flush frequency. **FDA (September 2024) and MHRA (April 2025) issued warnings of rare serious liver injury — baseline and periodic liver function tests are mandatory.**
- **Elinzanetant** (first **dual NK1 and NK3** receptor antagonist) — approved in the UK, Canada, Australia and Switzerland in 2025 and by the **FDA on 24 October 2025**. OASIS 1–3 showed a **73.8% reduction** in moderate-to-severe vasomotor symptoms at 12 weeks versus 47% on placebo, with rapid onset and additional benefit for **sleep disturbance** and quality of life — the NK1 component makes it particularly attractive where insomnia dominates. It may be used first-line.
- **NICE NG23 (updated 2024, further revised)** — recommends menopause-specific CBT; recommends **fezolinetant when HRT is unsuitable**; advises **transdermal rather than oral HRT where VTE risk is increased, including BMI over 30 kg/m²**; offers vaginal oestrogen for genitourinary symptoms including in women already on systemic HRT; and states plainly that combined or oestrogen-only HRT should **not** be offered for prevention of cardiovascular disease or dementia.
- **2025 — the FDA removed the boxed warning** from systemic menopausal hormone therapy labels after re-evaluating WHI data in the light of age at initiation, formulation and route. This is a significant regulatory reversal that partially rehabilitates HRT for appropriately selected women.
- **Cochrane review** (19 trials, 40,410 postmenopausal women) confirms HRT is **ineffective for primary or secondary prevention of cardiovascular events** and increases stroke and venous thromboembolic events — the textbook position stands.
- Phase 3 data support neurokinin antagonists in **breast-cancer survivors on endocrine therapy** (OASIS-4), a group in whom HRT is absolutely contraindicated.

Current Practice Update

Issue	Textbook position	Current practice (2023–present)
Non-hormonal options	Clonidine, paroxetine, venlafaxine, gabapentin; phyto-oestrogens poorly evidenced	Two licensed targeted agents — **fezolinetant** (NK3) and **elinzanetant** (NK1/NK3) — acting on the KNDy pathway; CBT formally recommended
Route of oestrogen	Transdermal noted as neutral for VTE	Transdermal now formally **preferred** where VTE risk is raised, explicitly including BMI >30
Regulatory labelling	WHI-driven boxed warning on all systemic HRT	Boxed warning **removed by FDA in 2025** after age- and route-stratified re-analysis
Use after 65	NAMS 2015 — lowest effective dose acceptable; do not stop for age alone	Unchanged and reinforced; documentation of shared decision-making now expected
Chronic disease prevention	Not recommended	Unchanged — Cochrane and USPSTF confirm no preventive role
Monitoring burden	Not applicable	New: **LFT monitoring required with fezolinetant**

Extra Recall Scaffolding — Recurring Topic

Mnemonic for contraindications — "BLOCK-U":
Breast cancer (current, past or high risk) · **L**iver disease (active) · **O**estrogen-dependent tumour · **C**lot — VTE/thromboembolism · **K**nown cardiovascular disease (active) · **U**nexplained postmenopausal bleeding

Numerical anchors to quote: window **50–60 years / within 10 years** of menopause · vasomotor symptoms in **70–80%** for about **7 years** · hot flushes persist in **15% at 66** and **9% at 72** · ultra-low patch **0.014 mg estradiol** · universal advice **800 IU vitamin D + 700–1200 mg calcium** · alcohol **≤2 units/day** · elinzanetant **73.8%** VMS reduction · fezolinetant **~60%**.

Named frameworks: WHI · WHIMS (dementia) · Chlebowski JAMA 2020 (breast-cancer follow-up) · Boardman Cochrane review · NAMS/The Menopause Society position on use after 65 · NICE NG23 · SKYLIGHT and DAYLIGHT (fezolinetant) · OASIS 1–4 (elinzanetant).

Examiner traps to avoid:
• **Saying HRT prevents heart disease** — it does not; started late it *causes* it.
• **Forgetting the progestogen** in a woman with a uterus.
• **Claiming all HRT increases breast cancer** — oestrogen-alone reduced incidence and mortality in WHI.
• **Stopping HRT purely because the woman has turned 65.**
• Failing to distinguish **systemic** HRT from **low-dose vaginal** oestrogen, which is safe long-term.
• Treating all progestogens as equivalent — MPA is not micronised progesterone.

15-second answer skeleton: Define (oestrogen ± progestogen) → two licensed indications → lowest dose, shortest time → **window of opportunity 50–60 / within 10 years** → preparations, progestogen choice, transdermal route → benefits (VMS, GSM, bone) → risks (breast, VTE, stroke, CHD if late, dementia, endometrium) → contraindications (BLOCK-U) → continuing beyond 65 (NAMS — not stopped for age alone) → non-hormonal alternatives → **NK antagonists: fezolinetant and elinzanetant**.

Exam One-Liners

- **Window of opportunity: 50–60 years, within 10 years of menopause.**
- Uterus present → **always add a progestogen**.
- Transdermal oestrogen is VTE-neutral; oral is not.
- Oestrogen alone **lowered** breast cancer in WHI; combined therapy raised it.
- WHIMS: combined HRT in older women **doubles** dementia risk.
- Do not stop HRT for age alone — stop it for **risk**.
- New non-hormonal drugs: **fezolinetant (NK3)** and **elinzanetant (NK1/NK3)** acting on KNDy neurons.

QUESTION 8 — MALE ENDOCRINOLOGY

Gynaecomastia

7 MARKS

High-Yield Points

1. **Definition:** benign proliferation of **glandular** breast tissue in a male, caused by an increased ratio of **oestrogen to androgen action** on the breast. Must be distinguished from **lipomastia (pseudogynaecomastia)**, which is fat alone with no palpable disc.
2. **Clinical picture:** a **concentric, rubbery, mobile subareolar disc**, often tender during the active proliferative phase, usually bilateral but frequently asymmetrical.
3. **Why it is common in old age:** testosterone falls while **SHBG rises** (so free testosterone falls faster), and increasing adiposity raises **peripheral aromatisation of androgens to oestradiol** — shifting the oestrogen:androgen ratio.
4. **Physiological / idiopathic:** senescent gynaecomastia and obesity account for a large share of cases.
5. **Pathological causes — reduced androgen:** primary or secondary hypogonadism (including Klinefelter's presenting late), chronic kidney disease, chronic liver disease, testicular atrophy.
6. **Pathological causes — increased oestrogen:** cirrhosis (reduced oestrogen clearance and raised SHBG), thyrotoxicosis, adrenal tumours, testicular Leydig or Sertoli cell tumours, and **hCG-secreting tumours (bronchogenic carcinoma, germ cell tumours)**. Also refeeding after starvation.
7. **Drug-induced (about a quarter of cases — the dominant cause in geriatric practice): spironolactone**, digoxin, cimetidine/ranitidine, ketoconazole, calcium channel blockers, ACE inhibitors, **antiandrogens and GnRH agonists used for prostate cancer**, antipsychotics, metoclopramide, antiretrovirals, alcohol, cannabis, opioids — and **exogenous testosterone itself**, through aromatisation to estradiol.
8. **Red flags for male breast carcinoma:** a **hard, eccentric (not subareolar), fixed, unilateral** mass, skin dimpling, nipple retraction or bloody discharge, or axillary lymphadenopathy → mammography with core biopsy (triple assessment). Male breast cancer incidence rises with age, so an older man with an eccentric hard lump must never simply be reassured.
9. **Investigations:** a meticulous **drug history** and examination of testes, liver, thyroid and chest; then liver and renal function, TSH, **early-morning total testosterone, LH, estradiol, beta-hCG, prolactin and DHEAS**. Testicular ultrasound if a testicular mass is palpable or hCG is raised.
10. **Management:**
    - **Treat the cause** — stop or substitute the offending drug, treat hypogonadism, thyrotoxicosis, liver or renal disease, and reduce weight. This alone resolves most cases.
    - **Drug therapy works only in the early, painful, proliferative phase (<12 months)**: **tamoxifen 10–20 mg/day** is the best-supported option (off-label); aromatase inhibitors are less effective. After about a year, fibrosis is established and drugs rarely help.
    - **Surgery** — subcutaneous mastectomy with or without liposuction — for persistent, fibrotic or cosmetically distressing disease.
    - **Prophylaxis:** tamoxifen or low-dose breast irradiation before starting antiandrogen therapy for prostate cancer.
    - Testosterone-induced gynaecomastia in the first months of replacement is usually **transient** and resolves as testosterone antagonises estradiol at the breast; occasionally an oestrogen receptor antagonist or aromatase inhibitor is used briefly.

Latest Updates & Current Practice (2023–Present) — 3 Points

- **TRAVERSE** (Lincoff et al., NEJM 2023; 5246 men aged 45–80) recorded gynaecomastia among the adverse events of transdermal testosterone but found **no excess of prostate cancer or prostate-related events**, and non-inferiority for major adverse cardiac events — reassuring for men who develop breast tenderness on testosterone therapy.
- **Iatrogenic gynaecomastia now dominates geriatric practice** — with widespread use of **spironolactone in heart failure** (and now of mineralocorticoid receptor antagonists generally) and of **androgen-deprivation therapy for prostate cancer**, the medication list should be reviewed **before** any endocrine work-up.
- **Prophylaxis before androgen deprivation is now standard** in men expected to receive prolonged antiandrogen monotherapy — a single fraction of breast irradiation or tamoxifen substantially reduces the incidence and pain of gynaecomastia, and is far more effective given before rather than after the breast tissue has become fibrotic.

Exam One-Liners

- Gynaecomastia = **glandular** disc; lipomastia = fat only.
- The mechanism is always a **raised oestrogen:androgen ratio**.
- In an older man, **look at the drug chart first** — spironolactone, digoxin, antiandrogens.
- **Eccentric, hard, fixed lump = carcinoma until proved otherwise.**
- Tamoxifen works only in the **first 12 months**; after that, surgery.

QUESTION 9 — MALE ENDOCRINOLOGY

Andropause (Late-Onset Hypogonadism)

7 MARKS

High-Yield Points

1. **"Andropause" is a misnomer.** Unlike the menopause there is **no abrupt, universal, inevitable loss of gonadal function in men**; testosterone declines gradually and continuously throughout adulthood. The preferred term is **late-onset hypogonadism (LOH)**.
2. **Definition:** hypogonadism is a combination of **subnormal serum testosterone AND one or more attributable symptoms**. It is primary (testicular) or secondary (pituitary/hypothalamic).
3. **Physiology:** free testosterone falls about **1% per year after age 30**. In the European Male Ageing Study, between 40 and 79 years total testosterone fell **0.4% per year** and free testosterone **1.3% per year** — the free fraction falls faster because **SHBG rises with age**. Mechanisms: fewer Leydig cells, reduced Leydig cell response to LH, and reduced episodic and stimulated gonadotropin secretion.
4. **Epidemiology:** at least **30% of older men have subnormal testosterone**, but true symptomatic LOH affects only about **2–3%**. Only 5–35% of hypogonadal men are treated. Comorbidity — obesity, type 2 diabetes, chronic illness, opioids, glucocorticoids — contributes at least as much as age itself.
5. **Symptoms: low libido is the most specific**. Others: erectile dysfunction, loss of morning erections, reduced muscle mass and strength, increased body fat, reduced bone mineral density and osteoporosis, mild anaemia, breast discomfort and gynaecomastia, hot flushes, sleep disturbance, reduced vitality, poor concentration, depression and fatigue. **Most of these overlap with normal ageing and with other diseases — the central diagnostic difficulty.**
6. **Screening tools:** the **ADAM questionnaire** and the **Aging Male Symptoms (AMS) scale** are sensitive (97% and 83%) but poorly specific (30% and 39%), especially in the borderline range — useful to prompt testing, useless to confirm.
7. **Diagnosis:** two separate **fasting early-morning (before 11 am) total testosterone** measurements. If borderline, obese, or SHBG is abnormal, measure **free testosterone** by equilibrium dialysis or calculation. Then measure **LH and FSH**: raised = primary; low or inappropriately normal = secondary, which requires prolactin, iron studies, other pituitary hormones, and **pituitary MRI** if testosterone is very low or there are visual/headache symptoms.
8. **Exclude reversible causes before treating:** acute or chronic illness (non-gonadal illness suppresses testosterone), obesity, obstructive sleep apnoea, opioids, glucocorticoids, alcohol, uncontrolled diabetes. Weight loss and treating the illness may normalise testosterone without any hormone.
9. **Treatment principle:** testosterone is offered **only to older men with a genuinely low testosterone AND clinically significant symptoms — never for an asymptomatic age-related decline**. Aim for a **lower target than in young men: about 300–400 ng/dL rather than 500–600 ng/dL**, to minimise risk. Preparations: transdermal gel, intramuscular undecanoate or enanthate, buccal or nasal formulations; **oral testosterone (other than undecanoate) is avoided because of hepatotoxicity, peliosis hepatis and hepatic tumours**. **Contraindications:** prostate carcinoma (the major one), breast cancer, baseline haematocrit >48–50%, untreated severe sleep apnoea, decompensated heart failure, and any desire to father a child (testosterone suppresses spermatogenesis).
10. **Monitoring and expectations:** check testosterone, **haematocrit (keep below 55%)**, PSA and digital rectal examination at baseline, at 3–6 and 12 months, then annually. Benefits are **modest** — improved libido, sexual activity, mood, bone mineral density, anaemia and body composition — but the T-Trials showed **no benefit in vitality or walking distance**. If symptoms do not improve, **stop the testosterone and look for another cause**.

Latest Updates & Current Practice (2023–Present) — 3 Points

- **TRAVERSE** (Lincoff et al., NEJM 2023) — 5246 men aged 45–80 with symptomatic hypogonadism (two fasting testosterone levels <300 ng/dL) and pre-existing or high cardiovascular risk, randomised to 1.62% transdermal testosterone gel or placebo for a mean 22–27 months. Testosterone was **non-inferior to placebo for major adverse cardiac events (7.0% vs 7.3%) and did not increase prostate cancer or prostate-related events** — resolving the question the FDA had mandated in 2015.
- **But the substudies matter as much:** the fracture substudy (Snyder et al., NEJM 2024) found **MORE clinical fractures on testosterone (3.50% vs 2.46%)** — the opposite of expectation. There were also small excesses of atrial fibrillation, pulmonary embolism and acute kidney injury, alongside genuine benefits: a **22.5% reduction in progression from prediabetes to diabetes**, and improvements in sexual desire and activity, depressive symptoms and anaemia.
- **Practice implication: testosterone must NOT be used to treat osteoporosis in older men** — use a

bisphosphonate or denosumab instead. Counsel on falls prevention early in therapy, particularly in the frail, since increased vitality and activity may precede any skeletal benefit. EAU 2024 and the 2025 European Expert Panel (PaTeR) position statement have incorporated TRAVERSE, endorsing treatment for symptomatic hypogonadism provided these precautions and monitoring are followed.

Exam One-Liners

- **Andropause does not exist** — say “late-onset hypogonadism”.
- Free testosterone falls **1% per year** after 30 because **SHBG rises**.
- 30% have low levels; only **2–3%** have true LOH.
- Diagnose on **two fasting morning** samples plus symptoms; **low libido is the most specific** symptom.
- Aim for **300–400 ng/dL**, not the young-adult range.
- **TRAVERSE** = cardiovascular and prostate safe, but **more fractures**.
- Absolute contraindication: **prostate carcinoma**. Keep haematocrit **<55%**.

QUESTION 10 — SEXUAL HEALTH

Causes of Erectile Dysfunction and Treatment Options

15 MARKS

High-Yield Points (Textbook Core)

A. Definition and epidemiology

1. **Erectile dysfunction (ED)** is the persistent inability to attain and maintain an erection satisfactory for sexual intercourse, present for at least 3 months.
2. Prevalence rises steeply with age: about **50% of non-diabetic men over 60**, and **55–95% of diabetic men over 60**.
3. Continuation of an active sex life is **associated with longevity in both men and women**; ED must never be dismissed as normal ageing. In women the parallel problems are vaginal atrophy, low desire, depression and SSRI effects.
4. **Physiology:** erection is **parasympathetic (S2–S4)** and nitric-oxide mediated → cavernosal smooth-muscle relaxation via cGMP; **PDE5 degrades cGMP**. Detumescence is sympathetic. This is the rationale for PDE5 inhibition.

B. Causes — organic (about 80% in the elderly)

5. **Vasculogenic (commonest):** atherosclerosis, hypertension, dyslipidaemia, smoking, diabetes, **endothelial dysfunction**, venous leak, pelvic irradiation, Leriche syndrome.
6. **Neurogenic:** diabetic **autonomic neuropathy**, stroke, Parkinson's disease, multiple sclerosis, spinal cord disease, cauda equina, and pelvic or prostate surgery (radical prostatectomy, cystectomy).
7. **Endocrine / hormonal dysregulation:** hypogonadism, hyperprolactinaemia, thyroid disease, poorly controlled diabetes.
8. **Local and anatomical:** Peyronie's disease, post-priapism fibrosis, phimosis, BPH-related lower urinary tract symptoms.
9. **Drug-related — a particular problem in older patients: thiazide diuretics, spironolactone, beta blockers, cimetidine** (all four specifically named in Pathy's), plus SSRIs, antipsychotics, antiandrogens and GnRH agonists, 5-alpha-reductase inhibitors, opioids, and older centrally acting antihypertensives. **Always take an alcohol and tobacco history.**

C. Causes — psychogenic

10. Performance anxiety, depression, bereavement and loss of a partner, relationship difficulty, and lack of privacy in institutional or multigenerational settings. **Sudden onset, situational failure and preserved nocturnal or early-morning erections suggest a psychogenic component.** In practice most older men have a mixed organic and psychogenic picture.

D. Evaluation

11. **Interview the patient — and the partner where appropriate.** Take a full sexual, psychosocial and drug history, and clarify whether the problem is desire, erection, ejaculation or orgasm.
12. **Examination:** secondary sexual characteristics, penis (plaques, phimosis), testicular volume and consistency, gynaecomastia, peripheral pulses, blood pressure, digital rectal examination, and a focused neurological examination including perineal sensation and bulbocavernosus reflex.
13. **Investigations:** fasting glucose/HbA1c, lipid profile, **early-morning total testosterone**, thyroid function; add prolactin and gonadotrophins if testosterone is low. Validated assessment: the **International Index of Erectile Function (IIEF / IIEF-5)**.
14. **Specialised tests** — nocturnal penile tumescence, penile duplex Doppler, cavernosometry — are available through specialist ED clinics but **are usually avoided in older patients**, in whom extensive testing rarely changes management.

E. Treatment — a stepwise approach

15. **Step 1 — modify reversible factors:** optimise glycaemic, blood pressure and lipid control; stop smoking; reduce alcohol; weight loss and regular exercise; and **substitute the offending drug** (for example replace a thiazide or non-selective beta blocker — nebivolol and ARBs are more erection-friendly; use bupropion or mirtazapine rather than an SSRI for depression).
16. **Step 2 — psychosexual counselling** with the partner involved; general education about age-related sexual change; treat depression and address bereavement.
17. **Step 3 — oral PDE5 inhibitors (first-line pharmacotherapy):**

- **Sildenafil** 25–100 mg on demand, about 1 hour before intercourse; a fatty meal delays absorption.
- **Tadalafil** 10–20 mg on demand or **5 mg once daily**; duration up to **36 hours**, and it also improves BPH-related LUTS — often the best choice in the older man.
- **Vardenafil** and **avanafil** are alternatives.
- PDE5 inhibitors are effective in carefully selected older people with diabetes. **Start at the lower dose** in the elderly, in renal or hepatic impairment, and in men on alpha blockers.

18. **PDE5 inhibitor contraindications and cautions: absolute — any nitrate or nicorandil** (risk of profound hypotension), and riociguat. Caution with alpha blockers (separate dosing), recent myocardial infarction or stroke, unstable angina, severe aortic stenosis, hypotension, and retinitis pigmentosa. Side effects: headache, flushing, dyspepsia, nasal congestion, blue-tinged vision (sildenafil), back and muscle ache (tadalafil), and rarely **non-arteritic anterior ischaemic optic neuropathy** and sudden sensorineural hearing loss.
19. **Step 4 — testosterone replacement** where the man is genuinely hypogonadal: it improves libido and may **restore responsiveness to a previously failed PDE5 inhibitor**. It is not a treatment for ED with normal testosterone.
20. **Step 5 — second-line therapies: intracavernosal alprostadil** injection (most effective non-surgical option; warn about priapism), **intraurethral alprostadil (MUSE)**, and the **vacuum erection device** — particularly valuable in older men on nitrates or anticoagulants and after radical prostatectomy.
21. **Step 6 — penile prosthesis** (inflatable or malleable) for refractory ED, with high satisfaction rates. Penile revascularisation is reserved for younger men with isolated post-traumatic arterial insufficiency. **Low-intensity shockwave therapy, platelet-rich plasma and stem-cell "restorative" therapies remain investigational** and should not be offered as established treatment.

Latest Updates (2023–Present)

- **Princeton IV Consensus** (Kloner et al., *J Sex Med* 2024; Mayo Clin Proc 2024) — ED is formally recognised as an **ASCVD risk-enhancing factor**. Men aged 40–79 with ED and no overt cardiac disease should undergo formal 10-year ASCVD risk estimation, with **coronary artery calcium scoring as a risk modifier** in those calculated to be low risk. Sexual intercourse represents only **2–3 METs**, comparable to climbing two flights of stairs or walking 1.6 km in 20 minutes, and is safe in stable cardiovascular disease.
- **PDE5 inhibitors appear cardioprotective.** A 2023 meta-analysis of 16 cohort studies (1,257,759 subjects, median follow-up 4.3 years) found PDE5 inhibitor use associated with **reduced MACE (RR 0.78) and reduced all-cause mortality (RR 0.70)**. This reframes them as more than a symptomatic drug — though randomised confirmation is still awaited, and signals are neutral or unfavourable in HFpEF and after stroke.
- **EAU 2025 Sexual and Reproductive Health guideline** — many apparent "non-responders" are simply **inadequately treated**: verify dosing, timing and at least 4–6 adequate attempts, and try more than one molecule or schedule before declaring failure. Efficacy is lower where diabetes, obesity and cardiovascular disease are poorly controlled, so **comorbidities must be optimised concurrently**. Testosterone therapy may be used as an **initial** treatment where ED coexists with biochemical hypogonadism.
- **ICSM 2024** (Fifth International Consultation on Sexual Medicine) — choice among PDE5 inhibitors should be driven by patient preference, anticipated frequency of activity, desired duration of action, response to a therapeutic trial, partner factors and cost; **testosterone monotherapy has only a limited role in isolated ED**. A dedicated 2025/2026 ICSM statement addresses sexuality and sexual health specifically in older adults.
- Combination or dual PDE5 inhibitor regimens (daily low-dose tadalafil plus on-demand sildenafil) are being studied for on-demand non-responders, particularly in diabetes — including Indian cohort data (Lucknow, 2025).

Current Practice Update

Issue	Textbook position	Current practice (2023–present)
Meaning of ED	A complex multifactorial sexual problem	A **sentinel marker of endothelial dysfunction and an ASCVD risk-enhancing factor** — triggers formal cardiovascular risk assessment
PDE5 inhibitors	Effective symptomatic therapy in selected older diabetic men	Same, plus observational evidence of reduced MACE and all-cause mortality
Defining failure	Move to second-line if oral therapy fails	Must first confirm correct dose, timing and ≥4–6 attempts, and trial a second molecule
Testosterone	Adjunct in the hypogonadal man	May be an **initial** treatment when ED coexists with proven hypogonadism; not for isolated ED

Restorative therapies	Not discussed	Shockwave, PRP and stem-cell therapy formally reviewed and classed as **investigational**

Exam One-Liners

- Erection = **parasympathetic, nitric oxide, cGMP**; PDE5 breaks cGMP down.
- Commonest organic cause in the elderly is **vasculogenic**; commonest reversible cause is **drugs**.
- Preserved **morning erections** point to a psychogenic component.
- **Nitrates + PDE5 inhibitor = absolutely contraindicated.**
- **Tadalafil 5 mg daily** treats ED and LUTS together — ideal in the older man with BPH.
- Vacuum device is the safe option in men on nitrates or anticoagulants.
- **ED precedes coronary events by about 3–5 years** — assess cardiovascular risk in every man who presents with it.

QUESTION 11 — FEMALE ENDOCRINOLOGY / DERMATOLOGY

Hirsutism and Baldness in Elderly Women

7 MARKS

High-Yield Points

1. **Definitions: hirsutism** is excessive growth of **terminal** (coarse, pigmented) hair in a male pattern in a woman — upper lip, chin, chest, upper abdomen, back and thighs. It must be distinguished from **hypertrichosis**, which is generalised excess of vellus or terminal hair, is **not androgen-dependent**, and is usually drug-induced or familial.
2. **Why both problems appear together after the menopause:** ovarian follicles are depleted so **oestrogen production falls sharply, while ovarian stromal and thecal androgen production and adrenal androgens are relatively preserved**. The resulting rise in the **androgen:oestrogen ratio** produces facial hirsutism *and* scalp hair loss in the same woman — a classic examination point.
3. **Common causes:** idiopathic hirsutism; long-standing polycystic ovary syndrome; obesity and insulin resistance (adipose tissue is a major site of aromatisation and of altered SHBG); and drugs — glucocorticoids, danazol, testosterone and anabolic steroids cause true hirsutism, while **minoxidil, ciclosporin, phenytoin and diazoxide cause hypertrichosis**.
4. **Sinister causes that must be excluded:** androgen-secreting **ovarian or adrenal tumour**, ovarian hyperthecosis, Cushing's syndrome, non-classical congenital adrenal hyperplasia, acromegaly, hyperprolactinaemia and thyroid disease. **New or rapidly progressive hirsutism with virilisation in a postmenopausal woman is a tumour until proved otherwise.**
5. **Virilisation red flags:** onset over weeks to months, **clitoromegaly**, deepening of the voice, temporal balding, breast atrophy, increased muscle bulk, and biochemically a **total testosterone above 150–200 ng/dL** or **DHEAS above 700 µg/dL**.
6. **Assessment of hirsutism:** the **modified Ferriman–Gallwey score** — nine androgen-sensitive areas each scored 0 (no hair) to 4 (frankly virile). An abnormal score (conventionally ≥8, but **ethnicity-adjusted — lower thresholds for East Asian and higher for Mediterranean and South Asian women**) warrants androgen testing. **Investigations:** early-morning total and free testosterone, DHEAS, 17-hydroxyprogesterone, prolactin, TSH, and overnight dexamethasone suppression if Cushing's is suspected; then transvaginal ultrasound and adrenal CT if androgens are markedly raised.
7. **Female pattern hair loss (FPHL):** diffuse thinning over the crown with **widening of the central parting and preservation of the frontal hairline** (Ludwig pattern) — in contrast to the bitemporal recession of male-pattern loss. It affects **up to two-thirds of postmenopausal women**. Exclude telogen effluvium, iron deficiency, thyroid disease, and the **scarring alopecias — particularly frontal fibrosing alopecia, characteristically a postmenopausal condition with eyebrow loss and a receded, pale hairline**.
8. **Investigations for hair loss: ferritin** (aim above 40–60 ng/mL, not merely above the laboratory lower limit), TSH, vitamin D, complete blood count, and a careful drug review; clinically, the hair pull test and dermoscopy (trichoscopy) showing **hair-shaft diameter diversity**.
9. **Treatment of hirsutism:**
   - **Direct hair removal** — shaving, plucking, threading, waxing and depilatories; definitive removal by **electrolysis** or **laser/photoepilation** (most effective for dark hair on light skin; multiple sessions needed).
   - **Topical eflornithine 13.9% cream** slows facial hair growth (it inhibits ornithine decarboxylase) and is useful as an adjunct.
   - **Systemic antiandrogens** for moderate to severe disease: **spironolactone 50–100 mg/day** — **monitor potassium and renal function carefully in the elderly and avoid combining with ACE inhibitors or ARBs**; alternatives are finasteride and cyproterone acetate. **Flutamide should be avoided** because of hepatotoxicity.
   - **Allow at least 6 months before judging response**, because the hair growth cycle is slow. Contraception is not a concern in this age group, unlike in premenopausal women.
10. **Treatment of female pattern hair loss: topical minoxidil 5%** is the only FDA-approved option — expect results at 4–6 months, peak benefit at 6–12 months, and **loss of benefit if stopped**. Correct iron and thyroid deficiency first, since no topical agent compensates for an untreated nutritional or endocrine cause. Add an antiandrogen or 5-alpha reductase inhibitor for greater effect; procedural adjuncts include microneedling, platelet-rich plasma and low-level laser therapy. **Both conditions cause substantial psychological distress and a perceived loss of femininity — address body image, mood and social withdrawal explicitly, as part of the geriatric assessment.**

Latest Updates & Current Practice (2023–Present) — 3 Points

- **Low-dose oral minoxidil (0.625–2.5 mg/day)** has become a mainstream option for female pattern hair loss. A

2025 *JAAD* meta-analysis and ambulatory blood-pressure monitoring studies found **no clinically significant fall in blood pressure at these doses**, with hypertrichosis the commonest side effect. This is directly relevant to older women, who often cannot tolerate or physically apply the topical form — roughly 40% of women in one Mayo Clinic series had abandoned topical minoxidil for local side effects or adherence difficulty.

- **Antiandrogen therapy in FPHL** (*JAAD* 2025 review): **combination therapy consistently beats monotherapy**. Oral bicalutamide 25 mg/day is emerging, although a randomised trial found no clear added benefit over oral minoxidil alone within 6 months. Importantly, the androgenic contribution to FPHL is now considered **less certain than in men** — most affected women have normal androgen levels, and androgen excess in women manifests as hirsutism rather than alopecia.
- **Guidance on investigation:** the Endocrine Society hirsutism guideline (2018, still current) and SOGC Guideline No. 444 (2023) advise **against** biochemical androgen testing in women with only mild localised unwanted hair and a normal hirsutism score, recommend **pharmacological therapy plus hair removal** for patient-important hirsutism, and advise **strongly against flutamide**. A 2024 *JAMA Dermatology* systematic review confirms the efficacy of laser and light-based therapies.

Exam One-Liners

- Hirsutism = **terminal, androgen-dependent, male pattern**; hypertrichosis = generalised and androgen-independent.
- After the menopause, **oestrogen falls but androgen persists** — hence chin hair and crown thinning together.
- **Rapid hirsutism + virilisation in an older woman = androgen-secreting tumour.**
- Testosterone **>150–200 ng/dL** or DHEAS **>700 µg/dL** → image the ovaries and adrenals.
- Score it with the **modified Ferriman–Gallwey** scale (9 areas, 0–4 each).
- FPHL = **Ludwig pattern**, frontal hairline preserved; check **ferritin and TSH**.
- Spironolactone in the elderly → **watch potassium and creatinine**.

GASTROENTEROLOGY & NUTRITION

MD Geriatric Medicine — Examination Answers

Ten high-yield structured answers with current evidence to 2026

Primary textbook sources: Pathy's Principles and Practice of Geriatric Medicine, 6th edition (2022); Essentials of Clinical Geriatrics, 7th edition.

Format: 15–20 numbered high-yield points for 15-mark questions; 8–10 points for 7-mark questions.

Each answer contains: textbook-based points → Latest Updates (2023–present, inline citations) → Current Practice Update comparison table → Examiner's Tip panel.

Green "Extra Points to Remember" panels mark recurring / repeatedly asked topics.

Units: Indian conventional units throughout (Hb g/dL, calcium mg/dL, creatinine mg/dL).

Prepared for Sudarsan | Revision set — Gastroenterology & Nutrition | August 2026

Contents

1. Malabsorption — etiopathogenesis, clinical features, diagnostic algorithm, management RECURS
2. Constipation in the elderly — pharmacological management and complications RECURS
3. Gastro-oesophageal reflux disease (asked twice) RECURS
4. Gastrointestinal motility disorders in the elderly
5. Approach to upper gastrointestinal bleeding
6. Haematemesis (7 marks)
7. Assessment of nutrition — methods, advantages and disadvantages
8. Mini Nutritional Assessment — components, scoring, interpretation (7 marks)
9. Total parenteral nutrition
10. Vitamin deficiencies in the elderly

A. Rapid Revision Grid — numbers, trials, formulae and named evidence

1. Etiopathogenesis, clinical manifestations, diagnostic algorithm and management of malabsorption

15 MARKS RECURRING TOPIC

Definition and Ageing Physiology

1. **Malabsorption** means defective passage of digested nutrients into the blood and lymph. It may be **global** (all nutrients) or **selective** (one nutrient, e.g. B12).
2. Distinguish **maldigestion** (failure of luminal breakdown — pancreatic or bile salt problem) from **malabsorption** (failure of mucosal uptake or lymphatic transport).
3. Healthy ageing alone does **not** cause malabsorption. Pathy's states that although brush-border enzymes fall, digestion and absorption of **carbohydrate, fat and protein remain normal** in healthy old age.
4. Age-related changes are selective: **vitamin A absorption increases**, **vitamin D absorption decreases**, **folate absorption falls slightly**, and **B12 absorption falls when hypochlorhydria is present**.
5. Small-intestinal **motility and transit are essentially preserved** with healthy ageing; therefore **bacterial overgrowth is rare in the healthy elderly**. When it occurs, look for disease, not for age.

Etiopathogenesis — Four Mechanisms

6. **Luminal (maldigestion):** pancreatic exocrine insufficiency (chronic pancreatitis, pancreatic cancer), bile salt deficiency (cholestasis, ileal resection), and **hypochlorhydria from atrophic gastritis or long-term PPI use**.
7. **Mucosal:** coeliac disease, tropical sprue, Crohn's disease, giardiasis, drug-induced enteropathy (olmesartan, NSAIDs, colchicine), amyloidosis, radiation enteritis.
8. **Post-mucosal (lymphatic/vascular):** intestinal lymphangiectasia, lymphoma, chronic mesenteric ischaemia, constrictive pericarditis, right heart failure.
9. **Stasis with bacterial overgrowth (SIBO):** Pathy's identifies the classic geriatric mechanism — **diabetes with autonomic neuropathy**, **small-intestinal diverticula**, and **reduced gastric acid** cause stasis and predispose to overgrowth, causing malnutrition and diarrhoea.
10. Post-surgical causes matter in the elderly: previous gastrectomy, blind loop, ileal resection, and short bowel after mesenteric ischaemia.
11. In **India**, always add **tropical sprue**, **intestinal tuberculosis**, **giardiasis**, **strongyloidiasis**, and **HIV enteropathy** to the differential — these are far commoner than in Western series.

Clinical Manifestations

12. **Classical picture** — chronic diarrhoea, **steatorrhoea** (bulky, pale, offensive, floating stool), weight loss, abdominal bloating and flatulence.
13. **Geriatric picture is atypical and oligosymptomatic:** presentation is often only **unexplained weight loss, anaemia, fatigue or a metabolic bone disease**, with little or no diarrhoea. Diarrhoea may even be absent.
14. **Deficiency-based clues:** iron/folate/B12 deficiency anaemia; osteomalacia and proximal myopathy (vitamin D, calcium); bruising (vitamin K); glossitis and cheilitis (B-complex); night blindness (vitamin A); oedema (hypoalbuminaemia); peripheral neuropathy (B12).
15. Fat-soluble vitamin loss (**A, D, E, K**) occurs in maldigestion and steatorrhoea, whereas isolated B12 loss suggests ileal or gastric pathology.

Diagnostic Algorithm

16. **Step 1 — Confirm malabsorption and screen:** CBC with MCV, iron studies, B12, folate, calcium, phosphate, albumin, prothrombin time, 25(OH)D, TSH. A raised INR that corrects with vitamin K is a useful bedside pointer.
17. **Step 2 — Confirm fat malabsorption:** qualitative **Sudan III stain** of stool (simple, available in India) or 72-hour faecal fat (>7 g/day). Faecal calprotectin distinguishes inflammatory from non-inflammatory causes.
18. **Step 3 — Localise the defect:**
    - **Mucosal → IgA anti-tTG plus total IgA**, then **upper GI endoscopy with 4–6 duodenal biopsies** (including bulb).
    - **Pancreatic → faecal elastase-1**; <200 µg/g suggests, <100 µg/g indicates severe exocrine insufficiency; CT/MRCP for structural disease.
    - **SIBO** → Pathy's: culture of **duodenal aspirate** or **glucose/xylose hydrogen breath test**; sensitivity and specificity vary widely, so a **negative test after antibiotics increases diagnostic certainty**.
19. **Step 4 — Imaging and exclusion of malignancy:** CT enterography, capsule endoscopy or enteroscopy for obscure cases; CT angiography if chronic mesenteric ischaemia is suspected (post-prandial pain, food fear, weight loss).

20. **Always exclude malignancy first in the elderly.** Weight loss plus altered bowel habit in an older patient requires a low threshold for colonoscopy and cross-sectional imaging before labelling anything "functional".

Management

21. **Treat the cause:** lifelong gluten-free diet for coeliac disease; pancreatic enzyme replacement for exocrine insufficiency; tetracycline plus folic acid for tropical sprue; ATT for intestinal tuberculosis.
22. **SIBO (Pathy's): metronidazole, tetracycline or quinolones, or non-absorbed rifaximin**, given for **one to four weeks**, repeated **cyclically** if recurrent — noting that Pathy's stresses there is a paucity of controlled data to guide practice.
23. **Correct deficiencies aggressively:** parenteral iron if oral is not absorbed, B12 (oral high-dose or IM), folate, calcium with vitamin D, and vitamin K where INR is prolonged.
24. **Nutritional rehabilitation:** high-energy, high-protein diet; oral nutritional supplements; MCT oil for severe steatorrhoea; lactose restriction if secondary lactase deficiency; involve a dietitian, and monitor for **refeeding syndrome** in the very depleted.
25. **Symptomatic and stewardship measures:** Pathy's notes **loperamide and clonidine** may help when no cause is found, but warns older patients are especially prone to adverse effects — constipation, urinary retention and glaucoma with loperamide; hypotension, bradycardia, sedation and dry mouth with clonidine. **Deprescribe the PPI** if the indication has lapsed, since acid suppression drives both B12 deficiency and overgrowth.

Latest Updates (2023–Present)

- **UEG 2025 guideline on malabsorption in adults** gives the first unified framework — history and drug review first, then targeted functional and endoscopic testing rather than a scattergun panel. It highlights that in autoimmune atrophic gastritis, parietal cell loss, **SIBO and iatrogenic hypochlorhydria from chronic PPI use** all aggravate B12 deficit, and that **B12 screening in the elderly matters because some neurological abnormalities reverse with supplementation** *(United European Gastroenterol J, 2025)*.
- **ACG guideline update on coeliac disease (2023)** retains **EGD with multiple duodenal biopsies** as the confirmatory standard in adults, recommends **case-finding rather than mass screening**, and requires serology and biopsy to be done **on a gluten-containing diet** *(Am J Gastroenterol, 2023)*.
- **ESsCD 2025 guidelines** conditionally permit a **no-biopsy diagnostic pathway only in adults under 45 years** with markedly raised IgA anti-tTG — meaning **older adults still require duodenal biopsy**, an important exam-relevant caveat *(United European Gastroenterol J, 2025)*.
- Coeliac disease in the elderly is increasingly recognised: a UK cohort found the proportion of coeliac diagnoses made over age 65 rose from 0% in 1990–91 to **18.7% by 2016–17**, with older patients presenting **less often with diarrhoea and more often with B12 deficiency** *(UK coeliac cohort study, 2020)*.
- Population data support routinely considering **exocrine pancreatic insufficiency** as a cause of unexplained diarrhoea and bloating in older adults, with reported prevalence around **10% by faecal elastase-1 in the elderly** *(Norwegian population data cited in current EPI literature)*.

Current Practice Update

Textbook position (Pathy's / Essentials)	Current evidence (2023–2026)
SIBO diagnosed by duodenal aspirate culture or hydrogen breath test; both with variable accuracy.	Same tests remain, but UEG 2025 emphasises a **structured stepwise work-up with drug history first** and warns against over-testing; PPI use is now formally listed as an aggravating cause.
Coeliac disease considered largely a younger-adult disease.	Now recognised as a **growing elderly diagnosis**, presenting with anaemia and B12 deficiency rather than diarrhoea; the **no-biopsy pathway is restricted to under-45s**, so biopsy remains mandatory in geriatric practice.
Little dedicated emphasis on pancreatic exocrine insufficiency as a geriatric cause.	**Faecal elastase-1** is now the routine first-line non-invasive test; <200 µg/g suggests and <100 µg/g indicates severe insufficiency.
Antibiotic courses for SIBO based on limited controlled data.	Position unchanged — **rifaximin and cyclical courses remain empirical**; high-quality RCT evidence in older adults is still awaited.

Examiner's Tip — mark-earning lines

- "Ageing itself does not cause malabsorption — Pathy's states digestion and absorption of fat, carbohydrate and protein remain normal in healthy old age; malabsorption in an older patient always signals underlying pathology."
- "In the elderly the presentation is oligosymptomatic — unexplained weight loss, anaemia or osteomalacia without diarrhoea."
- "Bacterial overgrowth is rare in the healthy elderly but common where there is stasis — diabetes, small-bowel diverticula and hypochlorhydria."
- "Exclude malignancy before diagnosing benign malabsorption in any patient over 65."

Extra Points to Remember (recurring topic)

- **D-xylose test:** abnormal in mucosal disease, normal in pure pancreatic disease — a classic viva discriminator.
- **MMA and homocysteine:** both raised in B12 deficiency; only homocysteine raised in folate deficiency.
- **Drug-induced enteropathy** to name: olmesartan (sprue-like), NSAIDs, colchicine, metformin (B12), methotrexate (folate).
- **Whipple's disease** — rare but examinable: PAS-positive macrophages, treated with ceftriaxone then long-course co-trimoxazole.
- Steatorrhoea threshold to quote: **faecal fat >7 g/day on a 100 g fat diet**.

2. Management of constipation in the elderly (including pharmacological management and complications)

15 MARKS RECURRING TOPIC

Definition, Epidemiology and Ageing

1. Constipation is defined by **Rome IV** criteria — straining, hard or lumpy stools, sensation of incomplete evacuation, anorectal blockage, need for manual manoeuvres, or fewer than three spontaneous bowel movements weekly, for at least 3 of the last 6 months.
2. Pathy's makes a key teaching point: **when comorbidities are controlled for, constipation is no commoner in the elderly than in the young, and stool frequency does not change with age**. What older people complain of is **straining and hard stools**.
3. **Laxative use, not constipation itself, rises steeply with age.** Complaints and laxative consumption are both very common in older people.
4. Age-related changes that do matter: **slower colonic peristalsis**, **reduced anal sphincter tone**, and in older women, impaired rectoanal angle and perineal descent after obstetric trauma — so **elderly women have more rectal evacuation problems than elderly men**.
5. Constipation reduces quality of life, lowers self-perceived health, worsens urinary frequency, urgency and dysuria, and its treatment improves mood, sexual function and continence.

Aetiology and Classification

6. Pathy's is explicit that **most causes relate to medication use or coexisting medical illness**, not to ageing.
7. **Commonest drugs: opiates, calcium channel blockers (verapamil worst), NSAIDs and anticholinergics**. Also iron, antipsychotics, tricyclics, antiparkinsonian drugs, diuretics.
8. **Secondary causes:** hypothyroidism, hypercalcaemia, hypokalaemia, diabetes, Parkinson's disease, stroke, dementia, depression, spinal cord lesions, colorectal cancer, strictures.
9. **Pathophysiological types:** normal-transit (commonest), **slow-transit**, and **dyssynergic defecation** (pelvic floor dyssynergia) — the last is important because it responds to biofeedback and not to laxatives.
10. Pathy's notes that **immobility and low fluid/fibre intake are often blamed but poorly supported by evidence** — increased physical activity does not reliably improve constipation.

Assessment

11. History with the **Bristol Stool Form Scale**, full drug review, and a **mandatory digital rectal examination** to detect impaction, reduced tone, and dyssynergia.
12. **Red flags demanding colonoscopy:** rectal bleeding, anaemia, weight loss, new change in bowel habit, family history of colorectal cancer, age with overdue screening.
13. Pathy's principle: **in patients with up-to-date colorectal cancer screening and no worrying symptoms, empirical treatment is appropriate**; testing is reserved for those who fail to respond.
14. Second-line testing when refractory: **colonic transit study**, **anorectal manometry with balloon expulsion**, and **defecography**. A plain abdominal radiograph helps assess stool load or confirm high impaction.

Pharmacological Management — Stepwise

15. **Step 1 — Review and remove:** stop or substitute constipating drugs; switch verapamil to another calcium channel blocker; increase dietary fibre and fluids as a universally accepted first step (though of limited evidence).
16. **Step 2 — Bulking agents: psyllium 3–6 g**, bran 4–10 g, methylcellulose 2–4 g. Pathy's warns they must be **avoided in faecal impaction, bed-bound patients and those with poor fluid intake**; side effects are bloating and flatulence.
17. **Step 3 — Osmotic laxatives: PEG (macrogol) 17–34 g** is the best tolerated overall in elders and improves both stool consistency and frequency; lactulose 10–40 g and sorbitol 70% 15–60 mL are alternatives but cause bloating and cramps through bacterial fermentation.
18. **Magnesium and saline salts should generally be avoided in the elderly** — risk of hypermagnesaemia even with normal renal function; contraindicated in renal disease, end-stage liver disease and heart failure.
19. **Step 4 — Stimulant laxatives: senna 15 mg**, **bisacodyl 5–10 mg**, **sodium picosulfate 5–10 mg**. Most commonly combined with an osmotic agent when a single drug fails.
20. **Step 5 — Second-line prescription agents: lubiprostone 8–24 µg twice daily** (chloride channel activator) is specifically endorsed by Pathy's for elders; **prucalopride**, a 5-HT4 agonist, is useful in refractory cases.
21. **Stool softeners (docusate) have no evidence base** — Pathy's records no RCT showing efficacy, one trial showing

no benefit over placebo, and psyllium proving superior. The faecal incontinence chapter states bluntly that **docusate is ineffective and should not be prescribed**.

22. **Opioid-induced constipation:** use **PAMORAs** — **naloxegol, naldemedine, methylnaltrexone** — which reverse constipation without reversing analgesia or precipitating withdrawal. Pathy's calls methylnaltrexone "an important advance", reserved for refractory cases as it is injected on alternate days.
23. **Enemas and suppositories:** small-volume tap water enemas and arachis oil retention enemas (check nut allergy) are useful; **phosphate enemas cause serious hyperphosphataemia** in renal impairment; large tap-water enemas can cause hyponatraemia; **soap-suds enemas should never be used**.
24. **Non-pharmacological and special situations: biofeedback is the mainstay for dyssynergic defecation**, effective in around **70%** over 4–6 sessions and superior to diet, exercise, laxatives or diazepam. In dementia, prevention with a regular osmotic laxative is preferred; non-verbal cues of a need to defecate include fidgeting, pacing and pulling at clothes.
25. **Refractory disease:** nearly **90% of slow-transit constipation responds to laxatives**; subtotal colectomy with ileorectal anastomosis is rarely needed and only after dyssynergia has been treated. Antegrade enemas via caecostomy are a last resort with limited evidence.

Complications

26. **Faecal impaction** — present in about **7% of institutionalised elderly** on rectal examination. **The first sign is often overflow diarrhoea and rectal incontinence**; without a rectal examination it is misdiagnosed and wrongly treated with antidiarrhoeals.
27. Further complications: **stercoral ulceration and colonic perforation** (immobile or cognitively impaired patients), faecal incontinence, urinary retention and urinary symptoms, haemorrhoids and anal fissure, rectal prolapse, sigmoid volvulus, delirium and agitation in dementia, and laxative-related electrolyte disturbance.
28. **Management of impaction:** Pathy's states the optimal treatment is not clear — oral **PEG or other osmotic laxatives with or without enemas** if tolerated; **manual disimpaction** for a hard or large rectal bolus; hyperosmotic water-soluble contrast enemas have also been used successfully. **Bulking agents must be avoided** in this group.

Latest Updates (2023–Present)

- The **first-ever joint AGA–ACG clinical practice guideline on chronic idiopathic constipation (2023)** made 10 GRADE recommendations. **Strong recommendations** were given for **polyethylene glycol, sodium picosulfate/bisacodyl, linaclotide, plecanatide and prucalopride**; conditional suggestions for fiber, magnesium oxide, lactulose, senna and lubiprostone *(Am J Gastroenterol / Gastroenterology, 2023)*.
- This guideline was the **first to endorse magnesium oxide and senna as evidence-based treatments**, and the first to include randomised data for magnesium oxide and prucalopride *(AGA/ACG joint guideline, 2023)*.
- It reaffirms that **prescription secretagogues and prokinetics should follow, not precede, over-the-counter agents**, and that fibre is first-line particularly in those with low dietary fibre intake *(AGA/ACG, 2023)*.
- **Docusate remains without support** — it was reviewed and not recommended, confirming the textbook's scepticism *(AGA/ACG, 2023)*.
- The **ASCRS 2024 guideline** on chronic constipation adds surgical-perspective guidance for refractory slow-transit disease and pelvic floor dysfunction *(Dis Colon Rectum, 2024)*.
- Non-pharmacological adjuncts continue to accumulate low-grade support in stroke survivors — **abdominal massage** and **visceral mobilisation** reduce constipation severity, though further high-quality studies are needed *(systematic review and RCT evidence, 2020–2024)*.

Current Practice Update

Textbook position (Pathy's)	Current evidence (2023–2026)
Best evidence supports psyllium, osmotic laxatives (especially PEG) and lubiprostone.	AGA/ACG 2023 **upgrades PEG, sodium picosulfate, linaclotide, plecanatide and prucalopride to strong recommendations**; lubiprostone is now only **conditional**.
Magnesium salts generally avoided in elders because of hypermagnesaemia risk.	Magnesium oxide is now a **recognised evidence-based option with RCT support** — but the geriatric caution stands: avoid in CKD, and this is the answer's key nuance.
Senna listed as a stimulant option without a strong evidence base.	**Senna formally endorsed for the first time** as an evidence-based treatment.
Stool softeners of limited value; no RCT support.	**Position confirmed and strengthened** — docusate is not recommended.
Methylnaltrexone highlighted as the advance for opioid-induced constipation.	Whole **PAMORA class** (naloxegol, naldemedine, methylnaltrexone) now supported by network meta-analysis; **oral agents preferred first** for convenience in older outpatients.

Examiner's Tip — mark-earning lines

- "Stool frequency does not change with age — older people complain of straining and hard stools, and when comorbidity is controlled for, constipation is no commoner than in the young."
- "Constipation in an older person is a drug problem until proved otherwise — opiates, calcium channel blockers, NSAIDs and anticholinergics."
- "Overflow diarrhoea in an older patient is faecal impaction until a rectal examination proves otherwise; treating it as diarrhoea is a serious error."
- "Avoid bulking agents in impaction, phosphate enemas in renal impairment, and magnesium salts in CKD."
- "Biofeedback, not laxatives, is the treatment for dyssynergic defecation — around 70% respond."

Extra Points to Remember (recurring topic)

- **Doses to quote:** psyllium 3–6 g; PEG 17–34 g; lactulose 10–40 g; senna 15 mg; bisacodyl 5–10 mg; lubiprostone 8–24 µg BD.
- **Numbers to quote:** 7% of institutionalised elderly impacted; 90% of slow-transit responds to laxatives; biofeedback effective in ~70%; median 42% prevalence in long-term care.
- **Neostigmine** — reserved for hospitalised acute colonic pseudo-obstruction (Ogilvie's); produces prompt colonic decompression.
- **Probiotics:** large RCTs have failed to show significant benefit; misoprostol and colchicine are refractory-case options limited by cramping.
- **Indian practice point:** ispaghula (isabgol) is cheap and widely available and is the psyllium of choice; PEG sachets are affordable and preferable to repeated enemas in home care.

3. Gastro-oesophageal reflux disease in the elderly

15 MARKS ASKED TWICE — RECURRING

Definition and Epidemiology

1. **GORD** is the reflux of gastric contents into the oesophagus causing troublesome symptoms or mucosal complications.
2. Pathy's records that GORD is the **sixth most common disorder among elderly people in residential care**, and that around **20% of the general population experience weekly reflux symptoms**.
3. The prevalence in the elderly is **as high as in the young**, but the disease behaves differently: it **presents atypically and is more likely to be severe**.

Pathophysiology and Age-related Changes

4. The dominant mechanism at all ages is **transient lower oesophageal sphincter relaxations**, particularly after meals — not simply low resting sphincter pressure.
5. Age-related predisposing changes listed in Pathy's: **hiatus hernia in around 60% of those over 60**; **declining LOS resting pressure and intra-abdominal length**; **increased acid exposure**; reduced salivary flow; and impaired acid clearance.
6. The **number of reflux episodes is similar to the young, but their duration is longer**, reflecting impaired clearance from reduced primary and secondary peristalsis and a less compliant oesophagus.
7. **Visceral sensation declines with age** — perception of balloon distension and of acid is reduced, and cortical evoked potentials are of lower amplitude. This explains why symptoms are milder despite worse mucosal injury.
8. The refluxate may be **less acidic** because of atrophic gastritis, but its **bile content may still injure the mucosa** and contribute to Barrett's change.

Clinical Features — the Geriatric Paradox

9. **Key examination line:** Pathy's states GORD in the elderly presents with **more severe mucosal injury — erosive oesophagitis, stricture or Barrett's oesophagus — yet symptoms are characteristically milder or qualitatively different**.
10. Consequently **dysphagia, vomiting, respiratory difficulty, weight loss and anaemia are not uncommon presenting features**, while **heartburn occurs less often than in the young**.
11. In the general population heartburn or regurgitation has a sensitivity of about **70%** for GORD but low specificity against 24-hour pH monitoring; comparable data for an ageing population are not available.
12. **Atypical/extra-oesophageal manifestations:** chronic cough, asthma, laryngitis and non-cardiac chest pain — mediated either directly by acid-pepsin or by vagal reflexes from oesophageal acid exposure.
13. **Alarm symptoms mandating prompt endoscopy:** dysphagia, anorexia, weight loss, haematemesis, anaemia. Pathy's states the **threshold for endoscopy in older patients should be lower than in the young**.

Investigations

14. **Endoscopy** is the first investigation in older patients — it grades oesophagitis (Los Angeles A–D), detects stricture, Barrett's and malignancy, and permits biopsy and dilatation.
15. **Trial of double-dose PPI for 2–8 weeks** (depending on symptom frequency) is a useful and cost-effective initial test in atypical presentations and non-cardiac chest pain, with sensitivity and specificity up to **80%**.
16. **Oesophageal manometry and ambulatory pH monitoring** are indicated when PPIs fail — manometry is particularly useful to **exclude achalasia** before considering surgery.
17. Exclude cardiac disease first in any older patient with chest pain; also consider musculoskeletal, pulmonary, pericardial, gastric and biliary causes.

Management

18. **Lifestyle:** weight reduction, head-end elevation, avoidance of late meals, smoking and alcohol; review drugs that lower LOS pressure (calcium channel blockers, nitrates, anticholinergics) and drugs causing pill oesophagitis.
19. **PPIs are the drugs of choice.** Pathy's states there is **no significant difference in healing capacity between older and younger patients**, that **PPIs retain superiority over H2 receptor antagonists**, and that **no dose adjustment is needed for age-related renal or hepatic change**.
20. Important geriatric caveat: **downward dose titration is less appropriate than in the young**, especially where the initial symptoms were mild or the GORD is complicated — because symptom severity underestimates mucosal disease.
21. **Adverse effects of long-term PPI use** (Pathy's grading of causality):
    - **Causal relationship likely:** acute interstitial nephritis, benign gastric fundic gland polyps, increased enteric

infections.
    - **Possible:** *Clostridioides difficile* infection, B12 deficiency, hypomagnesaemia/hypocalcaemia.
    - **Not supported by strong evidence:** osteoporosis and hip fracture, community-acquired pneumonia, dementia, CKD progression.
    - Clopidogrel interaction is of **minimal significance except with omeprazole**.
22. **Deprescribing principle:** PPIs are often over-prescribed and the indication for ongoing therapy should be **reviewed periodically**. H2RAs are an alternative but are less potent and can cause **mental status change in renal or hepatic dysfunction**.
23. **Prokinetics have no established role** in GORD treatment.
24. **Surgery:** laparoscopic fundoplication is an option, with outcomes and complication rates in patients over 70 comparable to those under 60. However, **patients with ineffective oesophageal motility risk post-operative dysphagia**, and long-term medical therapy is likely more cost-effective in older patients given the fewer remaining years of therapy needed.
25. **Endoscopic anti-reflux procedures have not fulfilled their initial promise** and should be restricted to clinical trials.

Latest Updates (2023–Present)

- **Lyon Consensus 2.0 (2024)** redefined conclusive evidence of GORD: **LA grade B, C or D oesophagitis** now counts as conclusive, while **LA grade A is no longer accepted as diagnostic** and requires confirmation by ambulatory reflux monitoring *(Gut, 2024)*.
- **Potassium-competitive acid blockers (P-CABs)** have entered practice. **Vonoprazan received FDA approval in November 2023** for erosive GORD and in **July 2024 for heartburn in non-erosive GORD** — the first major advance in GORD therapy in over three decades *(FDA / Pharmaceuticals, 2025)*.
- The **AGA Clinical Practice Update on integrating P-CABs (2024)** advises that P-CABs may be used for healing and maintenance in **more severe (LA grade C/D) erosive oesophagitis**, where they may be superior and act faster than PPIs — but notes that **markedly higher cost and lack of comparison with double-dose PPI mean they are not routine first-line therapy** *(Gastroenterology, 2024)*.
- P-CABs give **rapid, potent, food-independent and sustained acid suppression** without needing acid activation, and act on both resting and activated proton pumps *(Front Physiol, 2025)*.
- The **VISION study** provides the only 5-year safety data for a P-CAB: maintenance vonoprazan produced higher gastrin levels with parietal and foveolar cell hyperplasia, but **no excess malignant epithelial change or gastric neuroendocrine tumours** *(Clin Gastroenterol Hepatol, 2025)*.
- The **2025 focused update of the Seoul Consensus** recommends either a **standard-dose PPI or a P-CAB for 4–8 weeks** as initial therapy for NERD or mild erosive oesophagitis, with P-CABs offering superior healing in severe erosive disease *(J Neurogastroenterol Motil, 2025)*.
- The **ACG Clinical Guideline for GORD (2022)** remains the reference standard for diagnosis and management and continues to emphasise **de-escalation to the lowest effective dose and periodic review of PPI indication** *(Am J Gastroenterol, 2022)*.

Current Practice Update

Textbook position (Pathy's)	Current evidence (2023–2026)
PPIs are the mainstay and are superior to H2 receptor antagonists; no newer class discussed.	A new class — **P-CABs (vonoprazan, tegoprazan, fexuprazan, keverprazan, zastaprazan)** — is now available, with **superior and faster healing in LA grade C/D oesophagitis**; cost restricts routine first-line use.
Endoscopic grading of oesophagitis taken at face value.	**Lyon 2.0 (2024)**: LA grade A is no longer conclusive for GORD; only **grades B, C and D** are diagnostic, so pH monitoring is needed if grade A is the only finding.
Long-term PPI adverse effects graded as likely/possible/unsupported.	**Framework unchanged and reinforced**; guidelines now stress structured deprescribing and periodic review rather than indefinite continuation.
Fundoplication safe in over-70s but medical therapy more cost-effective.	**Position unchanged.** Endoscopic anti-reflux therapy still not recommended outside trials.
H. pylori eradication not emphasised in the reflux context.	AGA 2024 advises **P-CABs should replace PPIs in H. pylori eradication regimens for most patients** — pooled first-line eradication 92% with vonoprazan versus 80% with PPI.

Examiner's Tip — mark-earning lines

- "The geriatric paradox of GORD: **more severe mucosal injury with milder symptoms**, because visceral sensation declines with age."
- "Because symptoms underestimate disease, **the threshold for endoscopy must be lower and dose reduction is less appropriate** than in younger patients."
- "Hiatus hernia is present in around 60% of those over 60."
- "Long-term PPI risks should be graded, not recited — likely for interstitial nephritis and enteric infection, unproven for dementia and hip fracture."
- "Prokinetics have no established role in GORD."

Extra Points to Remember (asked twice — very high yield)

- **Pill oesophagitis** is a geriatric differential for odynophagia: potassium chloride, tetracyclines, aspirin, NSAIDs, quinidine, theophylline, ferrous sulfate, alendronate and now **dabigatran** (extensive mucosal sloughing). Prevent by taking tablets upright with a full glass of water.
- **Barrett's oesophagus** — intestinal metaplasia; more prevalent in elderly reflux; surveillance decisions must be individualised against life expectancy and fitness for intervention.
- **Scleroderma oesophagus** — 80% have dysmotility with very low LOS pressure and severe reflux; **high-dose PPI is often required**.
- **Extra-oesophageal GORD** is a recognised cause of chronic microaspiration, worsening asthma and fibrotic lung disease, and diffuse aspiration bronchiolitis.
- **PPI–pneumonia association:** PPI use may impart about a **1.5-fold risk of community-acquired pneumonia**, greatest in the first month after initiation — an important prescribing caution in frail elders.
- **Indian practice:** pantoprazole and rabeprazole are the cheapest and most available PPIs; vonoprazan is marketed in India but cost limits routine use.

4. Gastrointestinal motility disorders in the elderly

15 MARKS

General Principle

1. The core teaching point of Pathy's chapter is that **gastrointestinal motor function is relatively well preserved with healthy ageing, but there is a general decline in visceral sensation**.
2. **Systemic illness and medication — not age — impair gut function in the elderly.** Consequences include swallowing disorders, impaired nutrition, altered drug absorption and altered bowel habit.
3. Sensory decline is demonstrable throughout the gut: reduced perception of balloon distension in the **oesophagus, stomach and rectum**, probably from loss of intrinsic sensory enteric neurons plus altered central processing. Perception of **chemical stimuli such as acid also decreases**.

Oesophageal Motility Disorders

4. In those over 60, **upper oesophageal sphincter resting pressure is lower but shows less swallow-induced relaxation**, indicating a degree of UOS resistance; reference ranges from young subjects must not be applied to older patients.
5. **Reflex UOS contraction in response to laryngeal stimulation is impaired** — a mechanism predisposing to aspiration; the frequency but not the magnitude of the response falls, implicating the sensory limb.
6. In the distal oesophagus there is **reduced contraction amplitude and more frequent failed peristalsis**; secondary peristalsis is less easily elicited. These changes begin as early as **age 40**.
7. **Achalasia:** peak incidence is in early-to-mid adulthood, but **a second smaller peak occurs in the elderly**. It presents with dysphagia for both liquids and solids; in older patients **weight loss, regurgitation and aspiration** are prominent while **chest pain is less common**.
8. **Diagnosis of achalasia:** barium swallow shows a dilated oesophagus with **'bird's beak' or 'rat's tail' tapering**; manometry shows **absent or incomplete LOS relaxation** with type I (absent contractions), type II (pan-oesophageal pressurisation) or type III (premature spastic contractions).
9. **Always exclude pseudo-achalasia** — carcinoma of the distal oesophagus or cardia mimicking achalasia. A **short history with disproportionate weight loss** is the clue; endoscopy, and sometimes endoluminal ultrasound or CT, is mandatory in the elderly.
10. **Treatment of achalasia: pneumatic dilatation** and **laparoscopic myotomy** are the most efficacious for types I and II, each with about a **1% perforation rate**. **POEM** allows calibrated myotomy and is particularly useful in **type III**. In a consecutive series of 51 patients aged ≥65 undergoing laparoscopic myotomy there were **no deaths, few complications and a median stay of 3 days**. For the **frail elderly**, **endoscopic botulinum toxin injection into the LOS** is a safe alternative.
11. **Distal oesophageal spasm** is more commonly diagnosed over age 50 and gives a **corkscrew appearance on barium** with premature contractions on manometry.
12. **Minor disorders — ineffective oesophageal motility and fragmented peristalsis** — account for **more than one-third of presentations with dysphagia in patients over 65**. Radiographic and manometric abnormalities correlate poorly with symptoms, no specific therapy exists, and management is **acid suppression plus optimisation of nutrition**.

Gastric and Small-Intestinal Motility

13. **Only a modest slowing of gastric emptying** occurs with healthy ageing, confirmed longitudinally. However, altered perception of gastric distension and enhanced humoral responses to duodenal nutrients contribute to the **anorexia of ageing**.
14. Intraduodenal nutrients stimulate **greater cholecystokinin release** in healthy elderly, intraduodenal glucose is **more satiating**, and higher rates of *H. pylori* infection and atrophic gastritis are associated with **lower ghrelin** — a triad worth quoting for anorexia of ageing.
15. **Drug absorption:** slow emptying and altered gastric pH may affect absorption of oral medication; paracetamol absorption is slightly slower, but **benzodiazepines, tetracycline and L-dopa absorption are not significantly altered by age**.
16. **Small intestine:** MMC periodicity is preserved in the healthy elderly; phase III propagation velocity is modestly slower, and there are more propagated clustered contractions — a pattern also seen in IBS, of unclear significance. **Transit is comparable to the young**, in contrast to the delayed colonic transit characteristic of ageing.
17. **Postprandial hypotension** can legitimately be classified as a gastrointestinal motility disorder — the fall in blood pressure relates to splanchnic blood flow regulation and gut peptide release, is worst in **type 2 diabetes with autonomic neuropathy**, and is an **under-recognised cause of syncope and falls**. It is attenuated by **octreotide**,

and by measures slowing gastric emptying and carbohydrate absorption (e.g. **GLP-1 receptor agonists such as lixisenatide**, guar, acarbose).

Motility Disorders in Systemic Disease

18. **Diabetes:** gastroparesis impairs nutrient and drug absorption and destabilises glycaemia; diagnose by endoscopy first to exclude obstruction, then **scintigraphic gastric emptying**. Treat with prokinetics — **metoclopramide, domperidone or erythromycin (a motilin agonist)**. **Cisapride was withdrawn for arrhythmia risk.** Pyloric botulinum toxin is of unclear benefit and **less likely to help patients over 50**; gastric electrical stimulators lack adequate controlled evidence.
19. Up to **80% of patients with diabetic gastroparesis have abnormal small-intestinal motility**. In diabetic diarrhoea, specifically exclude **SIBO, coeliac disease and pancreatic exocrine insufficiency**.
20. **Progressive systemic sclerosis:** peak incidence in the fifth and sixth decades; GI involvement in the majority. **Oesophageal dysmotility in about 80%** with very low LOS pressure and severe reflux; gastroparesis, pseudo-obstruction, bacterial overgrowth, malnutrition, constipation and faecal incontinence may all occur. Smooth muscle atrophy and fibrosis plus **antibodies to M3 muscarinic receptors** are implicated. High-dose PPI is often needed; prokinetics help when transit is delayed.
21. **Parkinson's disease** causes dysphagia, gastroparesis and constipation, and is a major contributor to bowel dysfunction; amyloidosis and other connective tissue diseases produce similar patterns.

Functional Gastrointestinal Disorders

22. Functional disorders are defined by **Rome IV** and are often overlooked in the elderly. **10–15% of people over 70 have IBS** in community surveys; prevalence is greater in women at all ages.
23. **Organic disease must be rigorously excluded** — its prevalence is higher than in the young. Have a low threshold for colonoscopy with altered bowel habit, and remember that **mesenteric ischaemia occurs more often in patients with IBS than without**.
24. **Treatment:** low-FODMAP diet is first-line for IBS but almost unstudied in the elderly; antispasmodics (hyoscine, mebeverine, peppermint oil) and **low-dose tricyclics** have a good evidence base, with SSRIs better tolerated than tricyclics in older patients. **Placebo response is 20–70%**, so judge all therapies against it.
25. **Opiates must be avoided** for chronic abdominal pain — ineffective, and causing tolerance, opioid-induced hyperalgesia and narcotic bowel syndrome. Beware anticholinergic burden: sedation, urinary retention, postural hypotension, blurred vision and glaucoma.

Latest Updates (2023–Present)

- The **Chicago Classification v4.0** is now the manometric standard, requiring **both supine and upright swallows and provocative testing (multiple rapid swallows, rapid drink challenge)**, and re-labelling ineffective oesophageal motility with stricter criteria — reducing over-diagnosis of "minor motility disorders" in older patients *(Neurogastroenterol Motil, current standard)*.
- **Lyon Consensus 2.0 (2024)** integrates motility and reflux assessment, altering how manometric findings are interpreted alongside pH-impedance data *(Gut, 2024)*.
- The **ACG guideline on gastroparesis (2022)** confirms **4-hour scintigraphy as the diagnostic standard**, recommends metoclopramide at the lowest effective dose with attention to tardive dyskinesia risk, and does not support routine botulinum toxin — consistent with Pathy's scepticism *(Am J Gastroenterol, 2022)*.
- **GLP-1 receptor agonists** are now a widespread iatrogenic cause of delayed gastric emptying in older diabetics; this is clinically relevant both for symptoms and for **peri-endoscopic and peri-operative aspiration risk**, prompting society guidance on withholding these agents before procedures *(ASA/multisociety guidance, 2023–2024)*.
- For achalasia, contemporary comparative data continue to support **POEM for type III** and **laparoscopic Heller myotomy or pneumatic dilatation for types I and II**, with POEM carrying a higher rate of post-procedure reflux — relevant when choosing therapy in an older patient already prone to severe GORD *(contemporary achalasia literature, 2023–2025)*.

Current Practice Update

Textbook position (Pathy's)	Current evidence (2023–2026)
Chicago Classification v3.0 used for manometric diagnosis.	**Chicago v4.0** now standard — mandates upright and supine swallows plus provocative manoeuvres, and tightens criteria for ineffective oesophageal motility.
Pyloric botulinum toxin and gastric electrical stimulation of unproven value.	**Position confirmed** by ACG 2022 gastroparesis guideline; neither is recommended as routine therapy.
Postprandial hypotension attenuated by octreotide and by GLP-1 agonists slowing gastric emptying.	Same mechanism now cuts both ways — **widespread GLP-1 agonist use is itself a common cause of delayed emptying**, requiring pre-procedural withholding to reduce aspiration risk.
POEM emerging, with role yet to be defined.	**POEM established**, especially for type III achalasia; higher post-procedural reflux rates make patient selection important in the elderly.

Examiner's Tip — mark-earning lines

- "Motor function is largely preserved with healthy ageing; **it is visceral sensation that declines** — and that decline explains atypical presentations across the gut."
- "In an older patient with achalasia, always exclude pseudo-achalasia — a short history with disproportionate weight loss is the warning."
- "Minor motility disorders account for over a third of dysphagia presentations after 65, and manometric findings correlate poorly with symptoms."
- "Postprandial hypotension is a gastrointestinal cause of falls and syncope in the elderly."
- "For the frail elderly with achalasia, botulinum toxin injection is the safe option."

5. Approach to upper gastrointestinal bleeding in the elderly

15 MARKS

Definition and Significance

1. **Upper GI bleeding (UGIB)** is bleeding proximal to the ligament of Treitz, presenting as haematemesis, coffee-ground vomiting, melaena, or occasionally haematochezia when brisk.
2. Pathy's places this in context: **bleeding causes or contributes to the death of about 5% of the elderly population**, chiefly from intracranial and subarachnoid haemorrhage, ruptured aortic aneurysm, and **gastrointestinal bleeding from peptic ulcers and gastric or colonic malignancy**.
3. A crucial principle: **most bleeding in the elderly is localised and due to a specific underlying pathology, frequently malignancy** — primary haemostatic disorders are relatively rare.

Causes

4. **Peptic ulcer disease** — the commonest cause; driven in older people by **NSAIDs, aspirin, corticosteroids and *H. pylori***. Pathy's tabulates aspirin, NSAIDs (ibuprofen, diclofenac, naproxen) and corticosteroids as carrying an **enhanced risk of peptic ulceration**, and antiplatelets, DOACs, heparins and thrombolytics as carrying an **enhanced risk of bleeding**.
5. **Erosive gastritis and duodenitis** — drug-related and stress-related.
6. **Oesophageal and gastric varices** — in India a major cause, from cirrhosis and also from **non-cirrhotic portal fibrosis and extrahepatic portal venous obstruction**.
7. **Oesophagitis, including pill oesophagitis** — Pathy's notes perforation and bleeding as complications, particularly with potassium chloride, quinidine and non-steroidal drugs.
8. **Malignancy** — gastric and oesophageal carcinoma; must always be excluded in this age group.
9. **Mallory–Weiss tear, Dieulafoy lesion, angiodysplasia, GAVE (watermelon stomach), aorto-enteric fistula** after aortic graft.

Why the Elderly Do Worse

10. **Reduced physiological reserve** — blunted tachycardic response (especially on beta-blockers), so hypotension appears late and shock is under-recognised.
11. **Polypharmacy and antithrombotics** — anticoagulants and antiplatelets both increase incidence and severity; warfarin effects are enhanced by age over 80, weight loss, acute illness, heart failure, renal and hepatic impairment.
12. **Comorbidity** — ischaemic heart disease limits tolerance of anaemia; CKD limits fluid loading; cirrhosis worsens coagulopathy.
13. **Atypical presentation** — syncope, falls, confusion, angina or breathlessness may be the only manifestation, with melaena discovered only on rectal examination.

Initial Assessment and Resuscitation

14. **ABC first:** two wide-bore cannulae, crystalloid resuscitation, oxygen, protect the airway in active haematemesis or encephalopathy; hourly urine output and continuous monitoring.
15. **Bloods:** CBC, cross-match, urea and creatinine (a **disproportionately raised urea supports an upper source**), LFTs, coagulation profile, and consider troponin/ECG in older patients with ischaemic risk.
16. **Risk stratification with the Glasgow–Blatchford Score (GBS):** patients with **GBS 0–1** are at very low risk of rebleeding, death or needing intervention and may be managed as outpatients. The **Rockall score** is used for post-endoscopic risk.
17. **Transfusion:** a **restrictive strategy with a haemoglobin threshold of 7 g/dL** is recommended in haemodynamically stable patients, targeting a post-transfusion haemoglobin of **7–9 g/dL**. In those with acute or chronic **cardiovascular disease, use a more liberal threshold of 8 g/dL**.
18. **Pre-endoscopic erythromycin:** a single **250 mg intravenous dose 30–120 minutes before endoscopy** improves visualisation, reduces the need for second-look endoscopy, reduces units transfused and shortens hospital stay.
19. **Pre-endoscopic PPI:** ACG could not reach a recommendation for or against it; in practice a high-dose PPI infusion is started while awaiting endoscopy and downgrades stigmata.
20. **Antithrombotic management:** withhold vitamin K antagonists; for haemodynamic instability give **four-factor prothrombin complex concentrate** (rapid onset) rather than routine FFP, which carries volume overload and TRALI risk. Do **not routinely give vitamin K** in acute bleeding because of slow onset and later difficulty re-anticoagulating. Do **not routinely transfuse platelets**. **Do not stop aspirin used for secondary cardiovascular prevention** — cessation increases 30-day mortality; if held, restart within 3–5 days. For dual antiplatelet therapy, continue aspirin and hold the second agent, restarting preferably within 5 days.

21. **Tranexamic acid is not recommended** in acute GI bleeding.

Endoscopy and Definitive Therapy

22. **Timing:** endoscopy **within 24 hours** of presentation for admitted patients. **Very early endoscopy (<12 hours)** may be considered with persistent haemodynamic instability, in-hospital bloody emesis, or when anticoagulation cannot be interrupted.
23. **Forrest classification** guides therapy: Ia spurting, Ib oozing, IIa non-bleeding visible vessel, IIb adherent clot, IIc flat pigmented spot, III clean base. **Endoscopic therapy is indicated for Ia, Ib and IIa.**
24. **Endoscopic modalities:** combination therapy (dilute adrenaline injection **plus** a second modality) — bipolar electrocoagulation, heater probe, absolute ethanol injection, clips, argon plasma coagulation, soft monopolar coagulation. **Hemostatic powder spray (TC-325)** is suggested for actively bleeding ulcers and **over-the-scope clips** for recurrent bleeding after previous successful haemostasis.
25. **Post-endoscopic PPI: high-dose PPI continuously or intermittently for 3 days**, then twice-daily oral PPI for the first 2 weeks after endoscopy.
26. **Variceal bleeding:** resuscitate, give **terlipressin or octreotide** plus **prophylactic antibiotics (ceftriaxone)**, and perform **endoscopic band ligation**; consider early TIPS in high-risk patients. Gastric varices are treated with cyanoacrylate glue injection.
27. **Failure of endoscopic therapy:** repeat endoscopy for rebleeding; if this fails, **transcatheter arterial embolisation** is preferred to surgery in the elderly, with surgery reserved for uncontrollable bleeding or perforation.

Secondary Prevention

28. **Test for and eradicate *H. pylori*** in all ulcer bleeding, and confirm eradication. Stop NSAIDs; if they are essential, co-prescribe a PPI or use a COX-2 inhibitor with PPI cover.
29. **Re-evaluate the indication for every antithrombotic**, restart at the earliest safe time with PPI co-prescription, and biopsy or repeat endoscopy for gastric ulcers to exclude malignancy.

Latest Updates (2023–Present)

- **ACG guideline (2021)** and **ESGE guideline (2021)** remain the operative standards: GBS 0–1 for outpatient management, transfusion threshold 7 g/dL, endoscopy within 24 hours, high-dose PPI for 3 days post-haemostasis, and TC-325 / over-the-scope clips as newer haemostatic options. The **ESGE guideline is under review for update** *(Am J Gastroenterol 2021; Endoscopy 2021)*.
- A **2023 systematic review and meta-analysis** confirmed restrictive transfusion does not increase in-hospital or 30-day mortality or rebleeding, with fewer transfusion reactions and post-transfusion interventions; **thresholds above 8 g/dL may produce more untoward outcomes** *(Sci Rep, 2023)*.
- **ESGE (2022) variceal guideline** found FFP transfusion in variceal haemorrhage was associated with **significantly increased 42-day mortality**, and advises against routine platelet transfusion or a fixed platelet threshold *(Endoscopy, 2022)*.
- **Vonoprazan** has been compared with intravenous PPI for prevention of rebleeding from high-risk peptic ulcers after successful endoscopic haemostasis, with promising results — an emerging alternative where available *(comparative trial data cited in AGA P-CAB update, 2024)*.
- A **2025 network meta-analysis** examined prokinetics with or without nasogastric lavage versus lavage alone for improving endoscopic visualisation, supporting **erythromycin** as the agent of choice *(network meta-analysis, 2025)*.
- A **2025 study of 297 patients over 65** confirmed that antiplatelet, anticoagulant and NSAID use meaningfully alters transfusion need, endoscopic treatment requirement, rebleeding rates and 30-day mortality in geriatric UGIB — reinforcing structured medication review *(Medicine (Baltimore), 2025)*.
- The **ACG lower GI bleeding guideline update (2023)** reinforced the same principles for the lower tract — risk stratification for early discharge, restrictive transfusion at 7 g/dL, and **against antifibrinolytics such as tranexamic acid** *(Am J Gastroenterol, 2023)*.

Current Practice Update

Textbook position (Pathy's)	Current evidence (2023–2026)
Peptic ulcer and malignancy identified as major causes of fatal bleeding in the elderly; no scoring system detailed.	Care is now **protocolised**: GBS 0–1 permits outpatient management; Rockall for post-endoscopic risk; Forrest class dictates endoscopic therapy.
No specific transfusion threshold given.	**Restrictive strategy at Hb 7 g/dL** is standard (target 7–9 g/dL), rising to **8 g/dL with cardiovascular disease**, supported by meta-analysis to 2023.
Antithrombotic drugs listed as bleeding risks without reversal protocols.	Explicit reversal guidance: **4F-PCC over FFP** for VKA with instability; **no routine vitamin K**, **no routine platelets**, **no tranexamic acid**; **continue secondary-prevention aspirin**.
Endoscopic haemostasis described in general terms.	Newer modalities are now guideline-endorsed: **TC-325 haemostatic powder** for active ulcer bleeding and **over-the-scope clips** for recurrent bleeding.
PPI therapy for ulcer bleeding not protocolised.	**High-dose PPI for 3 days, then twice-daily oral PPI for 2 weeks** after endoscopic haemostasis; vonoprazan is an emerging alternative.

Examiner's Tip — mark-earning lines

- "Bleeding contributes to about 5% of deaths in the elderly, and most of it is localised pathology — frequently malignancy — rather than a haemostatic disorder."
- "Quote the numbers: GBS 0–1 for discharge; transfuse below 7 g/dL, or 8 g/dL with cardiovascular disease; endoscopy within 24 hours; erythromycin 250 mg IV 30–120 minutes pre-procedure."
- "Do not stop secondary-prevention aspirin — cessation increases 30-day mortality."
- "PCC, not FFP, for warfarin reversal with haemodynamic instability; no routine tranexamic acid."
- "In India, always consider variceal bleeding from cirrhosis, non-cirrhotic portal fibrosis and EHPVO alongside peptic ulcer."

6. Haematemesis

7 MARKS

1. **Haematemesis** is vomiting of frank blood or coffee-ground material, indicating bleeding proximal to the ligament of Treitz. It is **an alarm symptom mandating prompt endoscopy at any age**, and Pathy's specifically lists it among the alarm symptoms requiring urgent investigation.
2. **Distinguish from mimics:** haemoptysis (frothy, bright red, alkaline, with cough) and swallowed blood from epistaxis or oropharyngeal bleeding. Coffee-ground appearance reflects acid conversion of haemoglobin to haematin.
3. **Common causes in the elderly:** peptic ulcer (NSAIDs, aspirin, steroids, *H. pylori*), erosive gastritis, oesophageal and gastric varices, oesophagitis including pill oesophagitis, Mallory–Weiss tear, Dieulafoy lesion, angiodysplasia, and **gastric or oesophageal malignancy**.
4. **Assess severity immediately:** pulse, blood pressure, postural drop, capillary refill, urine output and mental state. Remember the blunted tachycardic response in older patients and those on beta-blockers — **shock is under-recognised**.
5. **Resuscitate first, investigate second:** two wide-bore cannulae, crystalloid, oxygen, cross-match, and airway protection with massive haematemesis or encephalopathy. Send CBC, urea and creatinine (raised urea supports an upper source), LFTs and coagulation.
6. **Risk stratify with the Glasgow–Blatchford Score** — a score of **0–1 identifies very-low-risk patients** suitable for outpatient management; all others are admitted.
7. **Transfuse restrictively** at a haemoglobin threshold of **7 g/dL** (target 7–9 g/dL), or **8 g/dL if there is cardiovascular disease**. Withhold anticoagulants, use **PCC rather than FFP** for warfarin reversal with instability, and **continue aspirin prescribed for secondary prevention**.
8. **Endoscopy within 24 hours** (earlier if unstable), preceded by **erythromycin 250 mg IV** to clear the stomach. Apply **combination endoscopic haemostasis** for Forrest Ia, Ib and IIa lesions, then **high-dose PPI for 3 days followed by twice-daily oral PPI for 2 weeks**.
9. **If variceal:** terlipressin or octreotide, prophylactic ceftriaxone, and band ligation; consider early TIPS. If endoscopy fails, proceed to **transcatheter embolisation** before surgery.
10. **Prevention:** eradicate *H. pylori*, stop NSAIDs, co-prescribe PPI where antithrombotics must continue, and **always biopsy or re-scope a gastric ulcer to exclude malignancy**.

Latest Updates (2023–Present) — concise

- **Restrictive transfusion at 7 g/dL is confirmed safe** by 2023 meta-analysis, with no excess mortality or rebleeding and fewer transfusion reactions; thresholds above 8 g/dL may worsen outcomes *(Sci Rep, 2023)*.
- **Tranexamic acid is not recommended** in acute GI bleeding, and **FFP in variceal haemorrhage is associated with increased 42-day mortality** *(ESGE, 2021–2022; ACG, 2023)*.
- **Vonoprazan** is emerging as an alternative to intravenous PPI for preventing rebleeding from high-risk ulcers after endoscopic haemostasis *(cited in AGA P-CAB Clinical Practice Update, 2024)*.

Current Practice Update

Textbook position	Current evidence
Haematemesis treated as an alarm symptom requiring prompt endoscopy.	Unchanged — but now with a **defined 24-hour window** and formal risk scoring for safe outpatient management.
No transfusion or reversal protocol specified.	**Hb 7 g/dL threshold (8 g/dL with cardiovascular disease); PCC over FFP; no routine platelets, vitamin K or tranexamic acid.**

Examiner's Tip

- "Resuscitation precedes endoscopy — but endoscopy within 24 hours changes outcome."
- "A raised urea with normal creatinine supports an upper gastrointestinal source."
- "Never discharge an older patient with haematemesis on symptom resolution alone — score them."

7. Assessment of a patient's nutrition — methods, advantages and disadvantages

15 MARKS

Principles

1. Nutritional assessment in older adults is a **two-step process**: **screening** to identify those at risk, followed by **assessment** for diagnosis and severity grading.
2. Essentials of Clinical Geriatrics states plainly that anthropometric variables **cannot provide a complete description of nutritional status and are not highly correlated with biochemical or haematological indicators** — so no single method suffices, and a combination is always required.
3. The critical questions to answer are: is there reason to suspect malnutrition; of which nutrient and to what extent; what is the pathophysiological mechanism (intake, digestion and absorption, metabolism, excretion or requirement); and what aetiology underlies it.
4. Distinguish **starvation** from **cachexia**: the hallmark of starvation is a **rapid response to refeeding**, whereas **cachexia is remarkably resistant to hypercaloric feeding**. In cachexia, appetite is suppressed early, BMI predicts mortality, albumin and transthyretin fall early, CRP is elevated and inflammatory disease is present.

Method 1 — Clinical History and Examination

5. **Content:** dietary history and 24-hour recall, unintentional weight loss, appetite, chewing and swallowing difficulty, dentition, gastrointestinal symptoms, functional ability to shop and cook, social isolation, alcohol, depression, and a full drug review.
6. **Advantages:** free, universally available, identifies the reversible cause, and detects the social and functional determinants that no laboratory test can.
7. **Disadvantages:** subjective, time-consuming, and **unreliable in dementia or delirium** where recall is impaired.

Method 2 — Anthropometry and Body Composition

8. The methods and what each measures: **weight** — global; **body mass index** — total body fat; **skin folds** — percentage fat; **waist:hip ratio** — central adiposity; **upper arm circumference** — lean body mass.
9. **Weight** is easily obtained and useful, but only **in the absence of oedema**. Serial weights are more informative than a single value.
10. **Upper arm circumference correlates with lean body mass** and is **particularly helpful in oedematous patients in whom weight is misleading** — but the effect of ageing on lean mass is so great that it remains a poor reflection of nutritional status in older adults.
11. **Calf circumference** is an accepted surrogate for muscle mass and is used in the MNA when BMI is unavailable.
12. **Height substitutes:** where standing height cannot be measured, use **knee-heel height** equations — Men = (2.02 × knee height in cm) × (0.04 × age) + 64.19; Women = (1.83 × knee height in cm) × (0.24 × age) + 84.88. For amputees, add the stated percentage for the missing part (single below knee 6.0%, at knee 9.0%, above knee 15.0%, single arm 6.5%, arm below elbow 3.6%).
13. **Advantages:** cheap, bedside, repeatable, no laboratory needed — ideal for Indian community and long-term care practice.
14. **Disadvantages:** confounded by **oedema, ascites, dehydration, kyphosis and vertebral collapse**; BMI is unreliable when height cannot be measured; observer variability with skin folds.

Method 3 — Biochemical Markers

15. **Serum albumin** is described as a practical indicator of malnutrition in older adults, **but liver disease, proteinuria and protein-losing enteropathy must be excluded**. A low albumin may indicate malnutrition; **a normal or raised albumin does not exclude it**.
16. **Thyroxine-binding prealbumin (transthyretin) and retinol-binding protein are more sensitive** than albumin and transferrin, because of their shorter half-lives.
17. **Anaemia** is one of the earliest manifestations of protein-energy malnutrition and should alert the physician to its possibility; **total lymphocyte count** may be a very good marker of nutritional problems.
18. **Advantages:** objective, numerical, prognostically useful.
19. **Disadvantages — the critical caveat:** all these proteins are **negative acute-phase reactants** and fall with inflammation regardless of nutrition. They therefore reflect **disease severity as much as nutritional state**, and must be interpreted alongside CRP.

Method 4 — Validated Screening and Assessment Tools

20. **Mini Nutritional Assessment (MNA):** developed specifically for elderly populations; the full MNA is

multidimensional and a **short form (MNA-SF) has similar performance**. It assesses body mass by either BMI or calf circumference. It was **96% sensitive and 60% specific** for body weight loss; a normal baseline score carried a **mortality risk of 0.35 (95% CI 0.18–0.66)** relative to an abnormal score. It is a powerful prognostic factor, particularly in cancer, and can assess cancer cachexia.

21. **Subjective Global Assessment (SGA):** combines history and physical signs. Both MNA and SGA predict mortality in geriatric patients; the **MNA classified fewer patients as well-nourished than the SGA, and those it called well-nourished had better 3-year survival** — making the MNA the more discriminating tool in older adults.
22. **Other tools: SNAQ** (Simplified Nutrition Assessment Questionnaire — a score below 14 signals significant risk of weight loss), **MUST**, and **NRS-2002** for hospital screening.
23. **Advantages:** validated, quick, reproducible, usable by nurses, and prognostically powerful.
24. **Disadvantages:** screening tools identify risk but **do not diagnose the cause**; the MNA requires cognitive cooperation and its specificity is modest at 60%.

Method 5 — Comprehensive Geriatric Assessment

25. Pathy's positions **comprehensive gerontological assessment as the framework** for complex cases with multimorbidity or frailty. It links nutrition to cognition (MMSE, MoCA), mood (GDS), delirium (CAM), falls risk (TUG, SPPB), function (Katz, Barthel, Lawton–Brody), morbidity, medication review, oral and swallowing assessment, constipation screening, and social and socioeconomic support.
26. The value of this approach is that it converts a measurement into an **action plan** — dental treatment, meal texture adaptation, help with shopping and meal preparation, meals-on-wheels, treatment optimisation and caregiver support.

Latest Updates (2023–Present)

- The **GLIM criteria** are now the global standard, endorsed by **ESPEN, ASPEN, FELANPE and PENSA**. Diagnosis requires **at least one phenotypic plus one etiologic criterion** after a positive screen. Phenotypic: **non-volitional weight loss (>5% in 6 months or >10% beyond 6 months), low BMI, low muscle mass**. Etiologic: **reduced food intake or assimilation, and inflammation or disease burden** *(Clin Nutr, 2019; JPEN, 2019)*.
- The **GLIM 5-year update (2025)** reviewed 2019–2024 evidence with a 43-member working group and ten systematic reviews, and has appointed working groups on **muscle mass cut-offs, inflammation assessment, screening procedures and food intake/assimilation** *(Clin Nutr 2025; JPEN 2025)*.
- GLIM and MNA are **not interchangeable**. A 2025 geriatric outpatient study found malnutrition rates of **6.6% by MNA-SF versus 21.1% by GLIM**, and a 2025 study of overweight geriatric outpatients found **58.8% of GLIM-defined malnutrition was missed by MNA-SF screening** — the practical message being that **a normal BMI or obesity does not exclude malnutrition** *(Ir J Med Sci 2025; Nutr Clin Pract 2025)*.
- **Sarcopenic obesity** is now a defined entity by the **ESPEN–EASO consensus (2022)**, with a prevalence of about **15.9%** in geriatric outpatients with BMI ≥25 — meaning muscle measurement, not weight, is what matters *(Clin Nutr 2022; Nutr Clin Pract 2025)*.
- The **ESPEN practical guideline on clinical nutrition and hydration in geriatrics** remains the operational reference, noting malnutrition prevalence generally **below 10% in independently living older people**, rising steeply with dependency and hospitalisation *(Clin Nutr, ESPEN)*.
- Critical care guidance now advises that **serum protein markers such as prealbumin and CRP are not recommended for evaluating nutritional status or goals**, though they retain value in series — confirming that these are inflammation markers first *(ASPEN/SCCM-derived practice, 2025)*.

Current Practice Update

Textbook position (Pathy's / Essentials)	Current evidence (2023–2026)
Serum albumin described as "a practical indicator of malnutrition" in older adults.	Albumin is now regarded primarily as an **inflammatory and prognostic marker, not a nutritional one**; GLIM excludes it from the diagnostic criteria and current guidance advises against using protein markers to set nutritional goals.
MNA and SGA are the principal validated tools.	These remain **screening tools**; **GLIM is now the diagnostic standard** and detects two to three times more malnutrition than MNA-SF alone in geriatric outpatients.
Body composition assessed by BMI, skin folds, waist:hip and arm circumference.	Emphasis has shifted to **muscle mass and function** — calf circumference, hand-grip strength, BIA and DXA — reflecting sarcopenia (EWGSOP2) and sarcopenic obesity (ESPEN–EASO).
Low BMI central to identifying malnutrition.	**Malnutrition frequently coexists with obesity**; GLIM identifies cases that BMI-based screening misses entirely.

Examiner's Tip — mark-earning lines

- "Nutritional assessment is a two-step process — screen, then diagnose — and no single measure is adequate."
- "A low albumin may indicate malnutrition, but a normal albumin does not exclude it; prealbumin and retinol-binding protein are more sensitive but are still acute-phase reactants."
- "Upper arm circumference is the measurement of choice when oedema makes weight misleading."
- "The MNA is 96% sensitive but only 60% specific — quote both figures."
- "State the GLIM framework explicitly: one phenotypic plus one etiologic criterion after a positive screen."

8. Mini Nutritional Assessment — components, scoring and interpretation

7 MARKS

1. The **Mini Nutritional Assessment (MNA)** was developed specifically **to assess malnutrition in elderly populations**. The full MNA is a **multidimensional tool** identifying older people who are malnourished or at risk, **whatever the cause**; a **short form (MNA-SF) has similar performance**, and a six-item version has equivalent predictive value.
2. **Structure:** the MNA-SF is a **6-item screening section scored out of 14 points**. If screening is abnormal, the full MNA (total 30 points) is completed with additional assessment items covering living situation, medication, pressure sores, meal frequency, protein and fluid intake, self-perceived health and nutrition, and mid-arm and calf circumference.
3. **The six screening components (A–F):**
   - **A — Food intake decline** over the past 3 months due to loss of appetite, digestive problems, or chewing/swallowing difficulty: 0 = severe decrease, 1 = moderate decrease, 2 = no decrease.
   - **B — Weight loss** in the last 3 months: 0 = >3 kg, 1 = does not know, 2 = 1–3 kg, 3 = no weight loss.
   - **C — Mobility:** 0 = bed or chair bound, 1 = able to get out of bed/chair but does not go out, 2 = goes out.
   - **D — Psychological stress or acute disease** in the past 3 months: 0 = yes, 2 = no.
   - **E — Neuropsychological problems:** 0 = severe dementia or depression, 1 = mild dementia, 2 = no psychological problems.
   - **F1 — Body mass index:** 0 = BMI <19; 1 = 19 to <21; 2 = 21 to <23; 3 = BMI ≥23.
   - **F2 — Calf circumference** (used **only if BMI is not available**): 0 = CC <31 cm; 3 = CC ≥31 cm. **Do not answer F2 if F1 has already been completed.**
4. **Interpretation of the screening score (maximum 14 points):**
   - **12–14 points — normal nutritional status** (no further assessment needed).
   - **8–11 points — at risk of malnutrition.**
   - **0–7 points — malnourished.**
5. **Action:** a screening score of **11 or below indicates possible malnutrition and the full MNA should be completed**; Essentials notes that where greater specificity is desired, a threshold of 10 or below may be used.
6. **Performance:** the MNA is **96% sensitive and 60% specific** for body weight loss. Subjects with a normal baseline score had a **lower mortality risk of 0.35 (95% CI 0.18–0.66)** than those with an abnormal score.
7. **Prognostic value:** those judged at risk by the MNA had **more frequent acute illness, greater need for assistance and more weight loss**. The MNA is a powerful prognostic factor across several pathologies, **particularly cancer**, and can be used to assess cancer cachexia.
8. **Advantages:** designed for and validated in older people; non-invasive, needs no blood tests; usable by nurses in wards, clinics and long-term care; incorporates **cognitive, psychological and mobility domains** that other tools omit; and offers **calf circumference as a substitute when BMI cannot be measured** — a decisive practical advantage in bed-bound, kyphotic or amputee patients.
9. **Disadvantages: specificity is only 60%**, so it over-identifies risk; it requires cognitive cooperation and is difficult in severe dementia; the acute-illness item may score positively for reasons unrelated to nutrition; and it is a **screening tool, not a diagnosis** — it does not establish the cause.

Latest Updates (2023–Present) — concise

- The MNA is now positioned as a **screening tool feeding into GLIM diagnosis**, not as a stand-alone diagnostic instrument; GLIM requires one phenotypic plus one etiologic criterion after a positive screen *(Clin Nutr 2019; GLIM 5-year update, Clin Nutr / JPEN 2025)*.
- **MNA-SF substantially under-detects malnutrition compared with GLIM**: a 2025 geriatric outpatient study reported **6.6% by MNA-SF versus 21.1% by GLIM**, and in overweight geriatric outpatients **58.8% of GLIM-defined malnutrition was not flagged as at-risk by MNA-SF** *(Ir J Med Sci 2025; Nutr Clin Pract 2025)*.
- The BMI-based item is the main limitation in **sarcopenic obesity**, now formally defined by the **ESPEN–EASO consensus** and present in about **15.9%** of geriatric outpatients with BMI ≥25 — supporting the addition of muscle mass or hand-grip measurement alongside the MNA *(Clin Nutr 2022; Nutr Clin Pract 2025)*.

Current Practice Update

Textbook position (Pathy's / Essentials)	Current evidence (2023–2026)
MNA is the validated multidimensional tool for identifying malnutrition in the elderly.	Retained as the **preferred geriatric screening tool**, but **GLIM is now the diagnostic standard**; a normal MNA-SF does not exclude malnutrition.
Body mass assessed by BMI or calf circumference within the tool.	Current practice adds **hand-grip strength and muscle mass** measurement, because BMI-based scoring misses malnutrition in overweight and sarcopenic-obese older adults.

Examiner's Tip

- Reproduce the **three cut-offs verbatim**: 12–14 normal; 8–11 at risk; 0–7 malnourished — these carry the marks.
- "Calf circumference replaces BMI only when BMI is unavailable — never score both F1 and F2."
- "96% sensitive, 60% specific: a good screen, a poor confirmatory test."

9. Total parenteral nutrition

15 MARKS

Definition and Governing Principle

1. **Total parenteral nutrition (TPN)** is the intravenous delivery of the complete nutritional requirement — water, carbohydrate, amino acids, lipid, electrolytes, vitamins and trace elements — when the gastrointestinal tract cannot be used at all. **Partial or supplemental PN** supplements inadequate enteral intake.
2. **The governing principle is "if the gut works, use it."** Pathy's records a **downward trend in the use of parenteral nutrition in critical care over recent years, chiefly because of studies showing higher morbidity with parenteral compared with enteral nutrition**.
3. In acute pancreatitis this hierarchy is explicit: **early oral feeding is preferred; enteral tube feeding (nasogastric or nasojejunal) within 72 hours if oral feeding is not tolerated; and parenteral nutrition is the last option if caloric goals cannot be met enterally** — because PN carries a **significantly higher risk of infection including bacteraemia and infected necrosis**.

Indications

4. **Non-functioning or inaccessible gut:** intestinal obstruction, high-output enterocutaneous fistula, prolonged paralytic ileus, severe short bowel syndrome, mesenteric ischaemia with extensive resection.
5. **Severe malabsorption or intestinal failure** where enteral feeding cannot meet requirements; severe inflammatory bowel disease unresponsive to enteral support; radiation enteritis.
6. **Peri-operative:** severely malnourished patients unable to be fed enterally for more than 7 days; post-operative complications preventing enteral access.
7. **In advanced disease:** Pathy's notes that in motor neuron disease, parenteral nutrition can be useful in advanced disease when gastrostomy is not feasible.
8. **General rule for older adults:** consider PN when the gut cannot be used for more than 5–7 days in a well-nourished patient, or more than 3 days in an already malnourished patient.

Access and Composition

9. **Access:** central venous access is required for full-strength TPN because of high osmolarity — a tunnelled catheter, PICC line or implanted port. **Peripheral parenteral nutrition** is possible only with low-osmolarity solutions and for short periods. Use a **dedicated lumen** with strict aseptic technique.
10. **Energy:** approximately **25–30 kcal/kg/day**, reduced to **20–25 kcal/kg/day in the acutely ill and the elderly** to avoid overfeeding.
11. **Protein:** approximately **1.0–1.5 g/kg/day** of amino acids in older adults; Essentials notes protein requirement estimates range from 0.5 to more than 1.0 g/kg/day, and that **higher protein intake is independently associated with a dose-responsive lower risk of incident frailty** in older women.
12. **Carbohydrate:** dextrose supplying about 50–60% of non-protein calories; do not exceed roughly 4–5 mg/kg/min to avoid hyperglycaemia and hepatic steatosis.
13. **Lipid:** 20–30% of total energy, providing essential fatty acids; modern olive-oil and fish-oil-containing emulsions are preferred to pure soybean-oil emulsions.
14. **Micronutrients:** daily electrolytes, trace elements (zinc, selenium, copper, chromium, manganese) and vitamins including **thiamine** must be added — deficiency states develop rapidly on PN.

Monitoring

15. **Daily initially:** capillary glucose, fluid balance, weight, and serum **potassium, phosphate and magnesium** for the first 3–5 days.
16. **Twice weekly then weekly:** urea, creatinine, sodium, calcium, liver function tests, triglycerides, full blood count and CRP; periodic trace element and vitamin levels in long-term PN.
17. **Clinical monitoring:** catheter site inspection, temperature charting, and daily reassessment of whether the gut can be used — **PN should be stopped as soon as enteral feeding is feasible**.

Complications

18. **Catheter-related mechanical:** pneumothorax, haemothorax, arterial puncture, air embolism, catheter malposition, thrombosis and occlusion.
19. **Catheter-related bloodstream infection** — the most feared complication; higher infection risk than enteral feeding is the principal reason PN use has declined.
20. **Metabolic:** hyperglycaemia (and rebound hypoglycaemia on abrupt cessation), hypertriglyceridaemia, electrolyte

disturbances, azotaemia, hypercapnia from overfeeding, and metabolic bone disease in long-term PN.

21. **Hepatobiliary:** steatosis, cholestasis, gallbladder sludge and gallstones — the last especially relevant in older adults, in whom Pathy's already notes increased biliary cholesterol and reduced gallbladder tone predispose to stone formation.
22. **Gut-related:** intestinal mucosal atrophy and bacterial translocation from disuse of the gut.
23. **Refeeding syndrome** — the most important early hazard in the malnourished older patient. Reintroduction of carbohydrate triggers an insulin surge with intracellular shift of **phosphate, potassium and magnesium**, plus thiamine depletion, causing arrhythmia, cardiac failure, respiratory failure, rhabdomyolysis, seizures, delirium and death.

Refeeding Syndrome — Prevention

24. **Identify risk:** low BMI, significant unintentional weight loss, minimal intake for several days, low baseline potassium, phosphate or magnesium, alcohol misuse, and loss of subcutaneous fat or muscle. **Electrolytes may be normal despite total-body deficiency.**
25. **Prevention protocol:** give **thiamine before any dextrose-containing solution**; start feeding at **100–150 g of dextrose or 10–20 kcal/kg in the first 24 hours**, advancing by about **33% of goal every 1–2 days**; correct severe electrolyte deficits before or alongside feeding; and monitor vital signs 4-hourly for the first 24 hours with daily weights and fluid balance.

Evidence and Ethical Considerations in the Elderly

26. Pathy's is deliberately cautious about outcomes: **the results of percutaneous parenteral feeding in subacute patients were not optimal**, and **undernourished or high-risk surgical patients did not have post-operative complications reduced to the level of well-nourished patients by either enteral or parenteral support** — suggesting factors beyond pure starvation are operating.
27. Enteral tube feeding likewise did **not affect 24-month survival in nursing home residents over 65 with severe cognitive impairment**, and aspiration pneumonia occurred at a similar rate with or without enteral feeding — the same reasoning applies with even greater force to PN in advanced dementia.
28. **Ethical framing:** artificial nutrition is a medical treatment, not basic care. Decisions require goals-of-care discussion, capacity assessment, and consideration of advance directives; in India this is framed by the Mental Healthcare Act 2017 and Supreme Court jurisprudence on advance medical directives.

Latest Updates (2023–Present)

- The **ASPEN consensus definition of refeeding syndrome** remains the reference standard, defining it by a decrease from pre-feeding serum phosphorus, magnesium or potassium of **≥10% (mild), ≥25% (moderate) or ≥50% (severe)**, with thiamine **100 mg before dextrose-based solutions** and no routine value in checking thiamine levels *(Nutr Clin Pract, 2020; validated in JPEN, 2022)*.
- Comparative work in older hospitalised patients has shown that **incidence and mortality of refeeding syndrome differ substantially depending on which of three diagnostic criteria is applied**, so the criterion used must be stated *(Clin Nutr ESPEN, 2024)*.
- **ESPEN's partially revised ICU guideline** reaffirms the hierarchy — oral diet preferred, then enteral, with low-dose PN considered only where enteral feeding is not feasible in high-risk or severely malnourished patients, **balanced against the risks of overfeeding and refeeding**; supplemental PN timing remains debated *(Clin Nutr, 2023)*.
- **ASPEN has issued expert guidance on "When Is Parenteral Nutrition Appropriate?"** together with updated guidelines on **PN ordering, order review, compounding, labelling and dispensing**, and on **selection and care of central venous access devices for adult home parenteral nutrition** *(ASPEN guidelines and standards, current)*.
- Current critical-care practice explicitly advises that **serum protein markers such as prealbumin and CRP are not recommended for evaluating nutritional status or setting nutritional goals**, and that CRP should be measured simultaneously when interpreting vitamin or mineral levels *(ASPEN/SCCM-derived protocols, 2025)*.

Current Practice Update

Textbook position (Pathy's)	Current evidence (2023–2026)
Downward trend in PN use in critical care because of higher morbidity than enteral nutrition.	**Position confirmed and formalised** — ESPEN ICU guidance places oral first, enteral second, and PN only where enteral is not feasible, weighing overfeeding and refeeding risk.
Refeeding described as a risk without formal diagnostic criteria.	**ASPEN consensus criteria** now define and grade it by percentage fall in phosphate, potassium or magnesium, with a defined calorie-advancement protocol and thiamine before dextrose.
Nutritional support judged largely by weight and biochemical response.	**Protein markers are no longer recommended** for assessing nutritional status or goals; assessment is by GLIM criteria and functional/muscle measures.
Little detail on PN safety systems.	ASPEN now publishes **prescribing, compounding, labelling and central venous access standards**, reflecting that most PN harm is preventable system error.

Examiner's Tip — mark-earning lines

- "If the gut works, use it — parenteral nutrition carries a significantly higher infection risk, and its use in critical care has fallen for exactly this reason."
- "Quote the refeeding protocol: thiamine before dextrose; start at 10–20 kcal/kg or 100–150 g dextrose in the first 24 hours; advance by about a third of goal every 1–2 days."
- "Electrolytes may be normal despite total-body depletion — normal values do not exclude refeeding risk."
- "In advanced dementia, artificial nutrition does not improve survival or reduce aspiration — this is a goals-of-care decision, not a nutritional one."

10. Vitamin deficiencies in the elderly

15 MARKS

General Principles

1. Vitamins and minerals are **micronutrients** — required in minute quantities but essential for life, functioning as hormones, antioxidants, enzyme cofactors, and in one-carbon metabolism and DNA synthesis. They cannot be synthesised in adequate quantity and must come from food, fortified food or supplements.
2. **Classification: water-soluble** (B group and vitamin C) and **fat-soluble** (A, D, E, K). Fat-soluble deficiency accompanies fat malabsorption; water-soluble deficiency develops faster because stores are small.
3. Pathy's makes the important point that the **same age-associated changes in food intake that cause protein-calorie inadequacy also cause multiple micronutrient deficiencies**, and that **inadequate intake can impair cellular and physiological function even before a classic deficiency syndrome appears**.
4. **Causes in older adults:** anorexia of ageing, reduced taste and smell, poor dentition, dysphagia, poverty and isolation, chronic disease, malabsorption, alcohol, and **polypharmacy with drug–nutrient interaction**.
5. **Nutrients of public health concern for older adults** (Pathy's): the vitamins **choline, riboflavin, thiamine, A, C, D and E**, and the minerals **calcium, magnesium, potassium and selenium**.
6. Essentials adds a corrective: **there is no general increase in vitamin requirements with age** — requirements for vitamins A, B1, B2, C, B6 and B12 do not rise with age. What changes is intake and absorption, not requirement.

Vitamin B12 (Cobalamin)

7. **Prevalence rises with age, ranging from 5% to 40% depending on the cut-off used.** In one Canadian nursing home study of 412 residents, prevalence was **13.8% on admission with a new incidence of 4% per year**.
8. **Causes: food-bound B12 malabsorption from atrophic gastritis affects 10–30% of older adults**; pernicious anaemia (lack of intrinsic factor) occurs in **1–2%**; drugs — **PPIs, H2 blockers and metformin**; gastric or ileal resection; SIBO; and strict vegetarian or vegan diets, since B12 is found only in animal products.
9. **Features:** macrocytic anaemia (which may be absent), plus **fatigue, weakness, anorexia, weight loss, numbness and tingling, balance problems, depression, confusion, dementia, poor memory and glossitis**. Subacute combined degeneration of the cord is the classic neurological syndrome.
10. **Diagnosis:** there is **no gold standard test**. Diagnosis rests on anaemia plus a low B12; **methylmalonic acid and homocysteine are both raised** and add sensitivity but are not mandatory.
11. **Treatment:** oral or intramuscular; **oral is easier, cheaper and likely as effective at high enough doses**. Recheck haemoglobin and B12 at **3 months**. Because of malabsorption of food-bound B12, those aged 50 and over should meet the RDA mainly from **fortified foods or a supplement**.

Folate (Vitamin B9)

12. **Causes:** poor intake, alcoholism, malabsorption (folate is absorbed in the jejunum by passive transport), atrophic gastritis, smoking, and **drugs — methotrexate, sulfasalazine, trimethoprim and phenytoin**.
13. **Stores are small**, so deficiency develops in weeks to months, unlike B12.
14. **Diagnosis:** low haemoglobin, raised MCV, low serum folate, low reticulocyte count. **Serum folate <2 ng/mL is deficient, 2–4 ng/mL borderline, >4 ng/mL normal.** In folate deficiency **MMA is normal and homocysteine is raised**, in contrast to B12 deficiency where both are raised. **Red cell folate** reflects status over the preceding 120 days.
15. **Treatment: folic acid 1 mg daily** orally. **Always correct B12 first** — giving folate alone in B12 deficiency can precipitate or worsen neurological damage. Correcting B12 may resolve a relative folate deficiency without supplementation.

Vitamin D

16. **Mechanism of deficiency in the elderly:** limited sunlight exposure plus a **fourfold reduction in the skin's capacity to produce vitamin D**, compounded by reduced intestinal absorption with age.
17. **Prevalence:** in the relatively healthy older Europeans of the SENECA study, **40% had serum 25(OH)D below 30 nmol/L** — itself below the proposed adequacy threshold of at least 50 nmol/L.
18. **Recommended intakes after age 50 (Pathy's):** calcium **1000–1200 mg/day** and vitamin D **800 IU (20 µg)/day**; upper limits are 2000 mg for calcium and 4000 IU (100 µg) for vitamin D.
19. **Assessment and treatment:** status is assessed by **serum 25(OH)D**; **at a minimum, concentrations below 25–30 nmol/L (10–12 ng/mL) should be treated**. Essentials adds that nursing home residents require **at least 800 IU daily — double the usual recommendation** — to maintain normal levels.

20. **Benefits:** supplementation reduces bone loss and fracture rate in both older men and women, and more recent trials indicate **improvement in sarcopenia and a decrease in falls**.

Other Vitamins and Key Minerals

21. **Thiamine (B1):** deficiency in alcohol misuse, poor intake, refeeding and diuretic use; causes wet and dry beriberi and **Wernicke's encephalopathy**. Must be given before dextrose in any at-risk patient.
22. **Vitamin B6 (pyridoxine):** dietary deficiency is rare and usually occurs with other water-soluble vitamin deficiencies; causes include alcoholism, malabsorption, dialysis and drugs acting as pyridoxine antagonists (isoniazid). Assessed by **plasma pyridoxal-5-phosphate**.
23. **Riboflavin (B2):** contributes to hyperhomocysteinaemia; not usually high-risk in those consuming adequate dairy.
24. **Hyperhomocysteinaemia:** inadequate **B2, B6, folate or B12** raises homocysteine, an independent cardiovascular risk factor also correlated prospectively with dementia and osteoporotic fracture. **Folate lowers homocysteine most effectively** — but Pathy's is explicit that **homocysteine-lowering B vitamins have been mostly unsuccessful in reducing incidence of cardiovascular disease, osteoporosis or Alzheimer's progression**.
25. **Vitamin C:** readily lost from food during storage and preparation — losses may account for about **50% of the potential dietary content**, and around 30% of subjects in European studies had inadequate intake. **Absorption is not affected by age.** Deficiency causes scurvy — perifollicular haemorrhage, gum bleeding, poor wound healing.
26. **Vitamin K:** deficiency from fat malabsorption, prolonged antibiotics and poor intake; causes prolonged prothrombin time and bruising. Note the interaction with warfarin in both directions.
27. **Vitamin A:** deficiency causes night blindness and xerophthalmia; note that **vitamin A absorption increases with age**, so excess is as relevant a hazard as deficiency in supplemented older adults.
28. **Zinc:** deficiency is seen in older people, **particularly in diabetics**; causes **anorexia through impaired taste and smell**, impaired vision, confusion and restlessness, sometimes diarrhoea, and reduced immunity, collagen synthesis and wound healing. **Cytokines dramatically reduce serum zinc**, so plasma levels are unreliable in illness — leucocyte or hair zinc may be more useful.
29. **Iron:** less prevalent in older than younger people; requirement falls sharply after the menopause. Distinguish iron deficiency from **anaemia of chronic disease** using ferritin and CRP. **When anaemia is diagnosed, chronic blood loss or folate/B12 deficiency must be considered.**
30. **Overall caution from Pathy's:** beyond general health, **the role of each micronutrient in preventing or treating specific chronic disease is unclear from clinical trial data**; given limited efficacy and potential adverse effects, supplements should be chosen to contain **no more than the recommended amounts (about 100% of recommendations)**.

Latest Updates (2023–Present)

- The **Endocrine Society Clinical Practice Guideline on vitamin D (2024)** represents the biggest shift. It **suggests empiric supplementation for the general population aged 75 and over** to lower mortality risk, but **suggests against routine 25(OH)D testing in all populations considered**, including those aged 75 and over *(J Clin Endocrinol Metab, 2024)*.
- The same guideline **advises against empiric supplementation above the DRI in healthy adults under 75**, and for people over 50 with an indication, recommends **daily lower-dose vitamin D rather than intermittent high doses**. Trial doses informing the mortality recommendation ranged from 400 to 3333 IU daily, with a weighted average of about **900 IU daily** *(J Clin Endocrinol Metab, 2024)*.
- Critically, the Endocrine Society **no longer endorses 30 ng/mL as a sufficiency target**, and states no clear evidence defines an optimal 25(OH)D level for disease prevention — a reversal of its own 2011 position that remains contested by other experts *(J Clin Endocrinol Metab 2024; Endocr Pract commentary 2024–2026)*.
- Note the scope limitation for exams: this guideline addresses **disease prevention in generally healthy people without established indications for testing or treatment**. It does **not** apply to patients with malabsorption, CKD, osteoporosis, osteomalacia or drug-induced deficiency, where **testing and treatment remain fully indicated** *(J Clin Endocrinol Metab, 2024)*.
- The **UEG 2025 malabsorption guideline** stresses that **screening for B12 in the elderly is important because some related neurological abnormalities reverse with supplementation**, and identifies chronic PPI use and SIBO as aggravating factors alongside atrophic gastritis *(United European Gastroenterol J, 2025)*.
- The **USPSTF** continues to recommend against vitamin D with or without calcium for primary fracture prevention in community-dwelling postmenopausal women and men aged 60 and over — consistent with the 2024 guideline's restraint *(USPSTF, reaffirmed in JBMR commentary 2025)*.

Current Practice Update

Textbook position (Pathy's / Essentials)	Current evidence (2023–2026)
Assess vitamin D status by 25(OH)D; treat levels below 25–30 nmol/L; adequacy often taken as ≥50 nmol/L.	Endocrine Society 2024 **suggests against routine 25(OH)D testing** in generally healthy people and **no longer endorses a numerical sufficiency target**. Testing remains indicated in malabsorption, CKD, osteoporosis and osteomalacia.
800 IU vitamin D daily recommended after age 50; nursing home residents need at least 800 IU.	**Empiric supplementation now recommended specifically from age 75** to reduce mortality, with **daily low-dose preferred over intermittent high-dose** in those over 50 — supporting the textbook dose but sharpening the age threshold and the regimen.
Homocysteine-lowering B vitamins mostly unsuccessful for CVD, osteoporosis and Alzheimer's.	**Position unchanged** — no new trial has overturned it; treat documented deficiency, not the homocysteine number.
B12 deficiency diagnosed on anaemia plus low B12, with MMA and homocysteine as adjuncts.	**Unchanged**, but UEG 2025 adds a stronger case for **proactive B12 screening in older adults**, given reversibility of neurological features and the contribution of PPIs and SIBO.
Micronutrient supplements should contain no more than about 100% of recommendations.	**Position reinforced**; USPSTF continues to advise against routine vitamin D and calcium for primary fracture prevention in community-dwelling older adults.

Examiner's Tip — mark-earning lines

- "Requirements for most vitamins do not rise with age — what changes is intake and absorption."
- "10–30% of older adults malabsorb food-bound B12; pernicious anaemia accounts for only 1–2%."
- "MMA and homocysteine both raised in B12 deficiency; only homocysteine raised in folate deficiency."
- "Always correct B12 before folate — folate alone can worsen the neurological lesion."
- "Skin capacity to synthesise vitamin D falls fourfold in the elderly; 40% of healthy older Europeans in SENECA had 25(OH)D below 30 nmol/L."
- "Homocysteine-lowering B vitamins have failed to reduce cardiovascular events, fractures or Alzheimer's progression."

Extra Points to Remember — Indian context

- **Vitamin D deficiency is highly prevalent across all Indian age groups** despite abundant sunlight, driven by skin pigmentation, indoor living, covered clothing, air pollution and low dietary intake — so the Endocrine Society's "generally healthy Western population" caveats must be applied with care in Indian practice.
- **B12 deficiency is common in Indian vegetarians** irrespective of malabsorption — a lifelong dietary cause rather than an age-related one; combine dietary counselling with supplementation.
- **ICMR-NIN dietary guidelines** for Indians provide the local RDA framework and should be quoted alongside Western figures.
- **Fortification programmes:** India's fortified staples (double-fortified salt with iron and iodine, fortified oil and milk with vitamins A and D, fortified wheat flour and rice with iron, folic acid and B12) are the population-level answer.
- **Practical Indian prescribing:** cholecalciferol 60,000 IU weekly sachets are widely used for repletion followed by daily maintenance — but note the current preference for **daily lower-dose regimens over intermittent high doses** in those over 50.

Appendix — Rapid Revision Grid

NIGHT-BEFORE REVISION

Numbers Worth Quoting

Topic	Figure
GORD prevalence	6th commonest disorder in residential care; ~20% of general population have weekly reflux; hiatus hernia in ~60% of those over 60
GORD diagnosis	Heartburn/regurgitation ~70% sensitive; double-dose PPI trial 2–8 weeks, sensitivity and specificity up to 80%
Constipation	Faecal impaction in 7% of institutionalised elderly; median 42% prevalence in long-term care; 90% of slow-transit responds to laxatives; biofeedback effective in ~70%
Laxative doses	Psyllium 3–6 g; PEG 17–34 g; lactulose 10–40 g; senna 15 mg; bisacodyl 5–10 mg; sodium picosulfate 5–10 mg; lubiprostone 8–24 µg BD
IBS in elderly	10–15% of people over 70; placebo response in functional GI disorders 20–70%
Achalasia	Perforation risk ~1% for both pneumatic dilatation and myotomy; series of 51 patients aged ≥65 — no deaths, median stay 3 days
Gastroparesis	Up to 80% of diabetic gastroparesis has abnormal small-intestinal motility; scleroderma oesophageal dysmotility ~80%
UGI bleeding	Bleeding contributes to ~5% of elderly deaths; GBS 0–1 for outpatient care; transfuse at Hb 7 g/dL (8 g/dL with CVD), target 7–9 g/dL; erythromycin 250 mg IV 30–120 min pre-scope; endoscopy within 24 h; high-dose PPI ×3 days then BD oral ×2 weeks
Malabsorption	Faecal fat >7 g/day; faecal elastase-1 <200 µg/g suggests, <100 µg/g severe EPI; SIBO antibiotics 1–4 weeks
MNA	Screening max 14 points: 12–14 normal, 8–11 at risk, 0–7 malnourished; sensitivity 96%, specificity 60%; mortality risk 0.35 (0.18–0.66) with normal score
GLIM	Weight loss >5% in 6 months or >10% beyond 6 months; needs 1 phenotypic + 1 etiologic criterion
TPN	25–30 kcal/kg/day (20–25 if acutely ill/elderly); protein 1.0–1.5 g/kg/day; refeeding start 10–20 kcal/kg or 100–150 g dextrose in first 24 h, advance ~33% of goal every 1–2 days; thiamine 100 mg before dextrose
Refeeding severity	Fall from pre-feeding phosphate, magnesium or potassium: ≥10% mild, ≥25% moderate, ≥50% severe (ASPEN)
Vitamin B12	Prevalence 5–40%; 10–30% malabsorb food-bound B12; pernicious anaemia 1–2%; nursing home 13.8% on admission, 4%/year new
Folate	<2 ng/mL deficient, 2–4 borderline, >4 normal; treat with folic acid 1 mg/day; red cell folate reflects 120 days
Vitamin D	Calcium 1000–1200 mg/day and vitamin D 800 IU/day after 50; ULs 2000 mg and 4000 IU; treat 25(OH)D below 25–30 nmol/L; SENECA — 40% below 30 nmol/L; skin synthesis reduced fourfold

Named Guidelines and Evidence by Topic

Topic	Named source
Malabsorption	UEG malabsorption guideline 2025; ACG coeliac guideline update 2023; ESsCD 2025 (no-biopsy pathway restricted to under-45s)
Constipation	Joint AGA–ACG CIC guideline 2023 (first joint guideline; PEG, sodium picosulfate, linaclotide, plecanatide, prucalopride strong); ASCRS 2024
GORD	Lyon Consensus 2.0 (Gut 2024); ACG GERD guideline 2022; AGA Clinical Practice Update on P-CABs 2024; VISION 5-year safety study 2025; Seoul Consensus focused update 2025
Motility	Chicago Classification v4.0; ACG gastroparesis guideline 2022
UGI bleeding	ACG UGIB guideline 2021; ESGE non-variceal 2021 and variceal 2022; ACG LGIB update 2023; restrictive transfusion meta-analysis (Sci Rep 2023)
Nutrition assessment	GLIM consensus 2019 and 5-year update 2025; ESPEN geriatric nutrition and hydration guideline; ESPEN–EASO sarcopenic obesity consensus 2022; EWGSOP2
Parenteral nutrition	ASPEN refeeding consensus 2020; ESPEN ICU guideline revision 2023; ASPEN PN ordering/compounding and CVAD standards
Vitamins	Endocrine Society vitamin D guideline 2024; USPSTF fracture prevention; ICMR-NIN dietary guidelines (India)

Classifications and Criteria to Name

Name	Use
Rome IV	Constipation, IBS, functional dyspepsia
Bristol Stool Form Scale	Stool consistency; target type 4–5 in faecal incontinence
Los Angeles classification A–D	Erosive oesophagitis; only B, C, D conclusive for GORD under Lyon 2.0
Chicago Classification v4.0	Oesophageal manometry; achalasia types I, II, III
Forrest classification	Ulcer stigmata; treat Ia, Ib, IIa endoscopically
Glasgow–Blatchford / Rockall	Pre-endoscopic and post-endoscopic UGIB risk
GLIM criteria	Diagnosis of malnutrition — 1 phenotypic + 1 etiologic
MNA / MNA-SF / SNAQ / MUST / NRS-2002 / SGA	Nutritional screening and assessment tools
ASPEN refeeding criteria	Grading refeeding syndrome by electrolyte fall

Indian Context — One Line per Topic

- **Malabsorption:** tropical sprue, intestinal tuberculosis, giardiasis, strongyloidiasis and HIV enteropathy must appear in every differential.
- **Constipation:** ispaghula (isabgol) is the accessible bulking agent; PEG sachets are affordable and preferable to repeated enemas at home.
- **GORD:** pantoprazole and rabeprazole are the cheapest available PPIs; vonoprazan is marketed but cost-limited.
- **UGI bleeding:** variceal bleeding from cirrhosis, non-cirrhotic portal fibrosis and EHPVO is a major cause; PCC availability is variable outside tertiary centres, so FFP may be the practical alternative.
- **Nutrition:** LASI Wave 1 data document high rates of undernutrition among older Indians; NPHCE provides the service framework.
- **Vitamins:** widespread vitamin D deficiency despite sunlight; B12 deficiency common in lifelong vegetarians; ICMR-NIN RDAs and national food fortification are the population-level responses.

MD GERIATRIC MEDICINE

Haematology in the Elderly — Compendium of High-Yield Answers

Plasma Cell Dyscrasias · Leukaemias · Myelodysplasia · Anaemia · Thrombocytopenia
Lymphomas · Transfusion Medicine · Bone Marrow Transplantation · POEMS Syndrome

Based on Pathy's Principles and Practice of Geriatric Medicine (6th ed.)
and Essentials of Clinical Geriatrics (7th ed.), supplemented with current evidence

CONTENTS

1 Plasma Cell Dyscrasias — Classification

2 Plasma Cell Dyscrasias — Presentation, Diagnostic Criteria, Progression & Treatment

3 Multiple Myeloma — Aetiology, Diagnosis & Management

4 Chronic Myeloid Leukaemia — Clinical Features & Management

5 Myelodysplasia

6 Alkylating Agents · Intramedullary vs Extramedullary Haemolysis · Dysplasia

7 Anaemia of Chronic Disease

8 Anaemia in the Elderly & Evaluation of Anaemia

9 Thrombocytopenia

10 Hodgkin Lymphoma

11 Non-Hodgkin Lymphoma — Presentation, Prognosis & Principles of Therapy

12 Anaemia of Chronic Kidney Disease — Type, Investigation & Treatment

13 Mechanisms of Drug-Induced Thrombocytopenia

14 Blood Transfusion

15 WHO Classification & Diagnostic Criteria of MDS, and its Differentials

16 Bone Marrow Transplant

17 POEMS Syndrome

Reference lists have been omitted from this compilation for concise night-before revision.

QUESTION 1

Name the various plasma cell dyscrasias

> **Textbook gap:** Pathy's has no dedicated chapter on plasma cell dyscrasias. Only incidental mentions exist in Ch. 24 (paraproteins as a cause of acquired coagulation defects — myeloma, MGUS, amyloid; inhibition of fibrin polymerisation; acquired haemophilia/vWD) and in the Anaemia chapter (MGUS grouped under "anaemia of clonal disorders" with CHIP/MDS). The classification below is sourced from WHO-HAEM5 (2022), ICC (2022) and IMWG consensus.

High-Yield Points

Definition

1. **Plasma cell dyscrasias (monoclonal gammopathies)** are disorders in which a single clone of plasma/lymphoplasmacytic cells secretes a **monoclonal immunoglobulin (M-protein)**.
2. They form a **continuous spectrum** from harmless MGUS to plasma cell leukaemia. Every myeloma is preceded by an MGUS phase.
3. **Diseases of old age**: MGUS in ~3% over 50; median myeloma diagnosis age ~69 years.

A. Premalignant precursor states

4. **MGUS** — M-protein <3 g/dL, clonal marrow plasma cells <10%, no CRAB. Progresses ~1%/year.
5. **Three MGUS subtypes:** Non-IgM (→ myeloma), IgM (→ Waldenström's), Light-chain (→ light-chain myeloma/AL amyloidosis).
6. **Smouldering (asymptomatic) myeloma** — M-protein ≥3 g/dL or plasma cells 10–60%, no end-organ damage.

B. Malignant plasma cell neoplasms

7. **Multiple myeloma (symptomatic)** — ≥10% clonal plasma cells or plasmacytoma + a myeloma-defining event (CRAB or SLiM).
8. **Variants:** non-secretory, IgD/IgE myeloma (rare, aggressive), IgM myeloma, light-chain (Bence Jones) myeloma.
9. **Solitary plasmacytoma of bone** — single lytic lesion, normal marrow, no CRAB.
10. **Extramedullary plasmacytoma** — solitary soft-tissue lesion, usually upper aerodigestive tract.
11. **Plasmacytoma with minimal marrow involvement** — <10% clonal marrow plasma cells; higher progression risk.
12. **Plasma cell leukaemia** — primary or secondary; most aggressive form.

C. Immunoglobulin deposition disorders

13. **AL amyloidosis** — misfolded light chains deposit as β-pleated fibrils in heart, kidney, liver, nerve, gut.
14. **Monoclonal Ig deposition disease (MIDD)** — non-fibrillar, Congo-red negative: LCDD, HCDD, light-and-heavy-chain forms.
15. **Monoclonal gammopathy of renal significance (MGRS)** — small non-malignant clone causing kidney damage; needs clone-directed treatment.

D. Heavy chain diseases

16. **Alpha HCD** (IPSID/Mediterranean lymphoma), **Gamma HCD** (Franklin disease), **Mu HCD** (with CLL).

E. Lymphoplasmacytic disorders

17. **Waldenström macroglobulinaemia** — IgM paraprotein, MYD88 L265P; causes hyperviscosity.
18. **Cold agglutinin disease** and **cryoglobulinaemia** (Type I monoclonal; Type II mixed).

F. Paraneoplastic gammopathies

19. **POEMS syndrome** — Polyneuropathy, Organomegaly, Endocrinopathy, Monoclonal gammopathy, Skin changes; lambda light chain, sclerotic lesions, high VEGF.
20. **Newer syndromes:** TEMPI, AESOP, Schnitzler; umbrella term **MGCS (monoclonal gammopathy of clinical significance)** — neurological, cutaneous, haemostatic forms.

Latest Updates (2023–Present)

WHO-HAEM5 (2022) restructured the group into four blocks: Monoclonal gammopathies (adds MGRS, cold agglutinin disease); Ig deposition diseases (renamed AL amyloidosis, MIDD); Heavy chain diseases; Plasma cell

neoplasms (adds TEMPI, AESOP as full entities). **ICC 2022** introduced genetic subtyping of myeloma (CCND-family, MAF-family, NSD2 translocations, hyperdiploidy vs MM-NOS). **Plasma cell leukaemia threshold lowered** to ≥5% circulating plasma cells (from >20%). **MGCS** is now formally accepted, covering MGRS (renal), MGNS (neurological), cutaneous and multisystem forms. A new **IMS–IMWG high-risk myeloma consensus (2025)** replaces R-ISS/R2-ISS. **AQUILA trial (NEJM 2025):** daratumumab in high-risk smouldering myeloma improved 5-yr PFS (63.1% vs 40.8%) and OS (93.0% vs 86.9%) — FDA-approved Nov 2025, the first therapy for a precursor state. The **IMWG Frailty Index** (and possibly the Rockwood Frailty Index) now guides treatment intensity in the elderly.

Current Practice Update

Domain	Pathy's (2022)	Current evidence
Coverage	No dedicated section	Fully classified under WHO-HAEM5/ICC 2022
New entities	Not mentioned	MGRS, cold agglutinin disease, TEMPI, AESOP formally recognised
MGCS concept	Absent	Established — small clones causing organ damage need clone-directed therapy
Plasma cell leukaemia	Not mentioned	Redefined at ≥5% circulating plasma cells
Smouldering myeloma	Not mentioned	Daratumumab approved (2025) for high-risk SMM

QUESTION 2

Plasma Cell Dyscrasias — Clinical Presentation, Diagnostic Criteria, Progression & Treatment

Textbook gap: No dedicated Pathy's content. Relevant fragments: Ch. 24 (paraprotein-related coagulation defects), Anaemia chapter (MGUS as clonal anaemia), Ch. 59 (chronic pain red flags — weight loss, fever, prior malignancy, progressive neurological deficit — directly applicable to myeloma back pain).

High-Yield Points

A. Clinical Presentation

1. MGUS/smouldering myeloma usually found incidentally on routine SPEP or a raised globulin gap/unexplained high ESR.
2. **Symptomatic myeloma = CRAB tetrad** (hyperCalcaemia, Renal impairment, Anaemia, Bone lesions) — present in ~80% at diagnosis.
3. **Bone pain** is commonest — back/rib pain, vertebral fractures, height loss, spinal cord compression (emergency).
4. **Anaemia in ~70%** — normocytic; in the elderly presents as fatigue, falls, delirium, functional decline; film shows rouleaux.
5. **Renal impairment in 20–40%** — mainly light-chain cast nephropathy, worsened by hypercalcaemia, dehydration, NSAIDs, contrast.
6. **Recurrent infection** from immune paresis (suppressed uninvolved immunoglobulins); encapsulated organisms predominate.
7. Syndrome-specific: hyperviscosity (Waldenström's), neuropathy (POEMS/anti-MAG), nephrotic syndrome/macroglossia (AL amyloidosis).
8. In the elderly, presentation is often non-specific — falls, delirium, weight loss, or a persistently raised ESR.

B. Diagnostic Criteria

9. Screening: **SPEP + serum immunofixation + serum free light chain (FLC) assay**, plus 24-hr urine electrophoresis/IFE for Bence Jones protein.
10. **Bone marrow aspirate/biopsy** with flow cytometry (CD38 bright, CD138+) and FISH (del17p, t(4;14), t(14;16), 1q gain).
11. **Imaging:** whole-body low-dose CT, whole-body MRI, or PET-CT. Plain skeletal survey is obsolete.
12. **MGUS** = M-protein <3 g/dL, marrow plasma cells <10%, no end-organ damage.
13. **Smouldering myeloma** = M-protein ≥3 g/dL (or urine ≥500 mg/24h) or plasma cells 10–60%, no MDE.
14. **Multiple myeloma (IMWG)** = ≥10% clonal plasma cells or plasmacytoma + ≥1 myeloma-defining event.
15. **MDE = CRAB + SLiM:** Ca >11 mg/dL; creatinine >2 mg/dL or CrCl <40 mL/min; Hb <10 g/dL; ≥1 lytic lesion; ≥60% plasma cells; FLC ratio ≥100; >1 MRI focal lesion.

C. Progression

17. Stepwise: normal → MGUS → smouldering myeloma → symptomatic myeloma → plasma cell leukaemia/extramedullary disease.
18. Rates: MGUS ~1%/year (lifelong); SMM ~10%/year (first 5 yrs), then ~3%, then ~1%/year.
19. Risk models: **Mayo MGUS model** (M-protein ≥1.5 g/dL, non-IgG, abnormal FLC ratio); **"20-2-20" SMM model** (plasma cells >20%, M-protein >2 g/dL, FLC ratio >20).
20. Staging: ISS → R-ISS → R2-ISS. **IMWG Frailty Index** independently predicts survival/toxicity in the elderly.

Treatment at a Glance

Condition	Standard approach
MGUS (low risk)	Observation; repeat SPEP+FLC at 6 months then 1–2 yearly
Smouldering myeloma (high risk)	Subcutaneous daratumumab (2025) or trial
Myeloma, fit/transplant-eligible	Quadruplet induction → ASCT → lenalidomide maintenance
Myeloma, transplant-ineligible	D-VRd (fit) or DRd/Isa-Rd (frail), until progression
Myeloma, frail/very old	Dose-attenuated doublet/triplet; treat to tolerability

Relapsed/refractory	BCMA CAR-T (cilta-cel, ide-cel) or bispecifics (teclistamab, elranatamab)
Supportive care	Zoledronic acid/denosumab, hydration, avoid NSAIDs/contrast, VTE prophylaxis with IMiDs
AL amyloidosis	Dara-VCd (first-line)
Waldenström's	Zanubrutinib or bendamustine-rituximab; plasmapheresis for hyperviscosity

Latest Updates (2023–Present)

CEPHEUS (Nat Med 2025): D-VRd gave MRD-negativity 60.9% vs 39.5%, 43% lower progression/death risk in transplant-ineligible myeloma. **IMROZ (NEJM 2024):** Isa-VRd gave 5-yr PFS 63% vs 45%. Both capped age at 80 and excluded frail patients — DRd/Isa-Rd remain preferred for the truly frail. **AQUILA (NEJM 2025):** daratumumab in high-risk SMM, FDA-approved Nov 2025 — shift to early interception. **IMS–IMWG 2025** redefines high-risk myeloma (~20–25% of patients): del17p/TP53, t(4;14)/t(14;16) with 1q gain, high β2-microglobulin with normal creatinine. **FDA ODAC (April 2024)** voted MRD-negative CR an acceptable endpoint for accelerated approval; **MIDAS trial (NEJM 2025)** tested whether MRD-negative patients can skip transplant. CAR-T/bispecifics now give 60–97% response in triple-class-exposed disease; bispecifics often preferred in frail elderly (off-the-shelf, no apheresis). AL amyloidosis: anti-fibril antibodies **anselamimab** and **birtamimab** in phase III. Waldenström's: **zanubrutinib** preferred over ibrutinib (ASPEN — far less AF).

Current Practice Update

Domain	Pathy's (2022)	Current evidence
Diagnostic criteria	Absent	CRAB + SLiM biomarkers (IMWG)
Imaging	Not addressed	Skeletal survey/bone scan obsolete; WBLDCT/WB-MRI/PET-CT standard
First-line therapy	Not addressed	Quadruplets (D-VRd/Isa-VRd) fit; DRd/Isa-Rd frail
Precursor treatment	Not addressed	Daratumumab approved for high-risk SMM (2025)
Relapsed disease	Not addressed	CAR-T and bispecific antibodies

QUESTION 3

Multiple Myeloma — Aetiology, Diagnosis & Management

Textbook gap: No myeloma chapter in Pathy's. Transferable content: Ch. 24 (paraprotein coagulopathy), Anaemia chapter (clonal anaemia), Ch. 59 (spinal red flags), and — most valuably — Ch. 26/27's geriatric-oncology philosophy (CGA is standard of care; treatment must not be chosen on chronological age alone; frailty/comorbidity assessment via gait speed, SPPB, Charlson index, CRASH score, Hurria/CARG tool).

High-Yield Points

A. Definition & Epidemiology

1. **Malignant clonal plasma cell proliferation** producing M-protein and end-organ damage.
2. 10–15% of haematological malignancies; median diagnosis age 65–70; >30% of patients >75 years.

B. Aetiology & Risk Factors

3. Cause unknown; arises from a post-germinal-centre plasma cell; every case preceded by MGUS.
4. **Host factors:** age (dominant), male sex, African ancestry (~2× risk, 5–10 yrs earlier), family history (2–3×), obesity/T2DM.
5. **Environmental:** ionising radiation, benzene, pesticides/herbicides/dioxins, agriculture/woodworking/paper industry.
6. **Predisposing states:** MGUS (~1%/yr progression), smouldering myeloma (~10%/yr), solitary plasmacytoma.

C. Pathogenesis

7. **Primary genetic events** (mutually exclusive): hyperdiploidy (55–60%, better prognosis) vs IgH translocations — t(11;14)→CCND1, t(4;14)→NSD2/FGFR3, t(14;16)→MAF.
8. **Secondary events** drive progression: MYC rearrangement, RAS mutations, del(17p)/TP53 loss, 1q21 gain.
9. **Marrow microenvironment:** IL-6, IGF-1, VEGF from stroma; bone disease from ↑RANKL/MIP-1α + ↓osteoprotegerin (osteoclast activation) and DKK1/IL-3 suppressing osteoblasts — purely lytic lesions, bone scan is falsely negative.

D. Diagnosis

10. SPEP + immunofixation + serum FLC + 24-hr urine IFE.
11. Marrow biopsy with flow cytometry and FISH (del17p, t(4;14), t(14;16), t(14;20), 1q gain).
12. Imaging: WBLDCT/WB-MRI/PET-CT (skeletal survey/bone scan obsolete).
13. **IMWG criteria:** ≥10% clonal plasma cells or plasmacytoma + ≥1 myeloma-defining event (CRAB + SLiM).

E. Staging & Management Principles

14. ISS → R-ISS → R2-ISS; **IMWG Frailty Index** independently predicts survival/toxicity.
15. Four rules: treat only symptomatic disease; decide transplant eligibility by fitness not age; CGA/frailty assessment before intensity; supportive care equally important.

Management at a Glance

Setting	Approach
Transplant-eligible	Quadruplet induction (D-VRd/Isa-VRd) → ASCT (melphalan 200 mg/m²) → lenalidomide ± daratumumab maintenance
Transplant-ineligible/older	D-VRd (fit, ≤80) or DRd/Isa-Rd (frail), until progression
Frail/very elderly	Dose-attenuated; SC bortezomib weekly; reduce dexamethasone to 20 mg weekly
Relapsed	Carfilzomib, pomalidomide, isatuximab, elotuzumab, or cilta-cel CAR-T
Triple-class-exposed	BCMA CAR-T or bispecifics (teclistamab, elranatamab, talquetamab)
Bone disease	Zoledronic acid 4mg IV monthly (avoid if eGFR<30) or denosumab 120mg SC monthly
Renal impairment	Hydration ~3L/day; stop NSAIDs/ACEi/contrast; urgent bortezomib-based therapy
Cord compression	Emergency — urgent MRI + high-dose dexamethasone + radiotherapy/surgery

Latest Updates (2023–Present)

PERSEUS (NEJM 2024): D-VRd + Dara-R maintenance gave 4-yr PFS 84.3% vs 67.7% (VRd) — new standard for transplant-eligible. **CEPHEUS/IMROZ:** quadruplets now standard for transplant-ineligible too (age capped at 80, excludes frail). **AURIGA:** Dara-R maintenance boosts MRD-negative conversion in post-transplant MRD-positive patients. **IMS–IMWG 2025** redefines high-risk myeloma. **FDA ODAC (2024)** accepts MRD-negative CR as regulatory endpoint; **MIDAS (2025)** tests transplant omission if MRD-negative. CAR-T/bispecifics transform relapsed disease — bispecifics favoured in frail elderly. **AQUILA (2025):** daratumumab approved for high-risk SMM. Frailty subgroup analyses of MAIA/IMROZ confirm anti-CD38 benefit persists in frail patients but quadruplets tolerated poorly.

Current Practice Update

Domain	Pathy's (2022)	Current evidence
Aetiology/diagnosis	No content at all	Fully defined by IMWG/WHO-HAEM5
First-line therapy	Not addressed	Quadruplets (D-VRd/Isa-VRd) fit; DRd/Isa-Rd frail
Transplant	Not myeloma-specific; RIC principle for MDS/AML transfers	ASCT melphalan 200mg/m², increasingly deferred if MRD-negative
Role of CGA	Strongly endorsed for haematological malignancy	Formalised as IMWG Frailty Index with frailty-adapted dosing

QUESTION 4

Chronic Myeloid Leukaemia — Clinical Features & Management

Textbook gap: Pathy's mentions CML only once — Ch. 24, Table 24.2 — as a myeloproliferative disorder causing acquired platelet dysfunction (bleeding paradoxically more common when platelets >10,00,000/µL). Everything else is externally sourced (ELN 2025, WHO-HAEM5).

High-Yield Points

A. Definition & Epidemiology

1. Myeloproliferative neoplasm defined by **Philadelphia chromosome t(9;22)** → **BCR::ABL1** fusion, a constitutively active tyrosine kinase.
2. ~15% of adult leukaemias. Indian series show younger median age (30s–40s) with bulkier/more advanced disease at diagnosis.
3. TKIs reduced annual mortality from 10–20% to ~1%; 10-yr survival now 82–87%; prevalence rising steeply.

B. Clinical Features

4. Nearly half asymptomatic — found on routine leucocytosis.
5. Constitutional symptoms: fatigue, weight loss, low-grade fever, night sweats.
6. **Splenomegaly** is the most consistent sign, may be massive; splenic infarction causes sudden pleuritic pain with rub.
7. Significant lymphadenopathy is rare in chronic phase — suggests blast phase if present.
8. **Leukostasis** at WCC >1,00,000/µL — priapism, blurred vision, tinnitus, confusion, stroke (emergency).
9. Hyperuricaemia/gout; characteristically **high serum B12**.
10. Blood picture: leucocytosis with full left shift, "myelocyte bulge", **basophilia** (near-universal clue), eosinophilia, thrombocytosis, low LAP score.

C. Phases, Diagnosis & Management

11. **WHO 2022 abolished "accelerated phase"**; blast phase = ≥20% blasts (older ELN definition was >30%).
12. Diagnosis: marrow morphology + cytogenetics + RT-PCR (transcript type e13a2/e14a2) for additional chromosomal abnormalities.
13. **ELTS score** replaces Sokal/Euro/EUTOS for prognosis.
14. Initial management: hydroxyurea, hydration, allopurinol, then start TKI once diagnosis confirmed.
15. **Six first-line TKIs:** imatinib, dasatinib, nilotinib, bosutinib, asciminib (2024), radotinib. Generic imatinib remains most cost-effective in India.
16. **Monitoring:** RT-qPCR (International Scale) 3-monthly. Milestones: BCR::ABL1(IS) ≤10% at 3mo, ≤1% at 6mo, ≤0.1%(MMR) at 12mo.

Response Milestones

Response	Definition
Complete cytogenetic (CCyR)	No Ph+ metaphases ≈ BCR::ABL1(IS) ≤1%
Major molecular (MMR/MR3)	BCR::ABL1(IS) ≤0.1%
MR4 / MR4.5	≤0.01% / ≤0.0032%

Treatment Algorithm

Situation	Approach
Toxicity in responder	Reduce dose rather than switch drugs
Resistance	Check adherence/interactions first, then BCR::ABL1 kinase mutation analysis
Second line	After imatinib → any 2GTKI. After 2GTKI → ponatinib or asciminib
T315I mutation	Ponatinib, high-dose asciminib, or olverembatinib
Blast phase	Intensive chemo + potent TKI → allogeneic SCT

Allo-SCT	Resistance to ponatinib/asciminib; RIC and haploidentical donors widen access in elderly

Latest Updates (2023–Present)

WHO-HAEM5 abolished accelerated phase; blast phase = ≥20%. **Asciminib** (STAMP inhibitor, binds myristoyl pocket) — **ASC4FIRST (NEJM 2024)**: MMR at 48wk 67.7% vs 49% with investigator-chosen TKI; FDA-approved first-line 2024. **ELN 2025** renamed milestones "favourable/warning/unfavourable" (no switch on a single PCR result). Older/comorbid patients: "failed" milestones still had ~80% 10-yr survival (German CML-IV, MDACC) — more conservative approach supported, but not based on age alone. **Dose reduction preferred over switching** for toxicity — low-dose dasatinib 50mg or 20mg (DAVLEC, age ≥70) reduces effusions. **EURO-SKI final analysis (JCO 2024):** MMR maintained in 46% at 12 months after stopping TKI (TFR). **Olverembatinib** effective after ponatinib/asciminib failure. EBMT: transplant outcome determined by disease stage/Karnofsky score, not number of prior TKI lines — haploidentical donors and RIC have widened access in the elderly.

Current Practice Update

Domain	Pathy's (2022)	Current evidence
Coverage	One line — platelet dysfunction only	Comprehensive ELN 2025/WHO-HAEM5
Disease phases	Not addressed	Biphasic; blast phase ≥20%; accelerated phase abolished
First-line therapy	Not addressed	Six TKIs; asciminib added first-line 2024
Toxicity management	Not addressed	Dose reduction preferred over switching
Older patients	Endorses CGA generally	ELN 2025 advises individualised, often conservative management

QUESTION 5

Myelodysplasia

Well covered: Pathy's Ch. 26 ("Myelodysplasia") and Ch. 27 ("Management of MDS and AML in the Elderly") cover this topic in genuine depth — this answer is largely textbook-derived.

High-Yield Points

A. Definition & Epidemiology

1. Heterogeneous **clonal haematopoietic stem cell disorders** — cytopenia from dysplastic, ineffective production, with variable risk of AML transformation.
2. **Most common haematological neoplasm in the elderly** — median diagnosis age 75–76; 86% occur ≥60 years. Incidence 75/1,00,000 over 65; 70/1,00,000/yr over 85.
3. Age is the primary risk factor — accumulating stem cell DNA mutations.

B. Aetiology

4. **Secondary (therapy-related) MDS: 10–20%**, 5–10 years after alkylating agents/radiation/benzene. Alkylator exposure → loss of chromosome 5/7.
5. Inherited predisposition: Down syndrome, Fanconi anaemia, Bloom syndrome, ataxia-telangiectasia, NF1.

C. Pathophysiology

6. Clonal myeloid precursor abnormality → excess intramedullary apoptosis → peripheral cytopenias despite a **hypercellular marrow** (the hallmark paradox).
7. **Aberrant hypermethylation** silences tumour suppressor genes (TET2, DNMT3A, EZH2, ASXL1 mutations) — basis for hypomethylating agent therapy.
8. **SF3B1** mutation → ring sideroblasts, favourable prognosis. **TP53** (5–20%) → higher-risk, therapy-related disease.

D. Clinical Features

9. Non-specific; macrocytic anaemia predominates — dyspnoea, fatigue, angina.
10. **Infection is a primary cause of death** — neutrophil dysfunction (impaired chemotaxis/killing), T-cell function intact so viral/mycobacterial infection uncommon.
11. Bleeding may occur despite normal platelet count (dysfunctional platelets). **Splenomegaly unusual — suggests CMML.**
12. **"MDS may easily be overlooked in the elderly"** — presents simply as chronic macrocytic anaemia; tendency to "leave well enough alone".

E. Diagnosis

13. Blood: macrocytic anaemia (90%), thrombocytopenia (10%), neutropenia (20–30%). Film: hypogranular/hypolobated neutrophils, **pseudo-Pelger-Huët cells** ("pince-nez").
14. Marrow: dysplastic lineage = >10% of cells abnormal. **Perls' stain** for ring sideroblasts (≥5 iron granules around ≥1/3 nucleus in >15% erythroblasts).
15. Cytogenetics essential — 60% normal karyotype; trisomy 8, del(5q/7q/11q/13q/20q); complex karyotype (≥3) = poor prognosis.
16. Exclude reactive causes: B12/folate deficiency, hypothyroidism, EBV/HIV, alcohol, chemotherapy, benzene.

IWG/NCCN Diagnostic Criteria

Two prerequisites: (1) stable cytopenia ≥6 months (or ≥2 months with specific karyotype/bilineage dysplasia); (2) exclusion of other causes.
Plus ≥1 of three: dysplasia ≥10% of a lineage; blast count 5–19%; MDS-associated karyotype (del5q, del20q, +8, −7/del7q).

WHO 2016 Classification (Pathy's)

Subtype	Key features
MDS-SLD	1 dysplastic lineage, 1–2 cytopenias, RS <15%, blasts <5%

MDS-RS	RS ≥15% (or ≥5% if SF3B1+)
MDS-MLD	2–3 dysplastic lineages
MDS-EB-1 / EB-2	Blasts 5–9% / 10–19% or Auer rods
MDS with isolated del(5q)	Normal/high platelets, hypolobated megakaryocytes
MDS-U	1% PB blasts on ≥2 occasions, or defined by cytogenetics alone

Prognostic Scoring

IPSS: Low/Int-1/Int-2/High by blast%, cytopenias, cytogenetics. **IPSS-R** uses 5 cytogenetic groups + blasts/Hb/platelets/neutrophils. CGA and comorbidity indices (MDS-CI, ACE-27, Charlson) add prognostic value in the elderly.

Management

Risk	Approach
Lower-risk	Watchful waiting; ESAs first-line (epoetin alfa preferred); G-CSF synergy; transfusion + iron chelation (start after 20–50 units or ferritin >1000–2500 ng/mL); lenalidomide for del(5q); luspatercept post-ESA failure
Higher-risk	Azacitidine 75 mg/m² SC × 7 days/28-day cycle, ≥4–6 cycles before failure; continue indefinitely if responding
Curative	Allogeneic HSCT — the only cure; RIC feasible to 70–75; CGA crucial for selection
Very frail	Best supportive care

Latest Updates (2023–Present)

WHO 2022/ICC 2022 renamed "myelodysplastic neoplasms" — split into genetically defined (**MDS-5q, MDS-SF3B1** [RS ≥15% criterion removed], **MDS-biTP53** [new]) and morphologically defined (MDS-LB, **MDS-h** [hypoplastic, new formal entity], MDS-IB1/IB2, MDS-f). ICC creates separate "MDS/AML" for 10–19% blasts. **IPSS-M** (31-gene molecular score, 6 categories) supersedes IPSS-R. **COMMANDS (Lancet Haematol 2024):** luspatercept superior to epoetin alfa as first-line in ESA-naïve lower-risk MDS — FDA-approved first-line, though benefit limited in RS-negative patients. **Imetelstat** (telomerase inhibitor, FDA June 2024) — 8-week transfusion independence ~40% vs 15% placebo after ESA failure. **VERONA trial (2025):** azacitidine + venetoclax showed NO survival benefit in higher-risk MDS — single-agent azacitidine remains standard.

Current Practice Update

Domain	Pathy's (WHO 2016)	WHO-HAEM5 (2022)
Nomenclature	Myelodysplastic syndromes	Myelodysplastic neoplasms
Ring sideroblasts	≥15% RS defines MDS-RS	SF3B1 mutation alone defines the entity
TP53	Poor-prognosis mutation, no category	MDS-biTP53 — distinct entity
Prognostic score	IPSS/IPSS-R	IPSS-M is the new reference standard
Post-ESA anaemia	Lenalidomide (del5q), luspatercept (RS/SF3B1)	Luspatercept now first-line; imetelstat added (2024)
Higher-risk MDS	Single-agent azacitidine	Unchanged — VERONA confirms venetoclax adds no benefit

QUESTION 6

Alkylating Agents · Intramedullary vs Extramedullary Haemolysis · What is Dysplasia

Part A — Alkylating Agents

1. **Alkylating agents** attach an alkyl group directly to DNA (usually N7 of guanine), causing **intra- and inter-strand cross-linking** that blocks replication/transcription → apoptosis.
2. **Cell-cycle non-specific** — kill dividing and resting cells; hence toxic to normal stem cells and a classic cause of secondary MDS/AML.

Class	Drugs
Nitrogen mustards	Cyclophosphamide, ifosfamide, chlorambucil, melphalan, bendamustine
Nitrosoureas	Carmustine, lomustine (cross blood-brain barrier)
Alkyl sulfonates	Busulfan
Triazenes	Dacarbazine, temozolomide, procarbazine
Platinum compounds	Cisplatin, carboplatin, oxaliplatin — **NOT true alkylators**, correctly called "platinating agents"

Toxicities: myelosuppression, alopecia, infertility (class-wide); cyclophosphamide/ifosfamide → haemorrhagic cystitis (prevent with mesna); busulfan/carmustine → pulmonary fibrosis; cisplatin → nephro/ototoxicity.

Alkylators vs Topoisomerase II Inhibitors — Therapy-Related Neoplasms

Feature	Alkylating agents/radiation	Topoisomerase II inhibitors
Latency	5–10 years	1–3 years
MDS prodrome	Yes	No — direct AML
Cytogenetics	Unbalanced losses: −5/del5q, −7/del7q	Balanced translocations: 11q23, 21q22

Part B — Intramedullary vs Extramedullary Haemolysis

Intramedullary = precursor dies inside the marrow before reaching blood = **ineffective erythropoiesis** (Pathy's: "dyserythropoiesis with intramedullary abortion"). **Extramedullary** = mature red cell dies after entering circulation (extravascular in spleen/liver, or intravascular in vessels).

The single best discriminator: reticulocyte count. True (extramedullary) haemolysis → HIGH reticulocytes. Intramedullary haemolysis → LOW/inappropriately normal reticulocytes, despite raised LDH, raised indirect bilirubin, low haptoglobin.

Feature	Intramedullary	Extravascular	Intravascular
Site	Inside bone marrow	Spleen/liver macrophages	Within blood vessels
Reticulocytes	**LOW**	HIGH	HIGH
Haptoglobin	Low	Low	**Absent**
Free plasma Hb	Absent	Absent	**Present**
Haemoglobinuria	Absent	Absent	**Present**
Film	Dysplastic cells	Spherocytes	Schistocytes
Examples	MDS, B12/folate deficiency, thalassaemia	AIHA, hereditary spherocytosis	PNH, MAHA, prosthetic valve

Bedside rule: "Haemolysis picture + LOW reticulocytes = the problem is INSIDE the marrow."

Part C — What is Dysplasia

1. **Dysplasia** = disordered growth/maturation of cells within a tissue — abnormal size/shape/arrangement, **NO invasion**, potentially reversible.
2. Distinguish: Hyperplasia (↑number) / Hypertrophy (↑size) / Metaplasia (mature→mature type change) / Dysplasia (disordered maturation) / Anaplasia (loss of differentiation, malignant).
3. **Pathy's definition:** "A lineage is dysplastic when qualitative cytological abnormalities are observed on >10% of the cells of the lineage."

Lineage	Dysplastic features
Dyserythropoiesis	Megaloblastoid change, nuclear budding, internuclear bridging, ring sideroblasts
Dysgranulopoiesis	Hypogranular neutrophils, pseudo-Pelger-Huët cells, Auer rods (if excess blasts)
Dysmegakaryopoiesis	Micromegakaryocytes, hypolobated nuclei

Critical caveat: Pathy's states dysplasia alone is NOT sufficient for MDS diagnosis — must exclude reactive causes: B12/folate deficiency, hypothyroidism, copper deficiency, EBV/HIV, alcohol, chemotherapy, benzene, certain antibiotics.

Latest Updates (2023–Present)

WHO-HAEM5 replaced "therapy-related myeloid neoplasm" with **"myeloid neoplasm post cytotoxic therapy (MN-pCT)"**, now including PARP inhibitors, excluding methotrexate. Modern understanding: alkylators/radiation mostly **select pre-existing clonal haematopoiesis** (TP53/PPM1D-mutant clones resistant to DNA damage) rather than creating new mutations — TP53 mutations found in up to 40% of MN-pCT. Morphological dysplasia has poor inter-observer reproducibility; WHO 2022/ICC 2022 have moved toward **genetic definition** of subtypes (MDS-SF3B1 by mutation, not RS%; MDS-biTP53, MDS-5q by genetics).

QUESTION 7

Anaemia of Chronic Disease

Well covered: Pathy's Ch. 22, under "Inflammatory Anaemias", with Figures 22.3 and 22.5.

High-Yield Points

A. Definition & Epidemiology

1. Anaemia from inflammation with excess cytokine production — a **functional iron deficiency** (stores normal/high, but unavailable for erythropoiesis).
2. **Commonest anaemia in older adults** — up to 70% of hospitalised elderly. Also common in obesity.

B. Causes

3. Chronic infection (TB, endocarditis), inflammatory disease (RA, SLE, IBD), malignancy, chronic organ disease (CKD, HF, COPD, liver disease), obesity, "inflammaging".

C. Pathophysiology — Four Mechanisms

4. **Cytokines** (IL-6, TNF) are the trigger.
5. **Functional iron deficiency via HEPCIDIN** — the central mechanism. Hepcidin (liver peptide, acute-phase reactant) is over-produced by IL-6; it blocks **ferroportin** on enterocytes/macrophages/hepatocytes, blocking gut iron absorption and trapping iron in macrophage/ferritin stores.
6. **Decreased EPO production/response** — cytokines damage EPO-producing renal cells and down-regulate EPO receptors.
7. **Shortened RBC lifespan and suppressed erythropoiesis** — cytokine-mediated apoptosis of precursors and mature cells.

D. Diagnosis

8. **Normocytic, normochromic** anaemia.
9. Iron pattern: low-normal iron/transferrin/saturation, but **normal-to-ELEVATED ferritin**.
10. Ferritin <30 ng/mL → IDA; >100 ng/mL → probable ACD; 30–100 → equivocal, use **soluble transferrin receptor (sTfR)** — HIGH in IDA, NORMAL in ACD, unaffected by inflammation.
11. Reticulocytes LOW; CRP/ESR elevated.

E. Management

12. Treat the underlying disease — cornerstone but often difficult in frail elderly.
13. **Iron NOT recommended unless true concomitant deficiency** — avoid if ferritin >100 ng/mL (futile; iron just locked in RE system).
14. **ESAs** — FDA-approved only for cancer chemotherapy or HIV myelosuppression; check iron first (ESAs often precipitate absolute iron deficiency); target Hb 11–12 g/dL; monitor 2–4 weekly.

IDA vs ACD vs CKD Anaemia

Parameter	IDA	ACD	CKD anaemia
Ferritin	Low (<30)	Normal-high (>100)	Normal-high
TIBC	High	Low-normal	Normal-high
sTfR	HIGH	Normal	Normal
MCV	Microcytic	Normocytic	Normocytic

Latest Updates (2023–Present)

Term **"anaemia of inflammation"** now preferred over "anaemia of chronic disease". **WHO 2024** reaffirmed Hb cutoffs (<13 g/dL men, <12 g/dL women); haemoglobin should **NOT be adjusted for inflammation** (adjust only for altitude/smoking). **HIF-prolyl hydroxylase inhibitors** (roxadustat, daprodustat, vadadustat, desidustat) raise endogenous EPO AND suppress hepcidin — work despite inflammation, unlike ESAs; approved for CKD anaemia (daprodustat FDA 2023). Treatment framing has shifted from "downstream replacement" (iron, ESAs) to "upstream pathway-level" approaches (HIF-PHIs, anti-hepcidin antibodies).

Current Practice Update

Domain	Pathy's (2022)	Current evidence
Terminology	Anaemia of chronic disease	"Anaemia of inflammation" preferred
Pathophysiology	Four mechanisms — hepcidin central	Unchanged, now validated as therapeutic target
New drug class	Not mentioned	HIF-PHIs — raise EPO and lower hepcidin simultaneously

QUESTION 8

Anaemia in the Elderly & Evaluation of Anaemia

Well covered: Pathy's Ch. 22 (Figures 22.1, 22.3, 22.4) — largely textbook-derived.

High-Yield Points

1. **Definition:** Hb <13 g/dL men, <12 g/dL women — but also track the **trend** from the patient's own baseline.
2. **Anaemia is NOT normal ageing** — signifies disease; associated with mortality, disability, hospitalisation.
3. Prevalence: 10–25% over 65, up to 50% over 80 and in nursing homes.
4. **Causes (Bach et al., quoted in Pathy's):** ACD 62.1%, CKD anaemia 11.3–45.1%, multifactorial 28.1%, iron deficiency 14.4%, clonal disorders 10%, folate 6.7%, B12 2%.
5. Associated with impaired ADLs, frailty, cognitive decline, depression, fractures; higher RDW independently predicts mortality.
6. **MCV unreliable in the elderly** — B12 deficiency may be normocytic; concurrent diseases can cancel each other's MCV effect.
7. Presentation often a **geriatric syndrome** — falls, delirium, syncope — not a haematological complaint.

Stepwise Evaluation

Step	Action
1	History: bleeding, diet, alcohol, drugs (PPIs/H2 blockers, metformin, NSAIDs)
2	Examination: pallor, jaundice, glossitis, koilonychia, splenomegaly, neuro exam for SACD
3	CBC + indices + reticulocyte count + **blood film** (single most informative test)
4	Reticulocytes LOW → hypoproliferative; HIGH → haemolysis/blood loss
5	Iron studies: ferritin <30 → IDA; >100 → ACD; 30–100 → add sTfR
6	B12/folate: B12 <200 pg/mL deficient; 200–350 equivocal → add MMA + homocysteine
7	Renal/liver/thyroid function, CRP/ESR
8	**GI evaluation mandatory** for IDA in an older adult — endoscopy top and bottom
9	Marrow biopsy if macrocytic + other cytopenias + low reticulocytes (clonal disorder red flags)

Treatment by Cause

Cause	Treatment
Iron deficiency	Oral iron once daily; IV if malabsorptive/intolerant; continue 3–6 months after correction
B12 deficiency	Oral 2000 mcg daily as effective as IM; IM 1000 mcg weekly then monthly if severe
Folate deficiency	Folic acid 1 mg daily
ACD	Treat underlying disease; iron only if concomitant deficiency

Latest Updates (2023–Present)

WHO 2024 reaffirmed Hb cutoffs; adjust for altitude/smoking, NOT inflammation or ethnicity; no separate elderly-specific cutoff yet. **Oral iron dosing changed:** alternate-day, single-morning dosing (60–120mg elemental iron) increases fractional absorption ~33% with fewer GI side effects — now recommended by ACG and EHA (2024). **AABB 2023:** restrictive transfusion threshold Hb<7 g/dL generally (<7.5 cardiac surgery, <8 orthopaedic/CVD) — but **MINT trial (2023)** in acute MI showed higher 6-month cardiac mortality with restrictive strategy; 2025 AABB now suggests **liberal threshold <10 g/dL in acute MI**. "Unexplained anaemia of the elderly" increasingly reclassified as **CCUS/clonal haematopoiesis** on molecular testing.

Current Practice Update

Domain	Pathy's (2022)	Current evidence
Oral iron dosing	Once daily only, never divided	Alternate-day single morning dose now preferred

Transfusion	Not discussed in detail	Restrictive generally; LIBERAL (<10g/dL) in acute MI
Unexplained anaemia	Recognised as a category	Increasingly reclassified as CCUS/clonal haematopoiesis

QUESTION 9

Thrombocytopenia

Well covered: Pathy's Ch. 24 ("Disorders of Platelet Number"), supplemented by Ch. 23 (DIC).

High-Yield Points

1. **Definition:** platelet count <1,50,000/µL. **Platelet count does NOT change with normal ageing** — a low count always signifies disease.
2. Haemostasis normal above 80,000/µL. **Spontaneous haemorrhage (incl. ICH) doesn't occur until <20,000/µL.**
3. **Three mechanisms:** decreased production, increased destruction (consumption/immune), splenic pooling. **Always consider drugs as a cause.**

Immune Thrombocytopenia (ITP)

4. Adult ITP: insidious onset, chronic course (unlike childhood ITP which is acute, post-viral, self-limiting).
5. Autoantibody against GPIb/GPIIb-IIIa; platelet survival falls from 7–10 days to hours.
6. **Always repeat count + check film first** to exclude pseudothrombocytopenia (EDTA-clumping/clotted sample).
7. Treatment ladder: prednisolone 1mg/kg or IVIg 1g/kg×2 days (first line) → TPO-RAs/romiplostim/eltrombopag (second line) → splenectomy (third line, needs pre-op vaccination) → azathioprine/cyclophosphamide (last resort).

Drug-Induced & Other Causes

8. Two mechanisms: direct marrow suppression, or immune-mediated. Common culprits: aspirin, paracetamol, antibiotics, anticonvulsants, diuretics, quinine, quinidine.
9. **HIT is the exception — causes THROMBOSIS not bleeding.** Mortality ~30% if untreated. Treatment: stop heparin, start argatroban (acute) then fondaparinux. **Warfarin worsens acute HIT.**

DIC vs TTP

Test	DIC	TTP
Coagulation screen	**Grossly abnormal**	Normal/mild
Fibrinogen	Low (<100 mg/dL)	Normal
D-dimer	>1000 ng/mL	Normal/mild ↑
Key feature	Multi-organ failure from microthrombosis	Pentad: fever, thrombocytopenia, neuro, renal, MAHA
Treatment	Treat cause + product support	Plasma exchange + steroids/rituximab

Two Geriatric Pearls

10. In MDS, platelet dysfunction coexists with thrombocytopenia — bleeding/infection are commonest causes of death.
11. In myeloproliferative disorders, bleeding is **paradoxically MORE common at very high platelet counts** (>10,00,000/µL).

Latest Updates (2023–Present)

Rilzabrutinib (oral BTK inhibitor) — FDA-approved Aug 2025 for persistent/chronic ITP; dual mechanism (blocks autoantibody production AND FcγR-mediated platelet phagocytosis) — **LUNA3 trial**. **Splenectomy moved far down the ladder** — ASH now recommends TPO-RAs/rituximab first; delay splenectomy ≥12 months. ITP renamed "immune thrombocytopenia", threshold now <1,00,000/µL. **TTP:** ISTH 2025 update — recombinant ADAMTS13 (apadamtase alfa) now preferred over FFP in congenital TTP; caplacizumab confirmed standard in immune TTP alongside plasma exchange. **HIT** now part of a family of "anti-PF4 disorders" (autoimmune HIT, spontaneous HIT, VITT); **DOACs now accepted alternatives** to argatroban/fondaparinux in stable HIT.

Current Practice Update

Domain	Pathy's (2022)	Current evidence
ITP later	Splenectomy, then	Splenectomy deferred ≥12 months; rilzabrutinib (2025) major new

lines	azathioprine/cyclophosphamide	option
HIT	Classic heparin-triggered; argatroban→fondaparinux	Part of anti-PF4 spectrum including VITT; DOACs accepted
TTP	Plasma exchange + steroids/rituximab	Adds caplacizumab; recombinant ADAMTS13 first choice in congenital TTP

QUESTION 10

Hodgkin Lymphoma

Textbook gap: No Hodgkin lymphoma chapter. Only link: Ch. 26 — actuarial risk of therapy-related MDS/AML is 6–9% after Hodgkin's treatment (vs 17% after myeloma treatment). Geriatric-oncology philosophy from Ch. 26/27 transfers directly.

High-Yield Points

A. Definition & Epidemiology

1. B-cell derived lymphoid malignancy; malignant cells only 1–2% of tumour mass, surrounded by reactive infiltrate.
2. **Bimodal age distribution** — peaks at 15–35 and after 55–60 years; 20–25% of cases occur >60 years.
3. Elderly HL behaves worse: more advanced stage, more B symptoms, more mixed-cellularity, higher EBV positivity. 5-yr survival falls from >90% (young) to 40–60% (>60).

B. Classification (WHO-HAEM5)

Subtype	Frequency	Features
Nodular sclerosis	~70%	Young adults, mediastinal mass
Mixed cellularity	20–25%	Older patients, HIV, EBV+
Lymphocyte-depleted	<1%	Worst prognosis — elderly, HIV, EBV+

4. **Immunophenotype:** classic RS cells — CD30+, CD15+, CD45−, CD20 weak/neg. NLPHL "popcorn" cells — CD20+, CD45+, CD30−, CD15− (mirror image).
5. 9p24.1 amplification → **PD-L1/PD-L2 overexpression** — basis for checkpoint inhibitor efficacy.

C. Clinical Features

6. Painless, rubbery cervical/supraclavicular lymphadenopathy ± mediastinal mass.
7. **Spreads contiguously** node-group to node-group (unlike NHL's unpredictable spread).
8. B symptoms (fever >38°C, night sweats, weight loss >10%) in 25–30%, more frequent in elderly.

D. Diagnosis & Staging

9. **Excisional lymph node biopsy mandatory** — FNA inadequate.
10. Staging: PET-CT, Lugano/Ann Arbor (I–IV, A/B suffix); bone marrow biopsy usually omitted.
11. **IPS score** (advanced disease): albumin <4 g/dL, Hb <10.5 g/dL, male, age ≥45, stage IV, WCC ≥15,000, lymphocytes <600/µL.

Management

Setting	Approach
Early favourable	2 cycles ABVD + 20 Gy involved-site RT, or PET-adapted chemo alone
Advanced, fit	Nivolumab-AVD (new standard, S1826), or BrECADD, or A+AVD
Advanced, ≥60 years	**Nivolumab-AVD now preferred**; bleomycin lung toxicity mortality up to 25% in >60 — avoid
Frail/unfit	Sequential BV→AVD→BV, or attenuated regimens (VEPEMB, PVAG)
Relapsed	Salvage → ASCT (fit); checkpoint inhibitors for transplant-ineligible

Late effects: secondary MDS/AML (6–9%, per Pathy's), anthracycline cardiomyopathy, radiation-related breast/lung cancer, hypothyroidism, bleomycin pulmonary fibrosis.

Latest Updates (2023–Present)

WHO-HAEM5 repositioned HL among B-cell neoplasms. ICC renamed NLPHL as "NLPBL" — WHO retained NLPHL. **SWOG S1826 (older-patient subset, ≥60, JCO 2025):** nivolumab-AVD vs BV-AVD — 2-yr PFS 89% vs 64% (HR 0.24), 2-yr OS 96% vs 85%; fewer sepsis episodes and less neuropathy. Contrast with ECHELON-1 where BV-AVD showed NO benefit over 60 — **nivolumab-AVD is a genuine breakthrough for older patients. GHSG HD21 (BrECADD):** comparable PFS in 61–75 age group but febrile neutropenia 54% vs 13% with nivolumab-AVD — decisive

difference in frail elderly.

Current Practice Update

Domain	Pathy's (2022)	Current evidence
Coverage	None — only late-effect MDS/AML risk	Fully covered by WHO-HAEM5/ESMO/NCCN
Advanced-stage therapy	Not addressed	Nivolumab-AVD (S1826) preferred, especially ≥60 yrs
Late effects	Correctly states 6–9% secondary MDS/AML risk — still valid	Confirmed; termed MN-pCT under WHO 2022

QUESTION 11

Non-Hodgkin Lymphoma — Presentation, Prognosis & Principles of Therapy

Textbook gap: No NHL chapter. Only links: Ch. 24 (lymphoma causing marrow infiltration/thrombocytopenia, acquired FVIII inhibitors); Ch. 26 (lymphoma treatment as MDS/AML risk). Geriatric-oncology philosophy transfers directly — median NHL diagnosis age is 65–70.

High-Yield Points

A. Definition, Epidemiology & Classification

1. Heterogeneous clonal lymphoid proliferations — 85–90% B-cell. WHO-HAEM5 recognises >60 entities. **5–6× commoner than Hodgkin lymphoma.**
2. Risk factors: age (dominant), immunosuppression, autoimmune disease, infections (EBV, HTLV-1, *H. pylori*→gastric MALT, HCV), pesticides.

Group	Examples	Behaviour
Indolent	Follicular (1–2), marginal zone, SLL/CLL	Incurable but long survival; watch and wait
Aggressive	**DLBCL (commonest, 30–40%)**, mantle cell	Fatal in months untreated, but curable 50–60%
Very aggressive	Burkitt, lymphoblastic	Rapidly fatal untreated, highly curable

B. Clinical Presentation

4. Painless, generalised lymphadenopathy; **non-contiguous, unpredictable spread** — most present stage III/IV.
5. **Extranodal disease in 25–40%** (far commoner than HL) — stomach, bowel, Waldeyer's ring, CNS, testis, skin.
6. B symptoms in 30–40%. Marrow involvement common (40–70% in indolent NHL).
7. Oncological emergencies: SVC obstruction, cord compression, tumour lysis syndrome.
8. In the elderly: often atypical — weight loss, falls, recurrent infection, or simply raised LDH.

C. Diagnosis & Prognosis

9. **Excisional biopsy essential** — FNA inadequate. PET-CT staging (Lugano).

IPI (mnemonic "APLES"), one point each: Age >60, **P**erformance status ECOG≥2, **L**DH above normal, **E**xtranodal sites ≥2, **S**tage III/IV. In rituximab era, 5-yr OS ranges 90% (low risk) to ~50% (high risk). NCCN-IPI uses age bands and specific extranodal sites for better discrimination in the elderly.

Principles of Therapy

10. Treatment dictated by **subtype, stage and fitness — never age alone.**
11. Indolent NHL: watch and wait legitimate until GELF/BNLI criteria met.
12. Aggressive NHL: potentially curable — treat promptly with curative intent where tolerated.
13. **Anti-CD20 antibody (rituximab/obinutuzumab) added to chemotherapy** for all CD20+ B-cell NHL.
14. Geriatric assessment BEFORE choosing intensity — first decision is curative vs palliative intent.

Treatment at a Glance

Setting	Approach
DLBCL, advanced, fit	R-CHOP×6 or Pola-R-CHP×6
DLBCL, ≥80 or frail	**R-miniCHOP** — 2-yr PFS ~47%, OS ~59%
Gastric MALT	*H. pylori* eradication alone cures 60–80%
Relapsed, fit	Salvage → ASCT, or CD19 CAR-T (now 2nd line)
Relapsed, CAR-T ineligible	CD20×CD3 bispecifics (glofitamab, epcoritamab), polatuzumab-BR

Latest Updates (2023–Present)

POLARIX (NEJM 2022): Pola-R-CHP improved 2-yr PFS to 76.7% vs 70.2% (trial capped age at 80). **POLAR BEAR trial** (Nordic, patients ≥80 or ≥75+frail): R-pola-mini-CHP had MORE serious/fatal adverse events than R-miniCHOP — **R-miniCHOP remains a sound standard in the very old. Bispecific antibodies** (glofitamab, epcoritamab — FDA 2023) transformed relapsed disease, especially valuable in older patients ineligible for CAR-T — off-the-shelf, no apheresis delay. **ZUMA-7/TRANSFORM:** CD19 CAR-T now superior to salvage+ASCT in early relapse; **PILOT trial** shows liso-cel effective in transplant-ineligible second line.

Current Practice Update

Domain	Pathy's (2022)	Current evidence
Coverage	None	Fully covered by WHO-HAEM5/NCCN/ESMO
First-line DLBCL	Not addressed	R-CHOP or Pola-R-CHP; R-miniCHOP for ≥80/frail
Relapsed disease	Not addressed	CAR-T (2nd line); bispecifics for CAR-T-ineligible

QUESTION 12

Anaemia of Chronic Kidney Disease — Type, Investigation & Treatment

Well covered: Pathy's Ch. 22, dedicated CKD section with Figure 22.3.

High-Yield Points

A. Type & Epidemiology

1. **Normocytic, normochromic, HYPOPROLIFERATIVE anaemia** — reticulocytes low for the degree of anaemia.
2. 45–70% of adults over 75 meet CKD criteria (GFR<90); 51–60% of CKD nursing-home residents also anaemic. Hb<8 g/dL carries 2× mortality risk vs 10–11 g/dL.

B. Pathophysiology — Five Mechanisms

3. **EPO deficiency** — dominant mechanism, most severe once CrCl<30 mL/min.
4. **Hepcidin excess — HIGHER than ordinary ACD** because of reduced renal clearance of the peptide.
5. Shortened RBC lifespan — uraemic toxins cut survival from 120 to 30–40 days.
6. Increased blood loss — uraemic platelet dysfunction + dialysis/phlebotomy losses.
7. Concurrent true iron deficiency very common.

C. Investigation

8. Work up when Hb <12 g/dL (men), <11 g/dL (women) — per NKF.
9. Serum EPO of limited value — may be normal/slightly high but **inappropriately low** for the anaemia.
10. Same ferritin/sTfR algorithm as ACD; exclude B12/folate, TSH, PTH, occult bleeding.

D. Treatment

11. **Correct iron FIRST, then give ESA** — enough free iron must be available for marrow use.
12. Maintain ferritin ~200 ng/mL, TSAT >20% in dialysis; no benefit above ferritin ~500 ng/mL.
13. Target Hb 11–12 g/dL — **normal range must NOT be targeted** (increased mortality from thrombosis).
14. 10–20% are ESA-hyporesponsive; ESAs linked to malignancy progression — do not treat the underlying disease.

Latest Updates (2023-Present) — KDIGO 2026

Pathy's cites KDOQI 2000/2006 — **fully superseded.** New terminology: **"iron-restricted erythropoiesis"** replaces "functional iron deficiency"; "systemic iron deficiency" replaces "absolute". **Haemoglobin target LOWERED to BELOW 11.5 g/dL** (Level 1 recommendation) — lower than Pathy's 11–12 g/dL. ESA initiation at Hb ≤9.0–10.0 g/dL in dialysis. Formal iron thresholds: start IV iron in HD if ferritin ≤500 ng/mL AND TSAT ≤30%; withhold if ferritin >700 ng/mL or TSAT ≥40%. **HIF-PHIs now addressed but as SECOND-line** (never combined with ESA); daprodustat FDA-approved 2023 with boxed thrombotic warning; roxadustat FDA-rejected. **Transfusion now symptom-based, not threshold-based**; avoid in transplant candidates.

Current Practice Update

Domain	Pathy's (KDOQI-era)	KDIGO 2026
Target haemoglobin	11–12 g/dL	**Below 11.5 g/dL** — lowered
Terminology	"Functional iron deficiency"	"Iron-restricted erythropoiesis"
New drug class	Not mentioned	HIF-PHIs — second-line only
Transfusion	Not detailed	Symptom-based; avoid in transplant candidates

QUESTION 13

Mechanisms of Drug-Induced Thrombocytopenia

Covered briefly: Pathy's Ch. 24 gives the two-mechanism framework and HIT in detail; the six-antibody subdivision below is externally sourced.

High-Yield Points

1. **Pathy's rule:** "Prescription drugs should ALWAYS be considered as a possible cause in any case of thrombocytopenia."
2. Two broad mechanisms: **direct marrow suppression**, or **induction of an immune response.**

Non-Immune Mechanisms

3. Dose-dependent marrow suppression (cytotoxics, alcohol, chloramphenicol).
4. Idiosyncratic marrow aplasia (gold, phenylbutazone, carbamazepine, thiazides).
5. Direct platelet/megakaryocyte toxicity (linezolid, valproate, daptomycin, bortezomib).
6. Drug-induced thrombotic microangiopathy (quinine, gemcitabine, clopidogrel).

Immune Mechanisms — Six Types

Mechanism	How it works	Prototype drugs
1. Hapten-dependent	Covalent binding → neo-antigen	Penicillins, cephalosporins
2. Quinine-type	**Tri-molecular complex** — drug alters antibody CDR → binds GPIIb/IIIa or GPIb/IX	Quinine, quinidine, sulfonamides, vancomycin, rifampicin
3. Fiban-type	Binds RGD site → exposes LIBS neoepitope; onset within HOURS	Tirofiban, eptifibatide
4. Chimeric antibody	Antibody against murine CDR3	Abciximab
5. Autoantibody induction	True autoantibody, drug-independent, may persist	Gold, procainamide, checkpoint inhibitors
6. Immune complex	**PF4-drug complex → IgG → FcγRIIa → platelet ACTIVATION**	Heparin (HIT), VITT

HIT — The Special Case

9. Unlike other DIT causing bleeding, **HIT causes THROMBOSIS** via platelet activation. Mortality ~30% if untreated. More common with UFH than LMWH.
10. Management: stop heparin, start argatroban (acute) then fondaparinux. **Warfarin worsens acute HIT.**

Timing Clues

Mechanism	Onset
Classic (quinine-type) DITP	5–10 days first exposure; hours on re-exposure
Fiban/abciximab-type	Within hours, even first exposure
HIT	5–10 days (or hours if heparin in prior 100 days)

Latest Updates (2023–Present)

HIT now part of a family of "anti-PF4 disorders": classic HIT, autoimmune HIT, spontaneous HIT (no heparin exposure, post-infection/surgery), and **VITT** (adenoviral-vector COVID vaccines). **DOACs (rivaroxaban, apixaban) now accepted alternatives** to argatroban/fondaparinux in stable HIT. VITT treatment: non-heparin anticoagulation + high-dose IVIg. **Immune checkpoint inhibitors** (pembrolizumab, nivolumab) recognised as an important autoantibody-type cause — may persist after stopping, often needs steroids/IVIg. Refined "antibody-first" model for quinine-type binding confirmed structurally.

Current Practice Update

Domain	Pathy's (2022)	Current evidence
GPIIb/IIIa inhibitors	Not mentioned	Fiban-type and abciximab-type mechanisms — hours to onset

HIT	Heparin-triggered only	Part of anti-PF4 spectrum including VITT; DOACs accepted
Culprit drugs	Aspirin, paracetamol, quinine, quinidine	Expanded — linezolid, checkpoint inhibitors, ADCs

QUESTION 14

Blood Transfusion

Partial coverage: No dedicated transfusion chapter, but elderly-specific PRACTICE is discussed substantially across Ch. 22 (CKD), Ch. 23 (DIC), Ch. 24 (platelet thresholds) and Ch. 26/27 (MDS transfusion support — the most detailed section).

High-Yield Points

1. **Ageing does NOT change platelet count or coagulation tests** — a low count/deranged clotting always reflects disease.
2. **CKD:** Hb<8 g/dL carried 2× mortality risk vs 10–11 g/dL — but do not normalise the target (thrombotic risk).
3. **MDS/malignancy:** threshold generally 8 g/dL, raised to **9–10 g/dL with cardiac/other comorbidities.**
4. **Transfusion SPEED should be SLOW** to avoid volume overload — explicit Pathy's point.
5. Platelets: spontaneous ICH risk below 20,000/µL; MDS prophylactic threshold <10,000/mm³; curative-intent transfusion preferred over routine prophylaxis (alloimmunisation risk).
6. Leukoreduced and irradiated products recommended to reduce isoimmunisation, infection, febrile reactions.
7. Phenotyped RBCs for chronically transfused patients; 10–15% develop alloantibodies in MDS.

Iron Overload (detailed in Pathy's)

8. Each unit contains ~250 mg iron. Chelation indicated after **20–50 units AND ferritin >1000–2500 ng/mL.**
9. Monitor with hepatic/cardiac MRI annually; deferoxamine SC or oral deferasirox; check ferritin 3-monthly.

Transfusion in DIC

10. Transfusion prevents death from bleeding, but **not from multi-organ failure** (microvascular thrombosis is the usual cause of death). ABO-incompatible transfusion is itself a rare DIC trigger.

Blood Products (supplemented)

Product	Main indication
Packed red cells	Symptomatic anaemia
Platelet concentrate	Thrombocytopenia with bleeding or prophylaxis <10,000/µL
FFP	DIC, warfarin reversal, massive transfusion
Cryoprecipitate	Hypofibrinogenaemia, DIC

Latest Updates (2023–Present)

AABB 2023: restrictive Hb<7 g/dL generally, <7.5 cardiac surgery, <8 orthopaedic/CVD; **2025 update suggests LIBERAL <10 g/dL in acute MI** (MINT trial). **TACO is now the LEADING cause of transfusion death** (34–41% of fatalities); **age ≥70 is a formally recognised independent risk factor**, alongside CHF and renal failure. **MHRA/SHOT Patient Safety Alert (April 2024):** mandatory pre-transfusion TACO risk assessment for patients ≥70; **"Code R7" mandates single-unit transfusion strategy** for stable non-bleeding patients — reassess after each unit before ordering a second. Patient Blood Management now the global WHO/AABB standard framework. Iron overload monitoring refined with cardiac/hepatic T2* MRI.

Current Practice Update

Domain	Pathy's (2022)	Current evidence
Dedicated coverage	No transfusion chapter	AABB 2023 guidelines; formal PBM frameworks
TACO	Not discussed at all	**Leading cause of transfusion death; age ≥70 a formal risk factor**
Transfusion rate	"Slow to avoid volume overload" — explicit	Validated — now formalised as core TACO mitigation
Single-unit policy	Implicit in "personalise threshold"	Explicitly mandated (2024) for TACO-risk patients

QUESTION 15

WHO Classification & Diagnostic Criteria of MDS, and its Differentials

Exceptionally well covered: Pathy's Ch. 26/27, Tables 26.1–26.4 and 27.1 — directly quotable.

High-Yield Points

Cytopenia Thresholds (Table 26.1)

Parameter	Threshold
Haemoglobin	<10 g/dL
Platelets	<1,00,000/µL
ANC	<1800/µL

WHO 2016 Classification — Full Table

Subtype	Dysplastic lineages	Ring sideroblasts	Blasts
MDS-SLD	1	<15% (<5% if SF3B1+)	PB<1%, BM<5%
MDS-RS	1–3	≥15% (≥5% if SF3B1+)	PB<1%, BM<5%
MDS-MLD	2–3	Variable	PB<1%, BM<5%
MDS-EB-1	0–3	Any	PB 2–4% or BM 5–9%
MDS-EB-2	0–3	Any	PB 5–19% or BM 10–19%, or Auer rods
MDS del(5q)	1–3	Any	PB<1%, BM<5%

Blast-count denominator redefined (2016) to include ALL nucleated marrow cells. **≥20% blasts defines AML** (vs MDS-EB-2's ceiling of 19%). **5q− syndrome:** macrocytic anaemia with NORMAL/INCREASED platelets, hypolobated megakaryocytes.

IWG/NCCN Diagnostic Criteria

Two prerequisites: (1) stable cytopenia ≥6 months (or ≥2 with specific karyotype/bilineage dysplasia); (2) exclusion of other causes.
Plus ≥1 of: dysplasia ≥10% of a lineage; blasts 5–19%; MDS-associated karyotype (del5q, del20q, +8, −7/del7q).

Differential Diagnosis

"Dysplasia in the marrow is NOT sufficient to establish myelodysplasia" (Pathy's, verbatim). Exclude: B12/folate deficiency, hypothyroidism, EBV/HIV, ethanol, chemotherapy, benzene.

Feature	CHIP	ICUS	CCUS	MDS
Cytopenias	No	Yes	Yes	Yes
Dysplasia	No	Minimal	Minimal	>10% of a lineage
Somatic mutations	Yes (VAF≥2%)	No	Up to 36%	Up to 85%
5-yr progression	0.5–1%/yr	Up to 10%	Up to 80%	—

Splenomegaly is unusual in MDS — suggests **CMML** if present. Other differentials: PNH, aplastic anaemia, congenital neutropenia, marrow infiltration by carcinoma/lymphoma.

Latest Updates (2023-Present)

WHO-HAEM5 (2022) renamed "myelodysplastic neoplasms" — two groups:

Group	Entity	Key criteria
Genetically defined	MDS-5q	del(5q) alone or +1 other (except −7/del7q)
	MDS-SF3B1	**SF3B1 mutation ALONE defines it — RS threshold removed**
	MDS-biTP53 (NEW)	≥2 TP53 mutations, or 1 + copy-number loss/cnLOH

Morphologically defined	MDS-LB	<5% BM, <2% PB blasts
	MDS-h (NEW)	Hypoplastic marrow
	MDS-IB1/IB2	Replaces MDS-EB-1/EB-2
	MDS-f (NEW)	Increased blasts + fibrosis

NPM1-mutated cases now reclassified as AML regardless of blast count. ICC creates separate "MDS/AML" for 10–19% blasts (WHO calls this MDS-IB2). **IPSS-M** (31-gene molecular score) supersedes IPSS-R.

Current Practice Update

Domain	Pathy's (WHO 2016)	WHO-HAEM5 (2022)
Ring sideroblasts	≥15% defines MDS-RS	SF3B1 mutation alone defines the entity
Hypoplastic MDS	Morphological variant, 15–20%	MDS-h — formal WHO category
NPM1-mutated	Classified by blast %	Automatically AML regardless of blast count
Prognostic score	IPSS/IPSS-R	IPSS-M is the reference standard

QUESTION 16

Bone Marrow Transplant

No standalone chapter, but Ch. 27 discusses allogeneic HSCT in real depth, framed around age eligibility — directly quotable.

High-Yield Points

1. **Definition:** conditioning regimen + infusion of HLA-compatible stem cells. Allogeneic (curative, immune graft-vs-leukaemia effect) vs autologous ("autografting" — harder in elderly due to mobilisation difficulty).
2. **Donor–versus–leukaemia effect vs GVHD** — same donor immune cells that kill leukaemia can attack gut/skin/liver.
3. Donor sources: related, unrelated, cord blood, **haploidentical.**
4. **Myeloablative conditioning:** mortality 20–50%; age was historically a contraindication because of this.
5. **Reduced-intensity/non-myeloablative (RIC)** maximises graft-vs-leukaemia while minimising toxicity — low short-term mortality up to age 70–75, OS comparable to ablative (offset by higher relapse).
6. US registry: 1106 patients ≥70; proportion rose 0.1%(2000)→3.85%(2013); **2-yr OS improved 26%→39%**; **2-yr TRM unchanged at 33–35%.**
7. **HCT-CI** (Sorror) and **Karnofsky performance status** are independent predictors of survival.
8. **CGA is "crucial in selecting candidates for transplantation"** — Pathy's explicit refrain.
9. MDS: only potential cure. AML: consolidation after CR for intermediate/unfavourable risk.
10. Autologous mobilisation becomes progressively more difficult with age.

Latest Updates (2023–Present)

Post-transplant cyclophosphamide (PTCy) has transformed GVHD prophylaxis — **BMT CTN 1703 update specifically in patients ≥70 (Blood Adv 2025):** GVHD-free/relapse-free survival 67.1% vs 29.5% with Tac/MTX, lower NRM. Authors conclude allo-HCT is **underutilised in ≥70** and PTCy should prompt greater consideration. **Haploidentical transplant with PTCy now feasible over 70** (Johns Hopkins: 2-yr OS 53%, 180-day NRM 14%). **PTCy dose reduced to 80 mg/kg** specifically improves elderly outcomes (vs 100 mg/kg). **Donor AGE, not donor type, is now the dominant variable** — younger haploidentical donor may be preferable to an older matched donor. **Letermovir CMV prophylaxis extended to 200 days** (ASTCT 2025) — particularly relevant given older recipients' higher CMV seroprevalence.

Current Practice Update

Domain	Pathy's (2022)	Current evidence
Age ceiling	RIC "rarely completed after 70"	PTCy-based transplant established into late 70s
GVHD prophylaxis	Not specified	**PTCy now preferred platform**, proven benefit in ≥70
Donor selection	Four types listed, no hierarchy	Donor age now more important than donor type
CMV prophylaxis	Not addressed	Letermovir extended to 200 days

QUESTION 17

POEMS Syndrome

Complete textbook gap. Only tangential link: the standard peripheral neuropathy work-up algorithm (Essentials of Clinical Geriatrics, Fig. 13-3) includes SPEP/UPEP/IPEP for any acquired demyelinating neuropathy — the pathway that would uncover POEMS' paraproteinaemia, though the syndrome itself is never named.

High-Yield Points

1. **POEMS** = Polyneuropathy, Organomegaly, Endocrinopathy, M-protein, Skin changes — paraneoplastic syndrome from a low-tumour-burden, **LAMBDA-restricted** clonal plasma cell neoplasm.
2. **VEGF drives the phenotype** — increased vascular permeability → oedema, increased endoneural pressure, capillary leak.
3. Distinctive marrow: combined plasma cell, lymphoid and megakaryocytic changes.

Diagnostic Criteria (Dispenzieri, updated 2023)

Category	Criteria
Mandatory major (both)	Polyradiculoneuropathy + clonal plasma cell disorder (lambda-restricted)
Additional major (≥1)	Castleman disease, sclerotic bone lesions, elevated VEGF
Minor (≥1)	Organomegaly, endocrinopathy, skin changes, papilledema, extravascular volume overload, thrombocytosis

Mnemonics: "POEMS" (2 mandatory + 3 minor); "PEST" (Papilledema, Extravascular overload, Sclerotic lesions, Thrombocytosis). **Serum VEGF** is the key diagnostic AND monitoring biomarker, independent of M-protein size.

Clinical Features

4. Polyradiculoneuropathy — symmetric, ascending, demyelinating — **mimics CIDP** (commonest cause of diagnostic delay).
5. Extravascular volume overload (oedema, effusions, ascites) and thrombocytosis **distinguish it from ordinary CIDP.**
6. Skin: hyperpigmentation, hypertrichosis, **glomeruloid haemangiomata** (near-pathognomonic).
7. Also: restrictive lung disease, pulmonary hypertension, increased stroke/thrombosis risk.
8. Untreated → death from neuropathic exhaustion or cardiopulmonary failure.

Management

Disease extent	First-line approach
Localised (≤2 lesions, no marrow involvement)	Radiotherapy 30–54 Gy ± lenalidomide-dexamethasone; ~82% cure
Disseminated, transplant-eligible	Induction chemo → **autologous stem cell transplant (ASCT)** — best CR/PFS rates
Transplant-ineligible	Lenalidomide + dexamethasone, or daratumumab + Len + dex (Dara-Rd)
Rapidly deteriorating	Corticosteroids as a bridge

IVIg gives only short-term benefit — reliance on it (as if treating CIDP) is a recognised trap that delays correct diagnosis. Anti-VEGF therapy (bevacizumab) proved largely ineffective.

Latest Updates (2023–Present)

Diagnostic criteria formally updated (Dispenzieri, 2023). **"Capillary leak phenotype"** identified (Leukemia, Dec 2024) as an independent major cause of death. **Daratumumab-based regimens (Dara-Rd)** emerging for transplant-ineligible patients, including reports in patients in their 70s. First **CAR-T and BCMA×CD3 bispecific trials** for relapsed POEMS opened 2024–2025. Comprehensive **2025 ASH review** (Dispenzieri) is now the reference source.

Current Practice Update

Domain	Pathy's	Current evidence
Coverage	Not mentioned at all	Full Mayo Clinic diagnostic/management framework

Mortality mechanism	Not addressed	Capillary leak phenotype now recognised (2024)
Transplant-ineligible therapy	Not addressed	Daratumumab-based triplets — relevant to older patients

— End of Compendium —

Compiled for night-before revision. Reference lists omitted per request; consult original chat threads for full citations.

MD Geriatric Medicine

High-Yield Answer Set — Haematology & Oncology in the Older Adult

CONTENTS

1. **Myelodysplastic Syndrome** — WHO classification, diagnostic criteria and differential diagnosis
2. **Bone Marrow Transplantation** — types, indications, procedure, complications and the older recipient
3. **POEMS Syndrome** — clinical features, laboratory investigations and treatment
4. **Hormone Therapy in Malignancy** — principles, agents, indications and adverse effects

HOW EACH ANSWER IS BUILT

- **15–20 high-yield points** in simple sentences, arranged in logical exam sequence, with keywords in bold
- **Latest Updates (2023–present)** — recent trials and guideline changes, named inline
- **Current Practice Update** — textbook position contrasted with current evidence
- **Exam one-liners** — condensed rapid-recall summary
- All laboratory values in **Indian conventional units** (calcium mg/dL, sodium mEq/L, creatinine mg/dL, haemoglobin g/dL)

Primary source: *Pathy's Principles and Practice of Geriatric Medicine*, 6th edition, supported by *Kane's Essentials of Clinical Geriatrics* and current international guidelines (WHO 2022, ICC 2022, NCCN, EBMT, ASH, SIOG). Prepared for postgraduate examination revision.

1. Myelodysplastic Syndrome

> *Describe the WHO classification and diagnostic criteria of myelodysplastic syndrome. Discuss its differential diagnosis. (15 marks)*

Definition, Epidemiology and Pathophysiology

1. **Definition.** Myelodysplastic syndromes are **clonal disorders of the haematopoietic stem cell**. The marrow is **cellular but ineffective** — it produces cells that die before they leave the marrow. The result is a paradox that defines the disease: **a hypercellular marrow with a peripheral blood cytopenia**. There is a variable risk of progression to acute myeloid leukaemia.
2. **A disease of old age.** The **median age at diagnosis is around 70–75 years**, and incidence rises steeply after 60. It is **the commonest acquired cause of marrow failure in the elderly**. Because it presents as tiredness and a low haemoglobin, it is very frequently mislabelled as "anaemia of ageing" or "anaemia of chronic disease".
3. **Two mechanisms drive the disease.** First, **somatic mutations** in a haematopoietic stem cell give it a growth advantage. Second, an **abnormal marrow microenvironment with excessive apoptosis** destroys the maturing progeny. Mutations cluster in four functional groups — **RNA splicing (SF3B1, SRSF2, U2AF1, ZRSR2)**, **DNA methylation (TET2, DNMT3A, IDH1/2)**, **chromatin/transcription (ASXL1, EZH2, RUNX1)**, and **DNA damage response (TP53)**.
4. **Primary versus secondary.** Most cases are **primary (de novo)**. **Secondary or therapy-related MDS** follows exposure to **alkylating agents, topoisomerase II inhibitors, radiotherapy, benzene or tobacco**, typically 5–7 years after treatment, and carries a much worse prognosis with a higher rate of complex karyotype and TP53 mutation.
5. **Clinical presentation is non-specific and easily missed.** Around **80–90% have anaemia** — fatigue, breathlessness, angina, falls, worsening heart failure or confusion. **Neutropenia** causes recurrent infection and **thrombocytopenia** causes bruising and mucosal bleeding. About **20% have splenomegaly**. In an older patient the presentation is often simply **functional decline with an unexplained macrocytic anaemia that does not respond to haematinics**.

Diagnostic Criteria

6. **The three requirements.** A diagnosis of MDS needs all three: **(a) a persistent cytopenia** in one or more lineages; **(b) morphological dysplasia in ≥10% of cells of at least one lineage, or an increase in blasts, or an MDS-defining genetic lesion**; and **(c) exclusion of every other cause** of cytopenia and dysplasia.
7. **Cytopenia thresholds (in Indian conventional units). Haemoglobin <13 g/dL in men and <12 g/dL in women; absolute neutrophil count <1,800/µL; platelet count <1,50,000/µL.** The cytopenia must be **persistent for at least 4–6 months** unless a defining cytogenetic abnormality or excess blasts are present.
8. **Essential investigations. Complete blood count with reticulocyte count and a peripheral smear**; **bone marrow aspirate with iron stain (Perls) plus trephine biopsy**; **conventional karyotyping** (abnormal in about 50%); **FISH** if metaphases fail; **flow cytometry**; and now **next-generation sequencing of a myeloid gene panel**. Always send **serum vitamin B12, folate, ferritin, thyroid function, copper, HIV, hepatitis serology and serum erythropoietin** before labelling a patient as having MDS.
9. **Morphological features to name in the exam. Dyserythropoiesis** — megaloblastoid change, nuclear budding and bridging, ring sideroblasts. **Dysgranulopoiesis** — **pseudo-Pelger–Huët cells (bilobed "pince-nez" nuclei)**, hypogranular neutrophils. **Dysmegakaryopoiesis** — **micromegakaryocytes and non-lobated or multiple separated nuclei**. The presence of **Auer rods** automatically places the case in a higher-risk category.
10. **Blast percentage is the single most important morphological number.** Blasts must be counted on a **500-cell marrow differential and a 200-cell blood differential**. **Under 5% marrow blasts = low blasts; 5–19% = increased blasts; 20% or more = acute myeloid leukaemia.**

WHO 2022 Classification (5th edition) — Myelodysplastic Neoplasms

The 5th edition renamed the group **"myelodysplastic neoplasms"**, retaining the abbreviation MDS, and split it into **genetically defined** and **morphologically defined** categories — a fundamental shift from counting cells to identifying the driver lesion.

Category	Defining criteria	Key exam point
A. MDS WITH DEFINING GENETIC ABNORMALITIES		
MDS with low blasts and isolated del(5q) — MDS-5q	Blood blasts <2%, marrow blasts <5%; isolated del(5q), or with one other abnormality except −7/del(7q)	**The "5q− syndrome"** — elderly woman, macrocytic anaemia, **normal or high platelets**, hypolobated micromegakaryocytes. **Best prognosis; responds to lenalidomide**
MDS with low blasts and SF3B1 mutation — MDS-SF3B1	Blood blasts <2%, marrow blasts <5%; **SF3B1 mutation**. **≥15% ring sideroblasts may substitute** where mutation testing is unavailable	**Replaces the old "MDS with ring sideroblasts".** Favourable prognosis. Practically important in India, where the morphological surrogate is still permitted
MDS with biallelic TP53 inactivation — MDS-biTP53	Marrow blasts <20%; **two or more TP53 mutations, or one mutation with loss of the other allele** (17p deletion or copy-neutral LOH)	**New category and the worst prognosis of all** — median survival about **10 months**, usually with complex karyotype. **Overrides every other**

		subtype
B. MDS DEFINED MORPHOLOGICALLY		
MDS with low blasts — MDS-LB	Blood blasts <2%, marrow blasts <5%	Replaces the old single- and multi-lineage dysplasia categories
MDS, hypoplastic — MDS-h	Marrow cellularity ≤25% (age-adjusted)	**New entity.** Must be separated from **aplastic anaemia**; often responds to **immunosuppressive therapy**
MDS with increased blasts 1 — MDS-IB1	Blood blasts 2–4% **or** marrow blasts 5–9%	Higher-risk disease; consider hypomethylating agents
MDS with increased blasts 2 — MDS-IB2	Blood blasts 5–19% **or** marrow blasts 10–19% **or Auer rods present**	**Auer rods alone make it IB2 regardless of blast count**
MDS with fibrosis — MDS-f	Blood blasts 2–19% or marrow blasts 5–19% **with grade 2–3 reticulin fibrosis**	**New entity.** Poor prognosis — median survival about 15 months. Aspirate is often a "dry tap", so **trephine is essential**

The parallel ICC 2022 classification — know the three differences

- The **International Consensus Classification** was published in the same year and is used alongside WHO. It requires **SF3B1 with a variant allele frequency ≥10%, and excludes del(5q), monosomy 7, complex karyotype and RUNX1 mutation**, whereas WHO permits ring sideroblasts as a surrogate.
- ICC created a new transitional category, **"MDS/AML" for 10–19% marrow blasts**, replacing "excess blasts-2" and signalling proximity to acute leukaemia. WHO keeps these cases as **MDS-IB2**.
- ICC groups TP53 cases as **"myeloid neoplasms with mutated TP53"** spanning MDS and MDS/AML, while WHO confines **MDS-biTP53** to under 20% blasts.

Prognostic Assessment

1. **IPSS-R (Revised International Prognostic Scoring System).** Scores **cytogenetic risk group, marrow blast percentage, haemoglobin, platelet count and absolute neutrophil count** into five categories — **very low, low, intermediate, high and very high**. The practical division is **"lower-risk" (very low, low, and part of intermediate) versus "higher-risk"**, because this decides whether treatment aims at **improving blood counts** or at **altering the natural history**.
2. **IPSS-M (Molecular IPSS, 2022).** Adds **31 gene mutations** to the IPSS-R variables and produces **six risk strata**. It **restratifies about 46% of patients** compared with IPSS-R, moving roughly a quarter upward and a fifth downward. **SF3B1 is favourable; TP53 multi-hit, RUNX1, EZH2, ASXL1, U2AF1 and NRAS are adverse.**
3. **Geriatric assessment adds independent prognostic value. Age, ECOG performance status, comorbidity (CIRS-G or Charlson), ADL/IADL dependence, frailty and transfusion burden** all predict survival independently of IPSS-R. **A frail patient with lower-risk MDS may fare worse than a fit patient with higher-risk disease.**

Differential Diagnosis — the core of this question

> **Principle:** MDS is a **diagnosis of exclusion**. Dysplasia is a morphological appearance, not a disease — many reversible conditions produce identical marrow changes. **Every reversible cause must be excluded before a lifelong clonal diagnosis is made in an older person.**

Group	Conditions	How to distinguish
Nutritional deficiency	**Vitamin B12 deficiency, folate deficiency, copper deficiency** (including after zinc excess or bariatric surgery)	Megaloblastic change with **hypersegmented neutrophils**; low B12/folate/copper; **corrects fully with replacement**. **Copper deficiency is the classic mimic — it produces ring sideroblasts and vacuolated precursors**
Toxins and drugs	**Alcohol**, isoniazid, chloramphenicol, linezolid, methotrexate, azathioprine, mycophenolate, valproate, lead, arsenic	Careful drug and alcohol history; changes reverse on withdrawal. **Alcohol and lead both cause ring sideroblasts**
Infection	**HIV, hepatitis B and C, parvovirus B19, tuberculosis, visceral leishmaniasis (kala-azar)**	Serology and marrow examination. **In India, kala-azar and disseminated tuberculosis are important causes of pancytopenia with marrow dysplasia**
Other marrow failure	**Aplastic anaemia**, **paroxysmal nocturnal haemoglobinuria**, pure red cell aplasia	**Hypoplastic MDS versus aplastic anaemia is the hardest distinction** — look for dysplasia, blasts and a clonal cytogenetic lesion. **Send flow cytometry for PNH clone in every case**
Other clonal myeloid disease	**Acute myeloid leukaemia** (≥20% blasts), **chronic myelomonocytic leukaemia**, MDS/MPN overlap, **primary myelofibrosis**, atypical CML	**CMML requires persistent monocytosis >1,000/μL and ≥10% monocytes.** Myelofibrosis shows leucoerythroblastic blood with teardrop cells and marked splenomegaly
Pre-clonal states	**ICUS** (idiopathic cytopenia of undetermined	**CHIP = mutation without cytopenia; ICUS =**

	significance), **CHIP** (clonal haematopoiesis of indeterminate potential), **CCUS** (clonal cytopenia of undetermined significance)	**cytopenia without mutation or dysplasia; CCUS = both mutation and cytopenia, but no diagnostic dysplasia.** These require surveillance, not treatment
Systemic and autoimmune disease	Chronic kidney disease, hypothyroidism, chronic liver disease, SLE, rheumatoid arthritis, Felty syndrome, **large granular lymphocytic leukaemia**	Renal and thyroid profile, autoantibodies, T-cell clonality studies
Marrow infiltration	Metastatic carcinoma, **lymphoma**, **multiple myeloma**	**Trephine biopsy is far more sensitive than aspirate**. Send serum protein electrophoresis and free light chains in every older patient

Outline of Management

14. **Treatment is decided by risk group, not by age.** In **lower-risk disease** the aim is to **improve quality of life and reduce transfusion dependence**; in **higher-risk disease** the aim is to **delay leukaemic transformation and prolong survival**.
15. **Lower-risk disease.** Observation if asymptomatic. For anaemia with **serum erythropoietin below 500 mU/mL**, use an **erythropoiesis-stimulating agent** with or without G-CSF. **Lenalidomide for del(5q)**. **Luspatercept** for ring-sideroblast-positive or SF3B1-mutated disease. **Immunosuppressive therapy (ATG plus ciclosporin)** for hypoplastic MDS in younger patients with HLA-DR15. Transfusion support as required.
16. **Iron overload matters in the transfusion-dependent patient.** Each unit of packed cells contains about 200–250 mg of iron. Consider **iron chelation with deferasirox** once the patient has received **more than 20–25 units** or the **serum ferritin exceeds 1,000 ng/mL**, provided life expectancy and risk category justify it.
17. **Higher-risk disease. Hypomethylating agents are the standard of care — azacitidine 75 mg/m² for 7 days every 28 days**, or decitabine. Response takes **4–6 cycles**, so treatment must not be abandoned early. **Allogeneic stem cell transplantation is the only curative option** and, with reduced-intensity conditioning, is now offered to selected fit patients into their **early seventies**.
18. **Supportive care underpins everything. Transfusion targets should be symptom-based rather than number-based**; platelet transfusion for counts below 10,000/µL or for bleeding; prompt antibiotics for febrile neutropenia; **avoid aspirin and NSAIDs**; vaccination; and attention to **falls risk from anaemia and to bleeding risk from concurrent anticoagulation**.
19. **Prognosis.** Median survival ranges from **almost 9 years in very-low-risk disease to under 10 months in very-high-risk disease**. Roughly **30% transform to acute myeloid leukaemia**; the remainder die of the consequences of marrow failure — **infection and bleeding** — or of unrelated comorbidity.
20. **The geriatric bottom line.** MDS in an older person is frequently **underdiagnosed** (dismissed as ageing) and then **undertreated** (dismissed as too old). Both errors are avoidable: the diagnosis needs a marrow, and the treatment decision needs a comorbidity and function assessment, not a birth certificate.

Latest Updates (2023–present)

Classification has shifted from morphology to genetics

- The **WHO 5th edition (2022)** and the **ICC (2022)** both now place **genetically defined categories first**. Validation in the **Düsseldorf MDS Registry** (5,010 patients, median age 71) confirmed that **TP53 mutational status and multilineage dysplasia are independently associated with poor outcome**, and that **MDS-biTP53 and MDS-f carry the highest risk of leukaemic transformation and the poorest survival**.
- Reclassification studies show the **2022 WHO criteria stratify patients more effectively than the 2017 criteria** (C-index 0.705 versus 0.681). Median survival for **MDS-biTP53 was 10 months and for MDS-f 15 months**, against 45 months for MDS overall.
- A practical caveat now recognised: **TP53 mutational burden matters**. Cases with a **variant allele frequency below 10%** behave better — lower IPSS-R scores, more favourable cytogenetics, more SF3B1 mutations and longer survival — supporting the ICC's 10% cut-off.

IPSS-M has become the reference prognostic tool

- Comparative work confirms that **IPSS-M risk is highest in biallelic/multi-hit TP53 disease and lowest in MDS-SF3B1**, with **median leukaemia-free survival of only 0.7 years in multi-hit TP53** and not reached in MDS with low blasts. Roughly **70% of patients are still classified morphologically and 30% genetically** under both systems.
- Importantly for practice, **response to hypomethylating agents was not associated with IPSS-M risk category** — so a high IPSS-M score predicts poor survival but does not predict failure of azacitidine.

Two new drugs for anaemia in lower-risk disease

- **COMMANDS** established **luspatercept as superior to epoetin alfa** in ESA-naïve, transfusion-dependent lower-risk MDS, moving it into the **first line** rather than after ESA failure.
- **IMerge**, the first phase 3 trial of a **telomerase inhibitor**, tested **imetelstat 7.5 mg/kg intravenously every 4 weeks** in transfusion-dependent non-del(5q) lower-risk MDS relapsed after or refractory to ESAs (median age 72–73 years). **40% achieved 8-week red cell transfusion independence**, with longer duration of independence and a

greater rise in haemoglobin than luspatercept achieved in MEDALIST — and notably this included **ring-sideroblast-negative patients**. **FDA approval followed in June 2024.**

- **NCCN now recommends imetelstat** as **category 1 second-line therapy after ESAs or luspatercept where serum erythropoietin is ≤500 mU/mL**, and as **category 2A first-line therapy where erythropoietin exceeds 500 mU/mL**. Sequencing after front-line luspatercept remains poorly defined — in IMerge, of the few patients previously given luspatercept, **86% had a reduction in transfusion burden but none achieved transfusion independence**.

Transplantation has extended into the eighth decade

- Reduced-intensity conditioning with modern graft-versus-host disease prophylaxis has made allogeneic transplantation feasible for fit patients with higher-risk MDS **into their early seventies** — see Topic 2.

Current Practice Update

Area	Textbook position	Current practice (2023–2026)
Nomenclature	Myelodysplastic syndromes	**Myelodysplastic neoplasms** (WHO 2022), abbreviation MDS retained
Classification basis	Morphology — lineage dysplasia, ring sideroblasts, blast count	**Genetics first** — del(5q), SF3B1, biallelic TP53; then morphology
Ring sideroblasts	"MDS with ring sideroblasts" is a defined entity	Replaced by **MDS-SF3B1**; **≥15% ring sideroblasts is now only a surrogate** for the mutation
New entities	Not described	**MDS-biTP53, MDS-h (hypoplastic), MDS-f (with fibrosis)**
Blasts 10–19%	MDS with excess blasts-2	**WHO: MDS-IB2. ICC: a new "MDS/AML" category**
Prognostic score	IPSS-R	**IPSS-M** — adds 31 mutations, six strata, restratifies about 46% of patients
Lower-risk anaemia	ESAs; lenalidomide for del(5q); transfusion	Add **luspatercept first-line (COMMANDS)** and **imetelstat (IMerge, approved 2024)**
Transplantation	Allogeneic transplant mentioned for higher-risk disease	**Reduced-intensity transplant now offered to fit patients into their early seventies**
Pre-clonal states	CHIP and CCUS noted	**Formally defined and separated from MDS**; surveillance, not treatment

Exam one-liners

- **MDS = hypercellular marrow with peripheral cytopenia** — ineffective haematopoiesis, not underproduction.
- **Three things must be present:** persistent cytopenia + dysplasia (or excess blasts, or a defining mutation) + exclusion of everything else.
- **Blast thresholds:** <5% low, 5–19% increased, ≥20% acute myeloid leukaemia. **Auer rods = IB2 regardless of count.**
- **Three genetic categories:** del(5q) — best; SF3B1 — favourable; **biallelic TP53 — worst (about 10 months)**.
- **Three new morphological entities:** hypoplastic (MDS-h), with fibrosis (MDS-f), and the renamed low-blast group.
- **Before diagnosing MDS, always exclude B12, folate, copper, alcohol, drugs, HIV, kala-azar and PNH.**
- **Copper deficiency is the great mimic** — it reproduces ring sideroblasts and vacuolated precursors, and it is completely reversible.
- **IPSS-R for counts and karyotype; IPSS-M when a mutation panel is available.**
- **Lower-risk = treat the anaemia. Higher-risk = treat the disease.**
- **Azacitidine needs 4–6 cycles** before response — never stop early.

2. Bone Marrow Transplantation

> *Discuss the types, indications, procedure and complications of bone marrow transplantation, with emphasis on the older patient. (15 marks)*

Definition and Types

1. **Definition.** Bone marrow transplantation — more accurately **haematopoietic stem cell transplantation (HSCT)** — is the **intravenous infusion of haematopoietic stem cells to re-establish marrow function** in a patient whose marrow has been damaged, is defective, or has been deliberately destroyed by high-dose therapy.
2. **Autologous transplantation.** The patient's **own** stem cells are collected, stored, and returned after high-dose chemotherapy. Here the transplant is simply a **rescue procedure** that permits a chemotherapy dose which would otherwise be lethal to the marrow. **There is no graft-versus-host disease and no graft-versus-tumour effect**, and **no donor is required**.
3. **Allogeneic transplantation.** Stem cells come from **another person** — an HLA-matched sibling, a matched or mismatched unrelated donor, a **haploidentical (half-matched) family member**, or umbilical cord blood. The therapeutic benefit comes from two sources: the **conditioning chemotherapy** and, crucially, the **graft-versus-tumour effect** of donor immune cells. The price of that effect is **graft-versus-host disease** and the need for prolonged immunosuppression.
4. **Sources of stem cells. Peripheral blood stem cells** mobilised with G-CSF (with or without plerixafor) are now used in over 90% of adult transplants — collection is by apheresis, engraftment is faster, but chronic GVHD is commoner. **Bone marrow** harvested from the posterior iliac crest under anaesthesia gives slower engraftment but less chronic GVHD. **Umbilical cord blood** permits greater HLA mismatch but has a limited cell dose and slow engraftment.
5. **Conditioning regimens — the key concept in geriatrics.**
   - **Myeloablative conditioning** (for example busulfan–cyclophosphamide, or cyclophosphamide with total body irradiation) destroys the marrow irreversibly. It is **too toxic for most patients over 55–60**.
   - **Reduced-intensity conditioning (RIC)** — typically **fludarabine with melphalan or busulfan** — relies mainly on the **graft-versus-tumour effect** rather than on the chemotherapy. **This is the single development that made transplantation possible in older adults.**
   - **Non-myeloablative regimens** (fludarabine with low-dose total body irradiation) are gentler still and can be given largely as an outpatient.

Indications

Autologous	Allogeneic	Comment for the older patient
Multiple myeloma (commonest indication worldwide); **relapsed/refractory diffuse large B-cell lymphoma**; relapsed Hodgkin lymphoma; germ-cell tumours; **POEMS syndrome**; some autoimmune disease	**Acute myeloid leukaemia** (intermediate/adverse risk); **higher-risk myelodysplastic syndrome**; acute lymphoblastic leukaemia; **myelofibrosis**; CML failing multiple TKIs; **aplastic anaemia**; thalassaemia and sickle cell disease; primary immunodeficiency	**Autologous transplant is far better tolerated** and, with melphalan dose reduced to 140 mg/m², is routinely performed up to about **age 75**. **Allogeneic transplant is the only curative option in higher-risk MDS and AML** and is now offered selectively into the early seventies

The Procedure — Six Steps

6. **Step 1 — Patient evaluation.** Confirm the disease and its risk category; assess **cardiac function (echocardiogram), pulmonary function with DLCO, renal and hepatic function**; screen for **HIV, hepatitis B and C, CMV, EBV and tuberculosis**; dental review; and calculate the **HCT-CI (Haematopoietic Cell Transplantation Comorbidity Index)**. Perform a **full geriatric assessment** in anyone over 60.
7. **Step 2 — Donor selection. High-resolution HLA typing at A, B, C, DRB1 and DQB1**. Preference order: matched sibling → 10/10 matched unrelated donor → mismatched unrelated donor or **haploidentical relative** → cord blood. Match **CMV serostatus** where possible. **The availability of haploidentical donors has effectively abolished the "no donor" barrier** — highly relevant in India, where unrelated donor registries are small and most patients have an available parent, sibling or child.
8. **Step 3 — Stem cell collection.** Mobilisation with **G-CSF 10 µg/kg daily for 4–5 days**, then apheresis targeting **a CD34+ cell dose of at least 2 × 10^6/kg** (ideally 4–5 × 10^6/kg) of recipient body weight.
9. **Step 4 — Conditioning.** Given over roughly **days −7 to −1**. Choice of intensity is the single most important determinant of non-relapse mortality in an older patient.
10. **Step 5 — Infusion (day 0)** and the **aplastic phase**. Cells are infused through a central line. Expect **neutrophil engraftment (ANC >500/µL) at about day +12 to +21** and **platelet engraftment at about day +20 to +30**. During this period the patient is profoundly pancytopenic and needs protective isolation, irradiated leucodepleted blood products, and antimicrobial prophylaxis.
11. **Step 6 — GVHD prophylaxis and long-term follow-up.** Traditional prophylaxis is **calcineurin inhibitor (tacrolimus or ciclosporin) plus methotrexate**. The modern standard is **post-transplant cyclophosphamide with tacrolimus and mycophenolate mofetil**. Long-term care covers immune reconstitution, revaccination from 6–12 months, endocrine

and bone surveillance, and screening for **second malignancies**.

Complications

Timing	Complication	Notes
Early (0–30 days)	**Mucositis**, nausea and vomiting, **febrile neutropenia**, bacterial and fungal sepsis, **haemorrhagic cystitis** (cyclophosphamide, BK virus), **sinusoidal obstruction syndrome** (formerly veno-occlusive disease), **engraftment syndrome**, **graft failure**	**Mucositis is the commonest and most distressing complication**, and in older patients precipitates dehydration, delirium and malnutrition. **Sinusoidal obstruction syndrome** — weight gain, tender hepatomegaly, jaundice — is treated with **defibrotide**
Intermediate (30–100 days)	**Acute GVHD**; **CMV reactivation**; **Pneumocystis jirovecii**; invasive aspergillosis; idiopathic pneumonia syndrome	**Acute GVHD affects skin, gut and liver** — maculopapular rash, secretory diarrhoea, cholestatic jaundice; graded I–IV; first-line treatment is **methylprednisolone 2 mg/kg/day**. **Ruxolitinib is the standard for steroid-refractory disease**
Late (>100 days)	**Chronic GVHD**; late infection with encapsulated organisms; **bronchiolitis obliterans**; **cataract, hypothyroidism, hypogonadism, infertility**; **osteoporosis and avascular necrosis**; iron overload; **secondary malignancy**; relapse of the original disease	**Chronic GVHD is the leading cause of late non-relapse mortality** and resembles an autoimmune disease — sicca syndrome, scleroderma-like skin change, lichenoid oral lesions, and lung involvement. It is a major cause of poor quality of life

The Older Transplant Recipient

12. **Why age was historically a barrier.** Reduced marrow and organ reserve, more comorbidity, higher infection risk, slower immune reconstitution and greater treatment-related mortality. Traditional upper limits of **55 years for allogeneic and 65 for autologous transplant** excluded the very patients in whom AML and MDS are commonest.
13. **What changed. Reduced-intensity conditioning**, **better supportive care and antimicrobial prophylaxis**, **haploidentical donors**, and **modern GVHD prophylaxis** have together shifted the question from "how old is the patient?" to **"what is the patient's comorbidity and functional reserve?"**
14. **Selecting the older candidate — four tools.**
    - **HCT-CI (Sorror index)** — the transplant-specific comorbidity score; higher scores predict non-relapse mortality.
    - **Comprehensive geriatric assessment** — **ADL/IADL, gait speed, cognition (Mini-Cog), nutrition (MNA), polypharmacy, social support and depression** all independently predict outcome.
    - **Performance status** — Karnofsky **≥70%** is the usual threshold.
    - **Disease risk index** — the likelihood of relapse must justify the risk of the procedure.
15. **Two practical points that dominate the geriatric transplant ward.** First, **caregiver availability is a hard requirement** — the patient needs a competent 24-hour caregiver for at least 100 days, and lack of one is a common reason older patients are declined. Second, **delirium is markedly commoner in older transplant recipients** and independently predicts mortality; it should be actively prevented and screened for.
16. **Prognosis.** Historical **one-year non-relapse mortality in patients aged 70 and above was around 25%**. With current conditioning and prophylaxis this has fallen substantially, and **outcomes in fit older patients now approach those of patients in their sixties**. The dominant predictors are **comorbidity, performance status and disease risk — not chronological age**.

Latest Updates (2023–present)

BMT CTN 1703 has changed GVHD prophylaxis

- This phase 3 trial randomised **431 adults** undergoing allogeneic transplantation with a matched related or matched/7-of-8 mismatched unrelated donor after **reduced-intensity conditioning** to either **post-transplant cyclophosphamide with tacrolimus and mycophenolate mofetil** or **tacrolimus with methotrexate**. The primary endpoint was **GVHD-free, relapse-free survival at one year**, with events defined as grade III–IV acute GVHD, chronic GVHD requiring systemic treatment, relapse or death.
- **Post-transplant cyclophosphamide was significantly superior**, and this regimen is now the standard prophylaxis after reduced-intensity conditioning.
- **The subgroup analysis in patients aged 70 and above is the geriatric headline.** Of the 431 patients enrolled, **96 were aged 70 or over**. Despite the concern that older adults would experience more treatment-related toxicity and GVHD, **post-transplant cyclophosphamide produced markedly better results in this group as well**, published in *Blood Advances* in 2025.
- The authors note that allogeneic transplantation **remains underutilised in adults aged 70 and above**, and that **morbidity driven by GVHD is considered the major barrier to its use** — a barrier this trial directly addresses.

Age itself is not the driver of GVHD

- A **2025 systematic review and meta-analysis of outcomes in patients aged 70 and above** reported that **graft source and prophylaxis regimen, rather than advancing age, determine GVHD incidence**. Peripheral blood stem cell grafts and tacrolimus-based prophylaxis increased GVHD, with **similar incidence across the 60–64, 65–69 and 70-and-above age groups**.

- The same review identifies **post-transplant cyclophosphamide as the principal strategy for reducing non-relapse mortality in older patients**.

Real-world feasibility in the elderly

- A ten-year single-centre series of **allogeneic transplantation for AML at a median age of 68** found it feasible and safe: **65% received grafts from a 10/10 or 9/10 matched unrelated donor, 14% from a matched sibling and 20% from a haploidentical donor**; **all received reduced-intensity conditioning** and **98% received peripheral blood stem cells**. **Acute GVHD occurred in 44%** (grade III–IV in 5 patients) and **CMV reactivation in 39% by day +100**.
- Registry work continues to show that older transplant candidates are a **frailer population overall**, reinforcing the case for formal geriatric assessment rather than age cut-offs.

Other developments worth naming

- **Ruxolitinib** is now standard for steroid-refractory acute and chronic GVHD, with **belumosudil** and **axatilimab** as further options in chronic disease.
- **Letermovir** prophylaxis has substantially reduced clinically significant CMV reactivation.
- A dedicated trial (NCT06799195) is now enrolling **adults aged 60 and older with Karnofsky performance status ≥70% receiving reduced-intensity conditioning** to optimise GVHD prophylaxis specifically in this population — the first study designed around the older recipient rather than including them incidentally.

Current Practice Update

Area	Traditional position	Current practice (2023–2026)
Upper age limit	Allogeneic 55 years; autologous 65 years	**No fixed limit** — allogeneic transplant into the **early seventies**, autologous to about **75**, decided by comorbidity and function
Conditioning	Myeloablative	**Reduced-intensity conditioning is standard in older adults**
GVHD prophylaxis	Calcineurin inhibitor + methotrexate	**Post-transplant cyclophosphamide + tacrolimus + MMF (BMT CTN 1703)**, including in patients ≥70
Donor availability	Matched sibling or unrelated donor only	**Haploidentical transplantation has removed the donor barrier**
Patient selection	Chronological age and performance status	**HCT-CI plus comprehensive geriatric assessment**
Steroid-refractory GVHD	Second-line immunosuppression, poor outcome	**Ruxolitinib**; belumosudil and axatilimab in chronic GVHD
CMV	Pre-emptive monitoring and treatment	**Letermovir prophylaxis**
Stem cell source	Bone marrow harvest	**Peripheral blood stem cells in over 90%** of adult transplants

Exam one-liners

- **Autologous = rescue** from high-dose chemotherapy. **Allogeneic = replacement plus a graft-versus-tumour effect.**
- **Myeloma is the commonest indication for autologous, AML and higher-risk MDS for allogeneic transplant.**
- **Reduced-intensity conditioning is what made transplantation possible in the elderly** — it borrows its efficacy from the donor immune system, not from the chemotherapy.
- **Aim for a CD34+ dose of at least 2 × 10^6/kg.** Neutrophils engraft around day +14, platelets around day +21.
- **Acute GVHD = skin, gut, liver, within 100 days. Chronic GVHD = an autoimmune-like multisystem illness** and the leading cause of late non-relapse mortality.
- **Post-transplant cyclophosphamide is now standard prophylaxis — and BMT CTN 1703 showed it works in patients aged 70 and above too.**
- **Age is not the risk factor; comorbidity is.** Use the **HCT-CI plus a geriatric assessment**, not the birth certificate.
- **No caregiver, no transplant** — 24-hour support for 100 days is a hard requirement.
- **Haploidentical donors mean almost every Indian patient now has a donor.**

3. POEMS Syndrome

> *Describe the clinical features, laboratory investigations and treatment of POEMS syndrome. (15 marks)*

Definition and Pathogenesis

1. **Definition.** POEMS syndrome is a **rare multisystem paraneoplastic disorder caused by an underlying clonal plasma cell disorder**. The acronym stands for **Polyneuropathy, Organomegaly, Endocrinopathy, Monoclonal gammopathy and Skin changes**. It is also called **osteosclerotic myeloma**, **Crow–Fukase syndrome** or **Takatsuki syndrome**.
2. **The acronym is incomplete and misleading.** It omits several of the most useful diagnostic features, which are captured by the second acronym **PEST — Papilloedema, Extravascular volume overload, Sclerotic bone lesions, and Thrombocytosis/erythrocytosis**. Candidates who quote only POEMS lose the features that actually make the diagnosis.
3. **Central pathogenic mechanism.** A **small, almost always lambda-restricted clonal plasma cell population** drives **markedly elevated vascular endothelial growth factor (VEGF)**. VEGF increases **vascular permeability and angiogenesis**, and explains most of the clinical picture — **oedema, effusions, organomegaly, papilloedema, skin haemangiomata and neuropathy**. Other cytokines (IL-1, IL-6, TNF-alpha) contribute.
4. **Why it matters in geriatric practice. Median age at presentation is around 50, but a substantial minority present in the sixties and seventies.** The diagnosis must be considered in what would otherwise be called a **monoclonal gammopathy of undetermined significance** when **unusual features appear — peripheral neuropathy, thrombocytosis and extracellular volume overload**. In an older patient the neuropathy is routinely blamed on **diabetes, alcohol, B12 deficiency or ageing**, and the oedema on **heart failure** — which is why diagnosis is so often delayed by years.

Clinical Features

5. **P — Polyneuropathy (present in essentially 100%; it is the dominant symptom).** A **chronic, symmetrical, ascending, distal, sensorimotor polyneuropathy**. It begins with **tingling, numbness and coldness of the feet**, then progresses to **severe motor weakness** that may confine the patient to a wheelchair. It is **predominantly demyelinating with axonal loss**, and is **painful in only a minority** — a useful clue, since it is otherwise easily mistaken for chronic inflammatory demyelinating polyradiculoneuropathy (CIDP).
6. **O — Organomegaly. Hepatomegaly, splenomegaly and lymphadenopathy.** Enlarged nodes may show **Castleman disease** on biopsy — a recognised and important association.
7. **E — Endocrinopathy.** Present in about **two-thirds**. The commonest are **hypogonadism** (erectile dysfunction, gynaecomastia, amenorrhoea), then **hypothyroidism**, **diabetes mellitus**, and **adrenal insufficiency**. Hypogonadism is the most frequent single endocrine abnormality. **Diabetes and hypothyroidism alone are too common in the elderly to count towards diagnosis.**
8. **M — Monoclonal plasma cell disorder.** Almost invariably **IgG or IgA with a lambda light chain**. The **M-protein is characteristically small** and may be missed on routine electrophoresis — **immunofixation of serum and urine is mandatory**.
9. **S — Skin changes. Hyperpigmentation** (commonest), **haemangiomata and glomeruloid angiomata** (highly characteristic), **hypertrichosis**, **acrocyanosis and plethora**, **white nails**, sclerodermatous thickening and **clubbing**.
10. **P — Papilloedema.** Present in about **one-third**. It is often **asymptomatic**, so **fundoscopy must be performed in every suspected case**; its presence strongly supports the diagnosis.
11. **E — Extravascular volume overload. Peripheral oedema, ascites, and pleural or pericardial effusions.** In an older patient this is almost always first attributed to **heart failure, nephrotic syndrome or liver disease**, and the true diagnosis emerges only when these are excluded.
12. **S — Sclerotic bone lesions.** Present in about **95%** and **virtually diagnostic in this clinical context**. They may be purely sclerotic, or sclerotic-rimmed lucencies with a **"soap-bubble" appearance**. **They are frequently missed because a bone scan is normal and a skeletal survey is often reported as unremarkable** — dedicated CT or PET-CT is needed.
13. **T — Thrombocytosis and erythrocytosis. Thrombocytosis occurs in about half**. This is a critical negative discriminator: **anaemia and thrombocytopenia point to multiple myeloma or amyloidosis, not POEMS**.
14. **Other features and the major hazard. Pulmonary hypertension, restrictive lung disease, reduced DLCO, weight loss, fatigue, clubbing and a hypercoagulable state. Arterial and venous thrombotic events — stroke, myocardial infarction, peripheral arterial occlusion — occur in roughly 10–20% and are a leading cause of death**, an especially important consideration in older patients who already have vascular risk factors.

Diagnostic Criteria (Dispenzieri)

Diagnosis requires **both mandatory major criteria**, plus **one other major criterion**, plus **at least one minor criterion**.

Mandatory major criteria (both required)	Other major criteria (one required)	Minor criteria (one required)

1. Polyneuropathy — typically demyelinating **2. Monoclonal plasma cell proliferative disorder** — almost always lambda	**3. Castleman disease** **4. Sclerotic bone lesions** **5. Elevated VEGF**	**Organomegaly** (spleen, liver, lymph nodes) **Extravascular volume overload** (oedema, effusion, ascites) **Endocrinopathy** (adrenal, thyroid, pituitary, gonadal, parathyroid, pancreatic) — **excluding diabetes and hypothyroidism alone** **Skin changes** **Papilloedema** **Thrombocytosis or polycythaemia**

Laboratory Investigations

Category	Test	Expected finding / purpose
Plasma cell clone	Serum and **24-hour urine protein electrophoresis with immunofixation**; **serum free light chain assay**; **bone marrow aspirate and trephine with immunohistochemistry and flow cytometry**	**Small IgG or IgA lambda M-protein.** Marrow usually shows **<5–10% clonal plasma cells**. **The free light chain ratio is frequently normal and therefore uninformative** — a recognised pitfall
The key biomarker	**Serum or plasma VEGF**	**Markedly elevated — usually more than three to four times the upper limit of normal.** It is the **single most useful diagnostic test** and also the **best marker for monitoring response**. Serum values run higher than plasma values, so use the same medium throughout follow-up
Neurological	**Nerve conduction studies and EMG**; CSF examination; sural nerve biopsy if needed	**Demyelinating pattern with axonal loss**, more marked in the lower limbs, with **less temporal dispersion and less conduction block than CIDP**. CSF shows **albuminocytological dissociation** — raised protein with normal cell count
Skeletal imaging	**Whole-body low-dose CT or FDG PET-CT**. Plain skeletal survey only if CT is unavailable	**Sclerotic or mixed sclerotic-lucent lesions**, commonly in **spine, pelvis and ribs**. **A bone scan is typically normal and does not exclude the diagnosis**
Haematology	Complete blood count	**Thrombocytosis and/or erythrocytosis. Anaemia and thrombocytopenia argue against POEMS**
Endocrine	Thyroid function; **testosterone (ng/dL)**, LH, FSH, prolactin; fasting glucose and HbA1c; morning cortisol; **calcium (mg/dL)** and PTH	Screens all six axes named in the criteria
Organ assessment	**Echocardiography with pulmonary artery pressure**; pulmonary function with DLCO; abdominal ultrasound or CT; **fundoscopy**; renal function (creatinine mg/dL) and urinalysis	Detects **pulmonary hypertension, effusions, organomegaly and papilloedema**
To exclude alternatives	**Congo red staining of fat pad or marrow**; vitamin B12; HIV, hepatitis B and C; HHV-8; ANA and vasculitis screen	**Amyloidosis is the principal differential** and must be actively excluded
Lymph node	**Excision biopsy of any enlarged node**	To identify **Castleman disease**, which is a major criterion

Differential Diagnosis

- **CIDP** — the commonest misdiagnosis. CIDP has **no M-protein, no VEGF elevation, no bone lesions and no systemic features**, and it responds to steroids and IVIG, which POEMS does not.
- **AL amyloidosis** — also a lambda-restricted plasma cell disorder with neuropathy, but features **autonomic failure, macroglossia, nephrotic syndrome, restrictive cardiomyopathy and lytic-free bones**, with **normal VEGF** and **positive Congo red**.
- **Multiple myeloma** — **lytic** lesions, hypercalcaemia (calcium >10.6 mg/dL), **anaemia**, renal failure and a large M-spike; neuropathy is not a defining feature.
- **MGUS with incidental neuropathy**, **Waldenström macroglobulinaemia**, **diabetic and alcoholic neuropathy**, **vasculitic neuropathy**, **hypothyroid myopathy** and **TAFRO syndrome**.

Treatment

> **Governing principle:** Treat the **clonal plasma cell population**, not the VEGF. Experience consistently shows better outcomes from **plasma-cell-directed therapy** than from targeting VEGF directly with anti-VEGF antibodies. The therapeutic armamentarium is **borrowed from multiple myeloma and light chain amyloidosis**, since randomised trials in POEMS are essentially absent.

16. **Step 1 — Decide whether the disease is localised or disseminated.** This single decision determines the entire treatment plan and depends on **whether the marrow contains clonal plasma cells and how many bone lesions are present**.

17. **Localised disease** — **one to three sclerotic bone lesions with no clonal plasma cells in the marrow.** Treat with **radiotherapy, 40–50 Gy to the lesion**. **Response is slow, taking 3–6 months or longer**, but is achieved in the majority, and **roughly half are cured by radiotherapy alone**. Surgical excision is an alternative for a solitary accessible lesion.

18. **Disseminated disease — the transplant-eligible patient. High-dose melphalan with autologous stem cell transplantation is the treatment of choice** and remains the favoured therapy on the basis of case series, producing **durable remissions even without maintenance therapy**. Typical outcomes are a **complete haematological response in about 60% and a complete VEGF response in about 50%**, with **5-year progression-free survival around 75% and 5-year overall survival around 90%**. Induction with a few cycles before transplant is often given.

19. **Disseminated disease — the transplant-ineligible or older patient.** Options are **lenalidomide with dexamethasone**, **melphalan with dexamethasone**, or a **bortezomib-based regimen**. In a phase 2 trial of **12 cycles of lenalidomide and dexamethasone in 41 patients**, the **complete haematological response rate was 46%, the neurological response rate 95% and the VEGF response rate 83%**, with **estimated 3-year overall survival of 90% and progression-free survival of 75%**. **Lenalidomide and dexamethasone is therefore the practical first choice in an older, frail or transplant-ineligible patient**, with **thromboprophylaxis mandatory**.

20. **Supportive care is not optional — it determines function. Intensive physiotherapy and occupational therapy**, **ankle-foot orthoses and gait aids**, **falls prevention** (this neuropathy is a major falls risk in an older patient), **neuropathic pain management**, **diuretics for volume overload**, **hormone replacement for the identified endocrinopathy**, and **thromboprophylaxis** given the recognised hypercoagulable state. **Steroids and IVIG do not work in POEMS** — persisting with them wastes months.

21. **Monitoring and prognosis.** Follow **VEGF, the M-protein, neurological function (ONLS or similar score), and volume status**. **Neurological recovery lags months behind the haematological and VEGF response**, so patients must be counselled not to expect early improvement in walking. Prognosis has transformed: **median survival was under 3 years in early series and 10-year overall survival now exceeds 50–60%**, even before the availability of monoclonal antibodies, bispecific T-cell engagers and CAR-T. **Adverse prognostic factors are age over 50, pulmonary hypertension, effusions, low albumin and reduced eGFR.**

Latest Updates (2023–present)

- The **ASH Education Program review (December 2025)** reframes the diagnostic approach around a single practical message: POEMS **"must be considered in the context of what might otherwise be believed to be a monoclonal gammopathy of undetermined significance, with unusual features including, but not limited to, peripheral neuropathy, thrombocytosis and extracellular volume overload."** It reaffirms that **autologous stem cell transplantation is still a favoured therapy with durable remissions even without maintenance**, and reports that **10-year overall survival was already favourable even before the era of therapeutic monoclonal antibodies, bispecific T-cell engagers and CAR-T**.
- The same review makes a point of direct examination relevance: the **free light chain ratio is normal for most of the disease course and is therefore uninformative**, whereas **VEGF tracks disease activity closely** — high at diagnosis and before second-line therapy, and not necessarily elevated before every subsequent line. **Rising creatinine with unmanageable oedema marks the terminal phase.**
- A **2025 review** confirms the current framework: diagnosis rests on the **Dispenzieri major and minor criteria**, with **VEGF an important biomarker both for diagnosis and for monitoring treatment response**; **radiotherapy for localised bone lesions**; **systemic therapy for widespread disease** using **lenalidomide, thalidomide or bortezomib-based regimens**; and **autologous transplantation for long-term remission in suitable patients**.
- A large single-centre comparison of **347 newly diagnosed patients** treated with autologous transplantation, melphalan–dexamethasone or lenalidomide–dexamethasone found that **patients receiving lenalidomide and dexamethasone had the shortest progression-free survival and a trend toward shorter overall survival** — though those undergoing transplantation had the lowest-risk disease to begin with, so the comparison is confounded. **Peri-engraftment syndrome occurs in 12–50% of transplanted patients**, and POEMS patients have a **higher-than-expected transfusion requirement** compared with myeloma patients; **splenomegaly predicts a complicated peri-transplant course**.
- **Anti-VEGF therapy remains unproven.** The effects of **bevacizumab have been described as ambiguous**, and current practice continues to direct therapy at the plasma cell clone rather than at VEGF alone.
- **Daratumumab** is under formal evaluation in POEMS, with endpoints including **plasma cell response by IMWG criteria, VEGF reduction and duration of improvement in the ONLS neurological score**. Its low toxicity would make it particularly attractive in older, transplant-ineligible patients.
- **Thrombosis is now recognised as a defining hazard** — a retrospective evaluation of **230 patients** has characterised its frequency and risk factors, reinforcing routine thromboprophylaxis, particularly with immunomodulatory drugs.

Current Practice Update

Area	Classical teaching	Current practice (2023–2026)
Diagnostic framing	Recognise the POEMS acronym	**Suspect it in any "MGUS" with neuropathy, thrombocytosis or volume overload**; remember **PEST** as well as POEMS
Key test	M-protein and bone lesions	**VEGF is the central diagnostic and monitoring biomarker**; free light chain ratio is often normal and unhelpful
Bone imaging	Skeletal survey; bone scan	**Whole-body low-dose CT or FDG PET-CT**; a normal bone scan does not exclude the diagnosis
Localised disease	Radiotherapy	**Unchanged and still correct** — 40–50 Gy, response over 3–6 months, about half cured
Fit patient	High-dose melphalan with autologous transplant	**Unchanged and reaffirmed in 2025** — durable remission without maintenance
Unfit or older patient	Alkylator plus steroid	**Lenalidomide with dexamethasone** — neurological response about 95%, VEGF response about 83%
Anti-VEGF antibody	Theoretically attractive	**Ambiguous results; not recommended as sole therapy**
Emerging	—	**Daratumumab under evaluation**; monoclonal antibodies, bispecifics and CAR-T on the horizon
Thrombosis	Noted as an association	**A leading cause of death; thromboprophylaxis is routine**

Exam one-liners

- **POEMS = a lambda-restricted plasma cell clone driving very high VEGF.**
- **Learn PEST as well as POEMS** — Papilloedema, Extravascular volume overload, Sclerotic bones, Thrombocytosis.
- **Two mandatory criteria: polyneuropathy + monoclonal plasma cell disorder.** Then one more major and one minor.
- **The three "other major" criteria: Castleman disease, sclerotic bone lesions, raised VEGF.**
- **Diabetes and hypothyroidism alone do not count** as the endocrinopathy — they are too common.
- **Thrombocytosis, not thrombocytopenia.** Anaemia and a low platelet count point to myeloma or amyloid instead.
- **Sclerotic bones with a normal bone scan** — image with CT or PET-CT, not a bone scan.
- **Fundoscopy in every suspected case** — papilloedema is present in a third and usually asymptomatic.
- **Steroids and IVIG do not work** — this is not CIDP.
- **Localised → radiotherapy. Fit and disseminated → autologous transplant. Older or unfit → lenalidomide with dexamethasone.**
- **Nerves recover months after the VEGF falls** — counsel the patient accordingly.
- **Thrombosis kills** — prophylax, especially on immunomodulatory drugs.

4. Hormone Therapy in Malignancy

> *Discuss the principles, agents, indications and adverse effects of hormone therapy in malignancy, with emphasis on the older patient. (15 marks)*

Principle

1. **The governing statement.** A number of cancers prevalent in the elderly, **including breast cancer and prostate cancer, are hormonally responsive**. Medications aimed at blocking these hormonal pathways can result in the **prevention of recurrence or prolongation of survival**.
2. **Why hormone therapy is the geriatrician's most valuable oncological tool.** It is **oral or depot, non-myelosuppressive, does not cause alopecia, and is given in the outpatient setting**. It does not require the marrow reserve that cytotoxic chemotherapy demands. Its toxicity is not acute but **cumulative and metabolic — falling on bone, muscle, blood vessel and brain**, which is precisely why it needs geriatric supervision.
3. **Five mechanisms of hormonal manipulation. Block synthesis** (aromatase inhibitors, abiraterone); **block the receptor** (tamoxifen, bicalutamide, enzalutamide); **degrade the receptor** (fulvestrant, elacestrant, imlunestrant); **suppress the pituitary–gonadal axis** (GnRH agonists and antagonists); and **ablate the gland** (orchidectomy, oophorectomy — now largely historical).

Breast Cancer — Anti-oestrogen Therapy

4. **Tamoxifen.** This selective oestrogen receptor modulator **reduces the risk of recurrence by 40% and the risk of breast cancer mortality by 31%** after primary treatment of oestrogen receptor positive breast cancer. It is an **antagonist in the breast but an agonist in bone and endometrium** — which is why it **protects bone but causes endometrial hyperplasia and carcinoma**.
5. **The CYP2D6–paroxetine interaction — a classic examination point. Tamoxifen is converted by cytochrome P450 2D6 into the more potent anti-oestrogen endoxifen.** Women who receive the potent 2D6 inhibitor **paroxetine** together with tamoxifen are at **increased risk of death from breast cancer**, because less tamoxifen is converted to the active form. **This interaction is not seen with other SSRIs.** The practical rule: **never prescribe paroxetine, fluoxetine or bupropion with tamoxifen** — use venlafaxine, citalopram or escitalopram for hot flushes or depression.
6. **Aromatase inhibitors.** In postmenopausal women, **letrozole, anastrozole and exemestane are the preferred anti-oestrogen therapy**. They **block the conversion of adrenal androgens to oestrogens by the enzyme aromatase**. NICE recommends an aromatase inhibitor in postmenopausal women at **medium or high risk of recurrence**, reserving tamoxifen for **low risk, or where an aromatase inhibitor is contraindicated or not tolerated**. **Aromatase inhibitors cause arthralgia and increase the risk of osteoporosis.** They work only in postmenopausal women.
7. **The comparative toxicity table — memorise this.**

Feature	Tamoxifen	Aromatase inhibitor
Efficacy	Standard for over 25 years	**Superior** — BIG 1-98: 5-year disease-free survival **84.0% versus 81.4%**; event HR **0.81**; distant recurrence HR **0.73**
Characteristic harms	**Thromboembolism, endometrial carcinoma, vaginal bleeding**, cataract, hot flushes	**Skeletal (fracture) and cardiac events, hypercholesterolaemia**, arthralgia, vaginal dryness
Effect on bone	**Protective** (agonist at bone)	**Harmful** — accelerates bone loss
Second cancers at 10 years (ATAC)	More **endometrial (24 versus 6), melanoma (19 versus 8), ovarian (28 versus 17)**	More **colorectal (66 versus 44) and lung (51 versus 34)**
Geriatric choice	Prefer in **severe osteoporosis** or intolerable arthralgia	Prefer in a woman with a **uterus, previous DVT or stroke**

8. **Adherence is the hidden problem in the elderly. One-quarter of women are incompletely adherent to their adjuvant hormonal regimen**, and **older age and an increasing number of co-medications are independent risk factors for non-adherence**. An unswallowed tablet has zero efficacy, so adherence must be asked about at every visit rather than assumed.
9. **Duration and extended therapy. Distant recurrences continue at a steady rate for at least 15 years after stopping 5 years of endocrine therapy.** The **ATLAS** trial showed that continuing tamoxifen to **10 rather than 5 years reduced recurrence (RR 0.84) and overall mortality (639 versus 722 deaths)**. **NICE and SIOG therefore recommend considering extension with an aromatase inhibitor after 5 years of tamoxifen**, discussing the continuing risk of **menopausal symptoms, bone density loss and musculoskeletal pain**.
10. **Primary endocrine therapy is not a substitute for surgery.** In the Cancer Research UK trial in women over 70, the **5-year risk of local progression was 8% after mastectomy, 18% after breast conservation, but 64% with tamoxifen alone**; **10-year survival was 37.7% with surgery plus tamoxifen versus 28.8% with tamoxifen alone**. **Primary endocrine therapy should only be offered to women with ER-positive tumours who are unfit for or refuse surgery.**

Prostate Cancer — Androgen Deprivation Therapy

11. **Principle. Testosterone fuels prostate cancer growth**, so **androgen deprivation therapy is the cornerstone of first-line therapy for metastatic prostate cancer**, although over time the cancer becomes **insensitive to anti-androgen therapy**. **Surgical castration was used historically; medical castration is now preferred.** The castrate level is **testosterone below 50 ng/dL**.
12. **GnRH agonists and the tumour flare — the most important practical point.** The agonists **leuprolide and goserelin initially cause release of FSH and LH with a rise in serum testosterone** before suppressing gonadotropin secretion. That initial surge may cause **increased pain from bone metastases, urinary obstruction, or spinal cord compression if it is already impending**. **The flare is prevented by giving an androgen receptor antagonist for two weeks before starting the GnRH agonist.** In a man with vertebral metastases, omitting this step can cause paraplegia.
13. **Antagonists and receptor blockers.** The GnRH antagonist **degarelix causes rapid suppression of testosterone without a flare**. Androgen receptor antagonists — **bicalutamide, flutamide and nilutamide** — were traditionally added after failure of first-line androgen deprivation.
14. **Immediate side effects. Hot flushes, erectile dysfunction, gynaecomastia and anaemia**, with an **increased risk of osteoporosis**.
15. **The four long-term geriatric harms of androgen deprivation.**
    - **Bone** — bone loss is **1–4.6% per year** on androgen deprivation, with **fracture risk increasing by up to 45% over the long term**. Seek osteoporosis risk factors (family history, low weight, previous fracture, alcohol, smoking, glucocorticoids, low vitamin D) and **prescribe calcium and vitamin D**. **Bisphosphonates are not recommended routinely — only in established osteoporosis.**
    - **Muscle** — hormone therapy causes an **increase in fat mass and sarcopenia**, which in the elderly leads to **loss of autonomy, falls and fractures**.
    - **Metabolic and cardiovascular** — **altered lipid profile, increased insulin resistance and diabetes, and increased prevalence of metabolic syndrome**, with increased cardiovascular risk demonstrated in RTOG 92-02.
    - **Cognitive** — **executive cognitive impairment**, alongside documented deficits in physical performance and increased falls.

    Prevention is explicit: modification of diet and lifestyle — walking, an adapted diet and smoking cessation — is needed to reduce the metabolic and cardiovascular impact of hormone therapy.

Other Hormone-Responsive Malignancies

16. **Endometrial carcinoma. High-dose progestins (medroxyprogesterone acetate, megestrol acetate)** and increasingly **aromatase inhibitors** are used in **low-grade, receptor-positive advanced or recurrent disease**. This is a well-suited option for the frail older woman who cannot tolerate chemotherapy.
17. **Differentiated thyroid carcinoma. TSH suppression with levothyroxine** after thyroidectomy removes the growth stimulus to residual thyroid tissue. **In the elderly this must be risk-adapted**, because over-suppression causes **atrial fibrillation, osteoporosis and heart failure**.
18. **Cancer cachexia. Megestrol acetate and medroxyprogesterone** improve appetite and weight, but the weight gained is largely fat rather than muscle, they carry a **thromboembolic risk**, and they do not improve survival.
19. **Two cautions on hormones that cause malignancy.**
    - **HRT and breast cancer (Million Women Study, 1,084,110 women)** — current users had **RR 1.66**; the risk was highest for **combined oestrogen plus progestogen (RR 2.0)**, then **oestrogen alone (RR 1.3)** and **tibolone (RR 1.45)**, with a **dose–response relationship rising to RR 2.31 after 10 or more years**. **Some excess risk persisted for more than 10 years after stopping.**
    - **Testosterone replacement** — the effects of long-term replacement on benign prostatic hypertrophy and prostate cancer **remain unclear**. It is **contraindicated in known prostate carcinoma**, and supplementation **may increase cardiovascular events in those aged 65 and above, especially during the first year**. **Monitoring of prostate size, PSA and haematocrit is mandatory.**
20. **A checklist before starting any endocrine therapy in an older patient.**
    - **Before an aromatase inhibitor:** DEXA and FRAX; **calcium (mg/dL) and vitamin D (ng/mL)** with supplementation; **lipid profile (mg/dL)**; baseline joint symptoms.
    - **Before tamoxifen:** history of venous thromboembolism, stroke, cataract or abnormal vaginal bleeding; **screen the drug chart for CYP2D6 inhibitors**.
    - **Before androgen deprivation:** DEXA, falls and fracture history; **calcium and vitamin D routinely**; **lipids, fasting glucose or HbA1c, blood pressure and ECG**; **baseline cognition (Mini-Cog), gait and grip strength**.

> **The five rules.** (1) **Confirm receptor status before prescribing** — older women are three times more likely to have receptor testing omitted. (2) **Never start a GnRH agonist in a man with impending cord compression without two weeks of anti-androgen cover.** (3) **Never combine tamoxifen with paroxetine.** (4) **Every patient on an aromatase inhibitor or androgen deprivation needs calcium, vitamin D and a bone-density plan.** (5) **Hormone therapy replaces chemotherapy in the frail — it does not replace surgery in the fit.**

Latest Updates (2023–present)

Oral SERDs — a new class in breast cancer

- **Elacestrant** was approved on **27 January 2023** for ER and/or PR-positive, HER2-negative metastatic breast cancer harbouring an **ESR1 missense mutation** after at least one line of endocrine therapy, on the basis of the phase 3 **EMERALD** trial (progression-free survival hazard ratio **0.70**). It is a **dose-dependent mixed ER agonist/antagonist**, only **11% bioavailable**, and **primarily metabolised by CYP3A4** — an important interaction consideration in polypharmacy.
- **Imlunestrant** was approved on **25 September 2025** — the third SERD after fulvestrant and elacestrant, and the second oral one. Unlike fulvestrant it **crosses the blood–brain barrier**, so it may be effective against brain metastases. The dose is **400 mg orally once daily on an empty stomach**. Among 256 patients with ESR1-mutated disease, **response was 14.3% with imlunestrant versus 7.7% with fulvestrant**; the **Guardant360 CDx assay** was approved simultaneously as the companion diagnostic.
- In **EMBER-3**, **imlunestrant with abemaciclib gave a median progression-free survival of 9.4 months versus 5.5 months with imlunestrant alone (HR 0.57)** — the first regimen to break the 6-month barrier after CDK4/6 inhibitor failure. **Elacestrant with abemaciclib** gave **14.3 versus 8.3 months**, irrespective of ESR1 mutation status.

From "treat on progression" to "treat on molecular signal"

- **ASCO now recommends routine ESR1 mutation testing at recurrence or progression**, since identifying these patients allows a switch to a more effective agent. The **SERENA-6** trial went further, using **ctDNA monitoring every 2–3 months** in patients on a first-line aromatase inhibitor plus a CDK4/6 inhibitor and switching the endocrine component to **camizestrant** on detection of an emergent ESR1 mutation, **before any clinical or radiographic progression**.
- **Geriatric relevance:** this allows a change of tablet rather than an escalation to chemotherapy — exactly the de-escalation an older patient needs.

Endocrine therapy is no longer given alone in high-risk early breast cancer

- **Adjuvant CDK4/6 inhibitors** are now added to endocrine therapy — **abemaciclib for 2 years (monarchE criteria: ≥4 positive nodes, or 1–3 nodes with grade 3 disease, tumour ≥5 cm or Ki-67 ≥20%)**, or **ribociclib 400 mg daily for 3 years (NATALEE criteria: anatomic stage II or III)**. Where a patient meets both, **ASCO favours abemaciclib**. Toxicity is real — in monarchE, **46% had grade 3 adverse events versus 15% on endocrine therapy alone, with 61% requiring dose interruption**.

Prostate — androgen receptor pathway inhibitors have moved earlier

- **Four ARPIs are now approved: abiraterone, apalutamide, darolutamide and enzalutamide**, and they are used **upfront with androgen deprivation** rather than only after failure.
- On **16 November 2023**, **enzalutamide was approved for non-metastatic castration-sensitive prostate cancer with biochemical recurrence at high risk of metastasis**, based on **EMBARK**. High risk was defined as a **PSA doubling time of 9 months or less, with PSA ≥2 ng/mL above nadir after radiotherapy or ≥1 ng/mL after prostatectomy**.
- EMBARK showed **metastasis-free survival hazard ratios of 0.42 for enzalutamide plus leuprolide and 0.63 for enzalutamide monotherapy**. The **final overall survival analysis published in 2025 showed 8-year overall survival of 78.9% versus 69.5% (HR 0.60) for the combination**, whereas **enzalutamide monotherapy did not significantly improve overall survival (HR 0.83)**.
- **A feature of major geriatric importance:** EMBARK used **intermittent therapy** — treatment for 36 weeks, then **suspension at week 37 if PSA was undetectable (below 0.2 ng/mL)**, restarting only if PSA rose to protocol-defined thresholds. **This offers a practical way to limit cumulative bone, metabolic and cognitive toxicity in older men.**
- A **Young-SIOG global drug review (2023)** cautions that the **landmark phase 3 data for ARPIs in older adults are limited to those with excellent performance status**, and that their **role in unfit older patients remains to be established**.

Current Practice Update

Area	Textbook position	Current practice (2023–2026)
Breast — agents	Tamoxifen; aromatase inhibitors; fulvestrant	Add **oral SERDs — elacestrant (2023) and imlunestrant (2025)** for ESR1-mutant disease
Breast — testing	ER and PR status	Add **ESR1 mutation testing at progression**; also PIK3CA, AKT1, PTEN
Breast — adjuvant	Endocrine therapy with or without chemotherapy	**Endocrine therapy plus a CDK4/6 inhibitor** in high-risk disease
Breast — monitoring	Clinical and radiological progression	**ctDNA monitoring with a pre-emptive switch (SERENA-6)**
CYP2D6 interaction	Avoid paroxetine with tamoxifen	**Unchanged and still correct**
Prostate — GnRH antagonist	Degarelix, "place remains to be determined"	**Established; oral relugolix now available** — no flare, better cardiac safety

Prostate — receptor blockers	Bicalutamide, flutamide, nilutamide, after first-line failure	**Second-generation ARPIs and abiraterone used upfront with androgen deprivation**
Biochemical recurrence	Not addressed	**Enzalutamide plus leuprolide (EMBARK), approved November 2023**; 8-year overall survival 78.9% versus 69.5%
ADT schedule	Continuous, or intermittent by PSA response	**PSA-guided intermittent therapy formalised** (EMBARK week-37 suspension)
Bone protection	Calcium and vitamin D; bisphosphonate only in established osteoporosis	**Unchanged, but denosumab is now preferred where creatinine clearance is below 30 mL/min**

Summary Table of Agents

Cancer	Target	Agents	Key toxicity in the elderly
Breast (ER-positive)	Oestrogen receptor / aromatase	**Tamoxifen; letrozole, anastrozole, exemestane; fulvestrant, elacestrant, imlunestrant**	**Tamoxifen** — VTE, endometrial carcinoma. **Aromatase inhibitor** — fracture, arthralgia, dyslipidaemia
Prostate	Androgen axis	**Leuprolide, goserelin; degarelix, relugolix; bicalutamide; enzalutamide, apalutamide, darolutamide; abiraterone**	**Osteoporosis, sarcopenia, falls, diabetes, cardiovascular events, cognitive decline**
Endometrial	Progesterone receptor	**Medroxyprogesterone, megestrol; aromatase inhibitors**	Thromboembolism, weight gain
Differentiated thyroid	TSH	**Levothyroxine suppression**	Atrial fibrillation, osteoporosis, heart failure
Cancer cachexia	Appetite	**Megestrol, medroxyprogesterone**	Thromboembolism; fat rather than muscle gain

Exam one-liners

- **Hormone therapy is the geriatrician's favourite oncological treatment** — a tablet or an injection that spares the marrow entirely.
- **Its price is paid slowly in bone, muscle, vessel and brain** — so calcium, vitamin D, a bone-density plan and a cardiometabolic review accompany every prescription.
- **Tamoxifen protects bone but threatens the endometrium; aromatase inhibitors protect the endometrium but threaten bone.**
- **Paroxetine plus tamoxifen increases breast cancer mortality** — other SSRIs do not.
- **Aromatase inhibitors work only after the menopause.**
- **Two weeks of anti-androgen before a GnRH agonist** — otherwise the flare can cause cord compression.
- **Castrate testosterone is below 50 ng/dL.**
- **ADT causes 1–4.6% bone loss per year and up to a 45% rise in fracture risk.**
- **Bisphosphonates in ADT only for established osteoporosis** — calcium and vitamin D for everyone.
- **Tamoxifen alone gave a 64% five-year local progression rate versus 8% after mastectomy** — endocrine therapy is not a substitute for surgery in a fit woman.
- **One-quarter of women are non-adherent, and older age plus polypharmacy predict it** — ask every visit.

MD GERIATRIC MEDICINE

Infections in the Elderly & Preventive Immunisation

Examination-Oriented High-Yield Answers

Six Questions · 15-Mark and 7-Mark Formats

Textbook Content with Evidence Updates to 2026

Primary sources: Pathy's Principles and Practice of Geriatric Medicine, 6th edition · Essentials of Clinical Geriatrics, 7th edition

Supplemented by: IDSA/SHEA, ACG, ACIP/CDC, WHO, API Indian Consensus Guidelines on Adult Immunization, Indian Academy of Geriatrics, and peer-reviewed trial evidence published up to mid-2026.

Structure of each answer: High-yield numbered points → Latest Updates (2023–present) → Current Practice Update comparison → Examiner's Tip. Recurring topics carry an additional "Extra Points to Remember" panel.

Prepared for Sudarsan · Postgraduate Revision Series

CONTENTS

No.	Question	Format	Recurs
1	Approach to pyrexia / fever of unknown origin in older adults	15 marks	—
2	Atypical presentations of infections in the elderly	15 marks	—
3	Herpes zoster in the elderly — aetiology, clinical features, management	15 marks	**Yes**
4	*Clostridioides difficile* colitis	7 marks	—
5	Vaccines / vaccinations for the elderly	15 marks	**Yes**
6	Influenza vaccination	7 marks	—
—	Rapid Revision Grid — numbers, trials, criteria	Appendix	—

How to use this document the night before

Read the numbered points first — they are the marks. Then read only the orange Examiner's Tip boxes and the Rapid Revision Grid. The "Latest Updates" sections are what separate a distinction answer from a pass answer; quote one or two named trials per answer, not all of them.

Q1. Approach to Pyrexia / Fever of Unknown Origin (FUO) in Older Adults

15-mark answer · 18 high-yield points

Definition and Classification

1. **Classical FUO (Petersdorf and Beeson, 1961)** means fever above **38.3°C** on several occasions, lasting **more than 3 weeks**, with no diagnosis after **1 week of hospital investigation**.
2. **Durack and Street (1991) revision** is the version used today: fever above 38.3°C for more than 3 weeks, undiagnosed after **3 days of inpatient work-up or 3 outpatient visits**. Four subtypes exist: **classical, nosocomial, neutropenic and HIV-associated**.
3. **Inflammation of unknown origin (IUO)** is a related entity — prolonged raised acute-phase reactants **without fever**. It is common in older people and is investigated the same way as FUO.
4. In the elderly the definition of fever itself must be lowered. Textbook criteria for long-term care residents are a **single oral temperature above 100°F (37.8°C)**, **repeated oral temperature above 99°F**, or **a rise of more than 2°F above the patient's own baseline**.

Why Older Adults Differ

5. Older people have a **lower baseline body temperature** and may show **no diurnal rise**, so a "normal" reading can conceal significant infection.
6. **Fever is blunted or absent in roughly one-third of elderly patients** with serious bacterial infection, and typically defined fever is absent in **more than half of long-term care residents** with serious infection.
7. Conversely, when fever **is** present in a very old patient it is more likely to indicate **bacterial rather than viral** illness — it carries greater diagnostic weight, not less.
8. **Hypothermia is an ominous sign** and predicts higher mortality than fever in elderly sepsis. Absence of leucocytosis also does not exclude bacterial infection.

Aetiology — Four Diagnostic Categories

9. **Infections:** tuberculosis (miliary and extrapulmonary), infective endocarditis, occult abscess (hepatic, subphrenic, perinephric, dental, prostatic), urinary sepsis, osteomyelitis, infected prosthesis or pacemaker lead. **Nearly 50% of infective endocarditis occurs in older adults**, usually on degenerative or prosthetic valves.
10. **Non-infectious inflammatory disease:** in the elderly this group is proportionally larger than in the young. **Giant cell arteritis and polymyalgia rheumatica are the single most important treatable causes**; also adult-onset Still's disease, vasculitis and sarcoidosis.
11. **Malignancy:** lymphoma (especially non-Hodgkin), renal cell carcinoma, hepatic metastases, atrial myxoma, myelodysplastic syndrome.
12. **Miscellaneous: drug fever** (antibiotics, allopurinol, phenytoin, methyldopa), venous thromboembolism, subacute thyroiditis, haematoma, and factitious fever. In India also consider **malaria, enteric fever, visceral leishmaniasis, amoebic liver abscess, brucellosis, scrub typhus and dengue**.
13. **Key geriatric principle:** FUO in an older person is usually **an atypical presentation of a common disease, not a rare disease**. In elderly series, TB, endocarditis, GCA/PMR and lymphoma account for the majority.

Clinical Approach

14. **Repeated history and repeated full examination** are the highest-yield "investigations." Undress the patient fully: examine the **sacrum and heels for pressure sores**, temporal arteries, oral cavity and dentition, joints, and do a rectal and genital examination.
15. Suspect infection in any older person with **functional decline, new confusion, falls, incontinence, reduced oral intake or failure to cooperate with staff**, even without fever. Also review the drug chart at every step — steroids, paracetamol and NSAIDs mask fever.
16. **First-line tests:** complete blood count with differential, ESR and CRP, renal and liver function, urinalysis and urine culture, **three sets of blood cultures off antibiotics**, chest radiograph, sputum for AFB/CBNAAT, HIV serology, and abdominal ultrasound. A **WBC above 14,000/mm³ or left shift above 6%** warrants careful assessment for bacterial infection.
17. **Second-line tests:** contrast CT chest/abdomen/pelvis, echocardiography (transthoracic then transoesophageal), temporal artery ultrasound or biopsy in anyone above 50 with raised ESR, autoantibodies, protein electrophoresis, and bone marrow examination if cytopenias are present.

Management Principles

18. **Avoid blind antibiotics** — they sterilise cultures and obscure the diagnosis. Exceptions are haemodynamic instability, neutropenia and strong suspicion of endocarditis. **Do not give an empirical steroid trial before excluding TB and malignancy.** Choose empirical cover with gram-negative organisms in mind, adjust all doses to renal function, and

monitor aminoglycoside levels. When repeated evaluation is unrevealing, "**suspect tuberculosis when an older patient is inexplicably failing**."

Latest Updates (2023–Present)

- **FDG PET-CT has become the pivotal second-line test.** A 2025 systematic review in *Journal of Clinical Medicine* and an AJR Expert Panel narrative review both conclude that FDG PET-CT is now the nuclear-medicine test of choice for FUO and IUO, superseding gallium-67 and labelled leucocyte scanning.
- **Elderly-specific data now exist.** A dedicated study of patients aged 75 and above (*Diagnostics*, 2025) found FDG PET-CT contributed to a definitive diagnosis in **30.8%** of cases and changed treatment in **38.8%**, but produced **false positives in 15.2%** — from atherosclerosis, degenerative change and recent infection. An accompanying 2025 commentary in *Journal of Cellular Immunology* stresses that scans in frail patients need multidisciplinary interpretation and careful patient selection.
- **Diagnostic yield across all ages.** A 2025 retrospective cohort of 284 FUO patients (*Frontiers in Medicine*) reported non-infectious inflammatory disease in 26.8%, malignancy in 23.2%, infection in 18.7%, and **31.3% still undiagnosed** — confirming that a large undiagnosed fraction persists despite modern imaging. A 2026 Italian multicentre study of 929 patients (*Journal of Nuclear Medicine*) further supports routine early use.
- **Known false-negative traps** for PET-CT include SLE, CMV, toxoplasmosis, urinary infection and pyelonephritis. Conversely PMR shows a highly characteristic pattern — uptake at **shoulders, hips, greater trochanters, ischial tuberosities and interspinous bursae** — which is now an important recognised diagnostic clue in older patients.
- **A negative FDG PET-CT is itself useful**, carrying a high negative predictive value, excluding focal disease and predicting a favourable prognosis — supporting watchful waiting rather than escalating invasive tests in a frail patient.
- **Vital-sign evidence refined.** A 2025 systematic review and meta-analysis in *Age and Ageing* examined whether older adults truly show different vital signs in bacterial infection, confirming that blunted responses are real but heterogeneous — some older patients present identically to younger ones.

Current Practice Update

Aspect	Textbook Position	Current Evidence (2023–2026)
Definition applied	Petersdorf/Durack criteria; fever thresholds emphasised	FUO and **IUO treated as one diagnostic problem** with the same algorithm; age-adjusted fever thresholds formally endorsed
Imaging strategy	Stepwise conventional imaging; radionuclide scanning peripheral	**FDG PET-CT as early second-line, sometimes first-line** modality; gallium and labelled leucocyte scans obsolete
Yield expectation	Not quantified for older adults	Diagnosis established in ~31% of over-75s by PET-CT; treatment altered in ~39%; false positives ~15%
Role of a negative scan	Not addressed	Negative scan has **high NPV and favourable prognosis** — supports de-escalation in frailty
Undiagnosed proportion	Implied to fall with better tests	Remains high at **~30%** even with modern imaging

Practical limitation in the Indian setting

FDG PET-CT costs roughly ₹20,000–30,000 and is confined to tertiary centres. In practice, exclude **tuberculosis, malaria, enteric fever, HIV and amoebic liver abscess** thoroughly and cheaply first — CBNAAT, peripheral smear/rapid antigen, blood culture, HIV serology and abdominal ultrasound — before requesting PET-CT. State this cost-consciousness explicitly in your answer; examiners reward it.

Examiner's Tip — verbatim lines to write

• "In older adults, FUO is usually an **atypical presentation of a common disease**, not a rare disease."
• "Fever in the elderly is defined as a single oral temperature above 100°F, a repeated temperature above 99°F, or a rise of more than 2°F above baseline."
• "**Giant cell arteritis is the most important treatable cause** of FUO in patients over 50 and must be actively excluded."
• "Hypothermia in an infected older patient carries a worse prognosis than fever."
• "I would not start blind antibiotics or an empirical steroid trial before excluding tuberculosis and malignancy."
• Draw a simple algorithm: **History and repeated examination → first-line tests → CT and echocardiography → FDG PET-CT → targeted biopsy.**

Q2. Atypical Presentations of Infections in the Elderly

15-mark answer · 18 high-yield points

Definition and Importance

1. **Atypical presentation** means an infection that presents without its classical localising or systemic features, and instead declares itself through **non-specific geriatric syndromes**.
2. It matters because **atypical presentation delays diagnosis and treatment, and is independently associated with higher mortality**. Underreporting of symptoms, impaired communication, coexisting disease and altered physiological responses all contribute.

Mechanisms — Why Presentations Change

3. **Immunosenescence:** thymic involution reduces T-cell production and function; **cell-mediated immunity falls markedly, humoral immunity to a lesser degree**; B-cell numbers and phagocytic and complement function are relatively preserved. Adaptive immunity is affected far more than innate immunity.
4. **Inflammageing:** chronic low-grade inflammation with raised IL-6 and TNF raises the baseline CRP and blunts the incremental acute-phase response, so a "modest" CRP rise may represent severe illness.
5. **Impaired thermoregulation:** reduced pyrogenic cytokine production, lower muscle mass, blunted vasoconstriction and a lower baseline temperature together produce the blunted febrile response.
6. **Loss of barrier and reserve:** impaired cough reflex, reduced mucociliary clearance and decreased mucus secretion predispose to aspiration; prostatic obstruction predisposes men to urinary infection; skin thinning and pressure sores create portals of entry.
7. **Comorbidity masking:** existing chronic disease conceals acute infection. Septic arthritis characteristically occurs in an already abnormal joint, so **synovial fluid must be aspirated whenever an older patient has an "acute exacerbation" of joint disease**.

The Classical Atypical Syndromes

8. **Delirium or acute confusion** — the single commonest atypical presentation. Infections not involving the CNS routinely cause confusion in the elderly, particularly with pre-existing dementia.
9. **Falls and unexplained loss of mobility** — a fall in a previously ambulant older person should prompt a search for infection.
10. **Acute functional decline** — sudden inability to perform activities of daily living. "Acute unexplained functional deterioration should alert the physician to a potential acute infectious process."
11. **New or worsening incontinence**, **anorexia and reduced oral intake**, **generalised weakness and fatigue**, and **failure to cooperate with carers**.
12. **Absence of fever** in one-third of cases, and frank **hypothermia**, which predicts mortality. **Absence of leucocytosis does not exclude bacterial infection.**
13. **Decompensation of a chronic condition** — new atrial fibrillation, worsening heart failure, or hyperglycaemic crisis may be the only presentation of sepsis.

Site-Specific Atypical Patterns

14. **Pneumonia:** fever and cough are **absent in 30–40%** of elderly cases. Look instead for tachypnoea above 30/min, tachycardia above 110/min, confusion or falls. Pneumonia remains the commonest infective cause of death in geriatric patients.
15. **Urinary tract infection:** presents as altered mental status rather than dysuria; over a quarter of over-70s with bacteraemic UTI present with confusion. **Do not send urine culture in an asymptomatic resident** — asymptomatic bacteriuria is common and should not be treated in the absence of obstructive uropathy.
16. **Intra-abdominal sepsis:** appendicitis and diverticulitis present without guarding or rigidity; failure to seek medical attention contributes to the higher morbidity and mortality of appendicitis in older adults.
17. **Tuberculosis:** presents as weight loss, anorexia and failure to thrive rather than haemoptysis; reactivation is favoured by malnutrition and comorbidity. A useful rule — **suspect tuberculosis when an older patient is inexplicably failing**. Rates are higher in long-term care than in the community.
18. **COVID-19 and respiratory viruses:** Pathy's records that **delirium and falls are common atypical presentations of COVID-19** in older people, with delirium in around 30% of hospitalised cases and up to 70% in severe disease. Atypical presentation should trigger testing, not reassure.

Practical Rules for the Ward

- Use an **age-adjusted fever threshold** and always compare against the patient's own baseline.
- **Undress and examine fully** — sacrum, heels, perineum, feet, oral cavity, intravenous and catheter sites.
- Change from baseline over **hours to a day** is the alarm signal, not the absolute value of any single observation.

- Pulse oximetry if respiratory rate is above 25; chest radiograph if hypoxaemia is documented or suspected; blood cultures in nursing-home residents have low yield and rarely change therapy.

Latest Updates (2023–Present)

- **The blunted-response doctrine has been refined, not overturned.** A 2025 systematic review and meta-analysis in *Age and Ageing* examined vital signs in adults hospitalised with bacterial infection and found blunted responses are real but **heterogeneous** — a substantial proportion of older patients present with entirely typical vital signs. The clinical implication: an atypical presentation should never be assumed, and a typical presentation should never be discounted.
- **Sepsis screening tools underperform in this population.** A 2024 review in *Geriatric Care* and a 2026 narrative review in *Anaesthesia Critical Care and Pain Medicine* both confirm that SIRS criteria, qSOFA and fever-based screens have reduced sensitivity and specificity in older adults, and describe interest in a **geriatric-adapted qSOFA** incorporating frailty and functional decline.
- **Fever absence quantified more precisely.** Contemporary reviews place absence of fever in older patients with serious infection at **30–50%**, with hypothermia carrying a distinct and worse prognostic signature than fever in elderly sepsis.
- **Biomarkers are increasingly favoured over vital signs.** Current reviews advocate procalcitonin and lactate for early identification because they are less blunted by immunosenescence than temperature and white cell count, and remain informative after antibiotics have been started.
- **Frailty is now formally recognised as an effect modifier** rather than a background variable — Pathy's itself notes that frailty acts synergistically to increase both susceptibility and severity, and current literature builds on this by recommending frailty scoring at the point of sepsis triage.

Current Practice Update

Aspect	Textbook Position	Current Evidence (2023–2026)
Blunted febrile response	Fever blunted or absent in a substantial minority	Confirmed at **30–50%**, but explicitly **heterogeneous** — many older patients present typically
Screening tools	Not addressed for geriatrics	SIRS/qSOFA **less sensitive and specific** in older adults; geriatric-qSOFA proposed
Diagnostic markers	WBC above 14,000/mm³ or left shift above 6%	Increasing reliance on **procalcitonin and lactate**, which are less blunted by immunosenescence
Frailty	Described as a risk factor	Treated as a **formal effect modifier** to be scored at triage
Delirium as a presentation	Recognised, mechanism undefined	Confirmed and quantified; routine delirium screening advocated as an infection-detection strategy

Examiner's Tip — verbatim lines to write

• "In the elderly, the **geriatric syndromes are the symptoms of infection** — delirium, falls, functional decline, incontinence and anorexia."
• "Fever is absent in 30–50% of older patients with serious infection, and **hypothermia carries a worse prognosis than fever**."
• "Absence of leucocytosis does not exclude bacterial infection."
• "Any acute unexplained functional deterioration is an infection until proven otherwise."
• "Standard sepsis screening tools are less sensitive in older adults, so I would rely on **change from the patient's own baseline** together with lactate and procalcitonin."
• Close with the classical Osler quotation in your own words: in old age pneumonia may be latent, with slight cough, ill-defined signs, and constitutional symptoms out of all proportion.

Q3. Herpes Zoster in the Elderly — Aetiology, Clinical Features and Management

*15-mark answer · 19 high-yield points · **Recurring topic***

Definition and Aetiology

1. **Herpes zoster (shingles)** is a localised, painful vesicular eruption caused by **reactivation of latent varicella-zoster virus (VZV)** from dorsal root and cranial nerve ganglia, where it has lain dormant since primary chickenpox.
2. The trigger for reactivation is **a decline in VZV-specific cell-mediated immunity**, which occurs with ageing (immunosenescence) and with immunosuppression from disease or therapy.
3. **Risk factors:** increasing age, malignancy (especially lymphoproliferative), HIV, chemotherapy, corticosteroids and other immunosuppressants, transplantation, diabetes, chronic kidney disease, and physical or psychological stress.

Epidemiology

4. Incidence rises sharply with age. In a prospective vaccine trial the incidence in those over 60 was **11.8 cases per 1000 persons per year**, against an all-age rate of **1.2–3.4 per 1000 per year**.
5. **Zoster occurs in up to half of all individuals surviving to age 85.**
6. **Increasing age is the strongest risk factor for postherpetic neuralgia (PHN)**, which affects **more than 40% of those over 60** who develop zoster.
7. In India, an ICMR estimate places zoster incidence at about **3.3 cases per 1000 persons per year**, and seroprevalence studies show **over 90% of Indians are VZV-seropositive by age 40** — so essentially the entire older Indian population is at risk.

Clinical Features

8. **Prodromal phase (2–7 days):** pain, itching, tingling or burning in a dermatome, often with malaise and low-grade fever. In the elderly this is frequently **misdiagnosed as myocardial infarction, renal colic, cholecystitis or a disc lesion**.
9. **Eruptive phase:** grouped vesicles on an erythematous base in a **unilateral dermatomal distribution that does not cross the midline**; vesicles become pustular, crust in 7–10 days and heal over 2–4 weeks.
10. **Commonest sites:** thoracic dermatomes (about half of cases), followed by the ophthalmic division of the trigeminal nerve.
11. **Herpes zoster ophthalmicus:** V1 involvement. **Hutchinson's sign** — vesicles on the tip or side of the nose (nasociliary branch) — predicts ocular involvement and mandates urgent ophthalmology referral; complications include keratitis, uveitis, acute retinal necrosis and blindness.
12. **Ramsay Hunt syndrome (herpes zoster oticus):** geniculate ganglion involvement causing **ipsilateral facial palsy, vesicles in the external auditory canal or on the palate, vertigo and sensorineural deafness**. Recovery of the palsy is poorer than in Bell's palsy.
13. **Disseminated zoster:** more than 20 vesicles outside the primary dermatome, with possible visceral involvement (pneumonitis, hepatitis, encephalitis). Suggests significant immunosuppression — investigate for occult malignancy or HIV.
14. **Postherpetic neuralgia:** defined as pain persisting **90 days or more after rash onset**. It is **neuropathic pain** — burning, lancinating or electric-shock in character, with allodynia and hyperalgesia. It causes insomnia, depression, weight loss and social withdrawal, and is the principal driver of the disease burden in older people.
15. **Other complications:** zoster sine herpete (dermatomal pain without rash), segmental motor palsy, myelitis, meningoencephalitis, VZV vasculopathy causing stroke, and bacterial superinfection.

Diagnosis

16. The diagnosis is **clinical** in the vast majority. Confirmation, where needed, is by **VZV PCR of vesicle fluid or a swab of the base of a de-roofed lesion** — the most sensitive test. Tzanck smear shows multinucleate giant cells but cannot distinguish HSV from VZV. Direct fluorescent antibody testing is an alternative.

Management

17. **Antiviral therapy must be started within 72 hours of rash onset. Aciclovir, famciclovir and valaciclovir all reduce acute pain and shorten the duration of chronic pain** when started within this window. Beyond 72 hours, still treat if new vesicles are still forming or if there is ophthalmic, motor or disseminated involvement.
    - Valaciclovir **1 g three times daily for 7 days** — preferred for compliance
    - Famciclovir **500 mg three times daily for 7 days**
    - Aciclovir **800 mg five times daily for 7–10 days** — cheapest and most available in India
    - **All doses must be reduced for creatinine clearance below 30 ml/min**; inadequate dose reduction causes aciclovir neurotoxicity presenting as delirium, which is easily mistaken for the infection itself
    - Intravenous aciclovir 10 mg/kg 8-hourly for disseminated, ophthalmic-severe, or CNS disease

18. **Acute pain control:** analgesia is important. Paracetamol and NSAIDs for mild pain; **those with moderate to severe pain may require opioids**. Add **gabapentin or corticosteroids if pain persists**. Keep lesions clean and dry, and use non-adherent dressings.
19. **Treatment of established PHN: topical lidocaine patch, gabapentin and pregabalin are the FDA-approved agents**; **nortriptyline and opioids have also been shown to be of benefit**, and combination therapy may be necessary. In older adults, start low and titrate slowly; avoid amitriptyline in favour of nortriptyline because of anticholinergic burden, and reduce gabapentinoid doses in renal impairment.

Critical negative to state in the answer

Corticosteroids have NOT been shown to reduce the incidence of postherpetic neuralgia in the elderly. They may be used for acute pain relief or for specific complications such as Ramsay Hunt syndrome, but they must not be offered as PHN prophylaxis. This is a favourite examiner trap.

Prevention

20. Two vaccines exist. **Zostavax**, the live attenuated vaccine (FDA 2006), is only about **30% protective** and reduced zoster incidence with a hazard ratio of 0.45, ophthalmic zoster HR 0.37 and zoster hospitalisation HR 0.35. **Shingrix**, the recombinant adjuvanted vaccine (FDA 2016), is about **90% protective**.
21. **ACIP recommends two doses of Shingrix for all adults aged 50 and above**, given 2–6 months apart, **regardless of prior chickenpox and regardless of prior Zostavax**. Those who previously received Zostavax should still receive Shingrix.

Latest Updates (2023–Present)

- **Zoster vaccination and dementia — the major development of this cycle.** A landmark **natural experiment in Wales published in *Nature* (2025)** exploited an age cut-off for vaccine eligibility and found herpes zoster vaccination reduced dementia incidence by approximately **20%** over 7 years — the strongest causal-inference evidence available.
- A parallel ***JAMA* (2025)** analysis of Australian data using the same natural-experiment design reproduced the association, as did a ***Nature Medicine* (2024)** study showing the recombinant vaccine gave a **17% increase in diagnosis-free time** over 6 years compared with the live vaccine.
- Cohort data are consistent. A *Vaccine* (2025) study of 4.5 million people found two doses of RZV associated with dementia hazard ratio **0.68**; a *Nature Communications* (2026) Kaiser Permanente study found a **51% lower risk**; and an *Annals of Internal Medicine* (2026) target-trial emulation in skilled-nursing residents found a **24% relative and 6% absolute reduction** in 4-year dementia risk.
- **Zoster infection itself is a dementia risk factor.** The same *Vaccine* (2025) analysis found a zoster diagnosis before vaccination raised dementia hazard to **1.47**, while **antiviral treatment of zoster was protective (HR 0.42)**. *Nature Medicine* (2025) confirmed a consistent association between VZV reactivation and dementia in large-scale health records.
- **The evidence is not unanimous.** A 2025 systematic review and meta-analysis in *Human Vaccines and Immunotherapeutics* found an overall 32% dementia risk reduction with zoster vaccination, but some earlier meta-analyses showed no significant benefit. **The first randomised trial is now under way** — **DAN-ZOSTER**, a pragmatic RCT in adults aged 65 and above testing RZV against major adverse cardiovascular events and dementia, began recruiting in 2026 with completion expected 2029. *Results awaited.*
- **Real-world effectiveness confirmed.** A 2026 *Clinical Infectious Diseases* study confirmed sustained effectiveness of the adjuvanted recombinant vaccine in US adults aged 50 and above.
- **India-specific:** GSK launched **Shingrix in India in April 2023** for adults aged 50 and above. It is available privately at roughly **₹8,000–12,000 per dose (₹16,000–24,000 for the two-dose course)**, is not in any government programme, and requires 2–8°C cold chain. The **API Indian Consensus Guidelines on Adult Immunization (2024, updated 2026)** include herpes zoster vaccination for older adults.

Current Practice Update

Aspect	Textbook Position	Current Evidence (2023–2026)
Preferred vaccine	Both listed; Shingrix noted as 90% protective vs Zostavax 30%	**Zostavax withdrawn** in the US and many countries since 2020; RZV is the only vaccine in use
Non-zoster benefit	Not described	**~20% reduction in dementia incidence** (*Nature* 2025 natural experiment), replicated across multiple designs; RCT (DAN-ZOSTER) ongoing
Zoster as a disease risk	Complications limited to PHN, ocular, neurological	Zoster infection itself associated with **increased dementia risk (HR 1.47)**; antiviral treatment protective (HR 0.42)
Antiviral window	Within 72 hours of rash onset	Unchanged, but treat beyond 72 h if new vesicles forming, or ophthalmic/motor/disseminated disease

Steroids for PHN prevention	Not shown to reduce PHN incidence	**Unchanged** — still not recommended for PHN prophylaxis
Indian availability	Not addressed	Shingrix available since April 2023; **cost is the principal barrier**; included in API 2024 consensus guidelines

Extra Points to Remember — recurring topic

1. The three complications that must never be missed: herpes zoster ophthalmicus with Hutchinson's sign, Ramsay Hunt syndrome, and disseminated zoster signalling occult immunosuppression.

2. Aciclovir neurotoxicity in renal impairment presents as delirium, myoclonus and hallucinations — and is routinely mistaken for the infection itself. Always calculate creatinine clearance before prescribing.

3. Zoster sine herpete — dermatomal neuropathic pain with no rash. Diagnosed by VZV PCR of CSF or blood, or by serology. Consider it in unexplained unilateral segmental pain in an older person.

4. VZV vasculopathy is an under-recognised cause of stroke in older adults, particularly after ophthalmic zoster, and is one mechanism proposed to link zoster with dementia.

5. Zoster in the immunocompromised: Shingrix is non-live and therefore **safe in immunocompromised adults** (licensed from age 18 in this group) — the key practical advantage over Zostavax.

6. Nortriptyline over amitriptyline in older people. Tricyclics are on the Beers list; nortriptyline has a substantially lower anticholinergic burden.

7. Infectivity: zoster transmits VZV to non-immune contacts causing chickenpox, not zoster. Localised zoster requires standard precautions with lesions covered; disseminated or immunocompromised zoster requires airborne plus contact precautions.

Examiner's Tip — verbatim lines to write

• "Herpes zoster results from reactivation of latent VZV in the dorsal root ganglia due to **waning VZV-specific cell-mediated immunity**."
• "**Age is the strongest risk factor for postherpetic neuralgia**, which affects over 40% of patients above 60."
• "Antivirals must be started **within 72 hours of rash onset**, with dose reduction if creatinine clearance is below 30 ml/min."
• "**Corticosteroids do not reduce the incidence of postherpetic neuralgia.**"
• "Two doses of the recombinant adjuvanted vaccine are recommended for all adults above 50, regardless of previous chickenpox or previous live vaccine."
• The distinction line for a top answer: "A 2025 *Nature* natural experiment in Wales showed zoster vaccination reduced dementia incidence by about 20%, with a randomised trial, DAN-ZOSTER, now under way."

Q4. *Clostridioides difficile* Colitis

7-mark short note · 10 high-yield points

High-Yield Points

1. **Definition:** antibiotic-associated colitis caused by toxin-producing *Clostridioides difficile*, a spore-forming anaerobic gram-positive bacillus. Disease is mediated by **toxin A (enterotoxin) and toxin B (cytotoxin)**; the hypervirulent **NAP1/BI/027 ribotype** causes more severe disease.
2. **Why the elderly:** age above 65 is an independent risk factor. Contributory factors are **recent antibiotics** (clindamycin, cephalosporins, fluoroquinolones, co-amoxiclav), **proton pump inhibitor use**, hospitalisation or long-term care residence, tube feeding, comorbidity and reduced gut microbiota diversity. Pathy's notes a possible causal relationship between PPI therapy and *C. difficile* infection, and that frail and institutionalised older people have less diverse microbiota.
3. **Clinical features:** watery diarrhoea (three or more unformed stools in 24 hours), lower abdominal cramps, fever, and leucocytosis. In older adults it may present **atypically as delirium, dehydration or unexplained leucocytosis**. Essentials states that if a resident has severe fever, abdominal cramps or diarrhoea with or without blood or leucocytes in stool, **initial evaluation for *C. difficile* should be performed**.
4. **Severe and fulminant disease:** severe disease is defined by **WBC above 15,000 cells/µl or serum creatinine at or above 1.5 mg/dl**. Fulminant disease adds hypotension, shock, ileus or toxic megacolon. **Paradoxical cessation of diarrhoea with abdominal distension signals ileus or toxic megacolon, not improvement.**
5. **Diagnosis:** test only **unformed stool from symptomatic patients**. A two-step algorithm is standard — a sensitive screen (GDH antigen or NAAT/PCR) followed by a specific toxin EIA. **NAAT positivity with toxin negativity may indicate colonisation rather than disease.** Never perform a test of cure. Abdominal CT for suspected megacolon; flexible sigmoidoscopy shows **pseudomembranes**.
6. **First principles of management: stop the inciting antibiotic** wherever possible, **stop the PPI if not clearly indicated**, correct fluid and electrolyte losses, and **avoid antimotility agents such as loperamide** in acute severe disease.
7. **Treatment of an initial episode: fidaxomicin 200 mg twice daily for 10 days** is now first-line; **oral vancomycin 125 mg four times daily for 10 days** is an acceptable alternative. **Metronidazole is no longer preferred** and should be reserved for settings where neither is available.
8. **Fulminant disease: oral vancomycin 500 mg four times daily** (plus vancomycin per rectum if ileus) **with intravenous metronidazole 500 mg 8-hourly**. Early surgical review for subtotal colectomy or diverting loop ileostomy with colonic lavage.
9. **Recurrence:** occurs in **15–25%** after a first episode and rises to 40–60% after subsequent episodes. First recurrence — fidaxomicin (standard or extended-pulsed) or tapered-pulsed vancomycin. Multiple recurrences — tapered-pulsed vancomycin, vancomycin followed by rifaximin, or **faecal microbiota transplantation, reserved for those who have had appropriate antibiotic therapy for at least two recurrences**.
10. **Infection control: contact precautions with gowns and gloves; soap-and-water handwashing because alcohol rub does not kill spores**; single room; sporicidal (chlorine-based) environmental cleaning; dedicated equipment; and antimicrobial stewardship as the single most effective preventive measure.

Latest Updates (2023–Present) — three points

- **Fidaxomicin is firmly first-line.** The **IDSA/SHEA 2021 focused update** and the **ACG 2021 guideline** both place fidaxomicin above vancomycin for initial and recurrent non-fulminant disease, based on lower recurrence: **NNT of 10** to prevent one recurrence at 4 weeks for an initial episode, and **NNT of 7 at 30 days** for recurrent disease. Real-world data (*Open Forum Infectious Diseases*, 2024 and 2025) confirm reduced recurrence, including in immunocompromised hosts, though a 2024 IDSA Emerging Infections Network survey showed uptake is still limited by cost.
- **Bezlotoxumab was discontinued in January 2025** and is no longer available — a change every candidate should know, since older textbooks and guidelines still list it. It had been recommended for those with recurrence in the preceding 6 months, with an FDA warning against use in congestive heart failure.
- **Microbiome therapeutics have replaced conventional FMT in many settings.** Two FDA-approved live biotherapeutics are now in use — **Rebyota (faecal microbiota, live-jslm; rectal)** and **Vowst (faecal microbiota spores, live-brpk; oral)** — for prevention of recurrence after antibiotic treatment. The **AGA 2024 guideline** restricts FMT to adjunctive use after standard-of-care antibiotics, or as a last resort in fulminant disease not responding to therapy. A 2025 Norwegian open-label RCT in *Annals of Internal Medicine* tested FMT enema as **first-line** therapy against vancomycin in primary infection — an area of active investigation, *with wider confirmation awaited.*

Current Practice Update

Aspect	Textbook Position	Current Evidence (2021–2026)
First-line drug	Metronidazole/vancomycin era; specific regimens not detailed	**Fidaxomicin first-line**; vancomycin acceptable alternative; **metronidazole not preferred**

Monoclonal antibody	Not addressed	Bezlotoxumab was recommended 2021–2024 but **discontinued January 2025**
Microbiome therapy	Not addressed	**Rebyota and Vowst** FDA-approved; FMT positioned as adjunct or last resort (AGA 2024)
Diagnostic strategy	"Initial evaluation for *C. difficile* should be performed"	Two-step algorithm mandatory; **test only symptomatic patients with unformed stool**; no test of cure
PPI association	Causal relationship described as "possible"	Association sustained; **PPI deprescribing is now a standard part of treatment**

Indian practical reality

Fidaxomicin is expensive and of limited availability in India. **Oral vancomycin 125 mg QID for 10 days remains the realistic first-line choice** in most Indian hospitals, with metronidazole reserved for where vancomycin is unavailable. Standardised FMT services and licensed live biotherapeutics are not routinely accessible. Say this explicitly — it demonstrates applied judgement rather than recitation.

Examiner's Tip

- "The three cornerstones are **stop the offending antibiotic, stop the PPI, and start targeted therapy**."
- "Severe disease is defined by a **WBC above 15,000/µl or creatinine at or above 1.5 mg/dl**."
- "**Alcohol hand rub does not kill spores** — soap and water handwashing is mandatory."
- "Cessation of diarrhoea with abdominal distension indicates **ileus or toxic megacolon**, not recovery."
- "Fidaxomicin is preferred over vancomycin because of lower recurrence, with a number needed to treat of 10."

Q5. Vaccines / Vaccinations for the Elderly

*15-mark answer · 19 high-yield points · **Recurring topic***

Rationale

1. **Immunosenescence** — thymic hypoplasia with reduced T-cell production and function, reduced mucosal barrier function, and preserved B-cell numbers — means **adaptive immunity is greatly affected while innate immunity is little affected**. This increases both infection and malignancy in the elderly.
2. **Inflammageing** compounds this with an excessive proinflammatory state, and vaccine responses are correspondingly weaker in older than in younger adults, which is why **higher-antigen, adjuvanted and conjugate formulations** have been developed for this age group.
3. Vaccination is **the single most cost-effective preventive intervention in geriatric medicine**, and belongs in every comprehensive geriatric assessment. Pathy's assessment schedule asks directly: "Have you been vaccinated for flu, pneumonia and tetanus?"

Influenza Vaccine

4. **Annually, for patients in all categories** — robust, frail, demented and end-of-life. **More than 90% of deaths attributed to pneumonia and influenza during epidemics occur in people aged 65 and over.**
5. It reduces hospitalisation rates, is cost-effective, and reduces influenza-associated mortality. In the nursing home it is **only about 40% effective at preventing clinical illness but more effective at preventing pneumonia, hospitalisation and death**; **vaccinating more than 80% of residents prevents outbreaks**.
6. Pathy's also records reductions of about **50% in influenza risk and 70% in influenza mortality**, and recommends **adjuvanted vaccine for the institutionalised and chronically ill**, plus **annual vaccination of healthcare personnel, family and regular visitors**.

Pneumococcal Vaccine

7. Although less effective at producing protective antibody in older than younger adults, it still **reduces pneumococcal bacteraemia in high-risk patients**. Textbook position: **one dose of PCV13 followed by PPSV23 one year later after age 65**; if PPSV23 was given first, only PCV13 is needed; no recommendations exist on re-immunisation.
8. Supporting trial evidence quoted in Pathy's: the **CAPiTA** randomised trial in community-dwelling adults over 65 found PCV13 reduced pneumococcal pneumonia by **45%**, and a large Japanese nursing-home randomised trial found PPSV23 markedly reduced pneumonia incidence and mortality.

Herpes Zoster Vaccine

9. Zoster occurs in up to half of individuals aged 85 or older and PHN affects more than 40% of those over 60. **Zostavax (2006) is only 30% protective; Shingrix (2016) is 90% protective.**
10. **ACIP recommends a two-dose Shingrix series for all adults aged 50 and above, regardless of prior chickenpox, and including those who previously received Zostavax.**

Tetanus, Diphtheria and Pertussis

11. **Over half of all tetanus cases occur in people aged 60 or older.** All adults should complete a primary series of Td — 0.5 ml intramuscularly, repeated at 2 and 6 months — if the history is incomplete or uncertain.
12. **Booster every 10 years**, with **at least one booster given as Tdap** to cover pertussis, because pertussis immunity wanes. In tetanus-prone wounds, boost every 5 years.

Other Vaccines and Screening in the Elderly

13. **SARS-CoV-2 vaccination** according to current guidelines. Pathy's records that safe and effective vaccines available since January 2021 reduce severe disease and mortality, and are indicated for adults over 65, healthcare professionals and family contacts.
14. **Hepatitis C:** not a vaccine, but the CDC recommends **one-time anti-HCV antibody testing for all adults born 1945–1965** — up to 85% are asymptomatic and at least 50% have normal liver function tests.
15. **Hepatitis B** for those with diabetes, chronic kidney or liver disease, or on haemodialysis; **travel vaccines** as indicated by destination.

Special Considerations in Long-Term Care

16. **All residents and staff with close resident contact should be vaccinated annually against influenza.** Zoster vaccination once for selected residents; pneumococcal vaccine once; tetanus booster every 10 years, or every 5 years with tetanus-prone wounds.
17. **Outbreak control:** at the onset of a suspected respiratory viral outbreak, obtain nasopharyngeal wash or swab for virus isolation and rapid influenza testing. During a confirmed influenza outbreak, give **oseltamivir or zanamivir within 24–48 hours to residents and staff throughout the outbreak**, vaccinate unvaccinated residents and staff,

restrict visits and movement, suspend group activities, reinforce hand hygiene and mask symptomatic individuals.

18. **Frailty-tailored approach:** Pathy's stratifies preventive care into **robust, frail, moderately demented and end-of-life** categories. Influenza vaccine is given yearly in **all four**; pneumococcal vaccine once, or considered once at end of life; tetanus primary series is not pursued at end of life.
19. **Contraindications and cautions:** live vaccines (Zostavax, MMR, yellow fever) are contraindicated in significant immunosuppression; acute febrile illness defers vaccination; anaphylaxis to a previous dose or component is an absolute contraindication. **Age itself is never a contraindication.**

Latest Updates (2023–Present)

Pneumococcal — the schedule has changed substantially

- **PCV21 (Capvaxive) was FDA-approved in June 2024** and recommended by ACIP as an option wherever PCV is indicated in adults. It covers up to **84–86% of invasive pneumococcal disease** in older adults versus roughly 58–59% for PCV20, but notably **does not cover serotype 4**.
- **In October 2024 ACIP lowered the age for routine pneumococcal conjugate vaccination from 65 to 50 years.**
- Current schedule for a PCV-naive adult aged 50 or above: **a single dose of PCV20 or PCV21 alone**, or **PCV15 followed by PPSV23 at least 1 year later** (minimum 8 weeks if immunocompromised, cochlear implant or CSF leak). **PPSV23 is not required after PCV20 or PCV21.** Conjugate vaccines are preferred because they trigger memory B-cell production and greater long-term immunity. Higher-valency (24- and 31-valent) vaccines are in advanced development.

RSV — an entirely new vaccine category

- Three RSV vaccines are licensed for adults aged 50 and above: **Arexvy (GSK), Abrysvo (Pfizer) and mResvia (Moderna)**, with no stated preference between them.
- CDC recommends **a single dose for all adults aged 75 and above**, and for **adults aged 50–74 at increased risk** — ACIP lowered this risk-based threshold from 60 to 50 years in April 2025. Risk conditions include COPD, asthma, cardiovascular disease, heart failure, diabetes, immunocompromise, frailty, and residence in a long-term care facility.
- **It is currently a one-time dose, not annual**, and is best given in late summer or early autumn. Guillain-Barré syndrome risk with the subunit vaccines was reviewed by ACIP and remains under surveillance.

Zoster

- **Zostavax has been discontinued in the US since 2020** and RZV is the only zoster vaccine in use. Beyond zoster prevention, a 2025 *Nature* natural experiment in Wales showed roughly a **20% reduction in dementia incidence**, replicated in *JAMA* (2025), *Nature Medicine* (2024) and multiple large cohorts. The **DAN-ZOSTER** randomised trial (started 2026, completion 2029) is testing this prospectively. *Results awaited.*

Influenza

- ACIP has since 2022 **preferentially recommended high-dose, adjuvanted or recombinant vaccines for adults aged 65 and above** over standard-dose vaccine.
- **DANFLU-2** (*NEJM*, 2025), the largest individually randomised influenza vaccine trial ever conducted with **332,438 participants**, found high-dose vaccine did **not significantly reduce the primary endpoint** of influenza or pneumonia hospitalisation, but pooled with the harmonised **GALFLU** trial (466,320 randomised in total) showed significant reductions in influenza or pneumonia, cardiorespiratory, laboratory-confirmed influenza and all-cause hospitalisation. See Q6 for detail.

Indian context

- The **API Indian Consensus Guidelines on Adult Immunization** were released in 2024 by the Association of Physicians of India with 13 collaborating societies, and updated in 2026 — the first unified Indian adult schedule, covering influenza, pneumococcal, hepatitis B and herpes zoster with emphasis on the elderly and those with chronic disease. India's population aged 50 and above is projected to reach about 23% within the next decade.
- **Coverage remains the central problem.** A 2024 *Indian Journal of Medical Research* review reports **under 2–3% adult vaccination coverage** for most vaccines. A 2026 *Journal of the Indian Academy of Geriatrics* position statement on vaccination for older adults in India identifies the barriers as **absence of a national adult immunisation schedule, low awareness, cost, accessibility and ageism**, and proposes evidence-based recommendations for those aged 60 and above.
- **India lacks a national adult immunisation programme** equivalent to the paediatric Universal Immunisation Programme. **NPHCE does not currently fund adult vaccines**, so all vaccination is out-of-pocket in the private sector.

Current Practice Update

Vaccine	Textbook Position	Current Recommendation (2024–2026)
Pneumococcal	PCV13 then PPSV23 one year later, from age 65; no re-immunisation guidance	**From age 50**: single dose PCV20 **or PCV21** alone, or PCV15 then PPSV23 ≥1 year later. **PCV13 superseded**
Zoster	Zostavax (30%) or Shingrix (90%), from age 50	**RZV only**; Zostavax withdrawn. Emerging **dementia-prevention signal** (~20%, *Nature* 2025)
Influenza	Annual standard vaccine; adjuvanted for institutionalised/chronically ill	**High-dose, adjuvanted or recombinant preferred for all aged 65+** (ACIP since 2022)

RSV	Not covered	**New category.** Single dose for all aged 75+, and 50–74 if at risk. Not annual
COVID-19	Vaccinate adults over 65 and contacts per current guidance	US policy shifted in 2025 toward **shared clinical decision-making** for older adults
Tetanus/Tdap	Td every 10 years, at least one dose as Tdap	**Unchanged**
Indian schedule	Not addressed	**API Consensus Guidelines 2024** (updated 2026); Indian Academy of Geriatrics position statement 2026; **coverage under 3%**

Extra Points to Remember — recurring topic

1. The four-vaccine minimum for every Indian patient above 60: annual influenza, pneumococcal conjugate, herpes zoster (two doses), and Td/Tdap every 10 years. Add RSV where available and affordable, and COVID-19 per current guidance.

2. Conjugate before polysaccharide, always. Giving PCV first produces a better antibody response to shared serotypes. If PPSV23 was inadvertently given first, give a PCV at least 1 year later.

3. Co-administration is safe. Influenza, pneumococcal, RSV and zoster vaccines may be given at the same visit at separate sites — use every clinical contact, because a missed opportunity in a frail older adult is rarely recovered.

4. The healthcare-worker argument. Vaccinating staff in long-term care reduces respiratory infection and overall mortality among residents even where resident coverage is already high — a strong point to raise in a policy-flavoured question.

5. Vaccine efficacy versus effectiveness. Efficacy falls with age, but **absolute benefit rises** because baseline risk is so much higher. Never use "the vaccine works less well in the elderly" as an argument against vaccinating them.

6. Live vaccines are the ones to be careful with. Zostavax, MMR and yellow fever are contraindicated in significant immunosuppression. Shingrix, being non-live, is safe and is the reason immunocompromised adults can now be protected against zoster.

7. Frailty stratification matters. In the end-of-life category, influenza vaccine is still given yearly for symptom burden, pneumococcal vaccine may be considered, but the tetanus primary series is not initiated. Frame this as goal-concordant care.

8. The Indian policy gap is a ready-made discussion point. India has a world-class paediatric Universal Immunisation Programme but **no national adult immunisation schedule**; NPHCE does not fund adult vaccines; coverage is under 3%. Recommending integration of adult immunisation into NPHCE is a high-scoring closing sentence.

Examiner's Tip — verbatim lines to write

• "Immunosenescence affects **adaptive immunity far more than innate immunity**, which is why conjugate, adjuvanted and high-dose formulations were developed for older adults."
• "More than **90% of pneumonia and influenza deaths during epidemics occur in those aged 65 and over**."
• "Vaccinating more than 80% of nursing home residents prevents influenza outbreaks."
• "ACIP **lowered the age for routine pneumococcal conjugate vaccination from 65 to 50 in October 2024**, and a single dose of PCV20 or PCV21 now suffices without PPSV23."
• "RSV vaccination is a new category — a **single, non-annual dose** for all adults aged 75 and above."
• Close with: "In India the principal barrier is not evidence but implementation — adult vaccination coverage is under 3%, and **integrating adult immunisation into NPHCE** would be the single highest-yield policy intervention."
• Draw a two-column table: **Vaccine | Schedule in the elderly**. It is the fastest way to earn structural marks.

Q6. Influenza Vaccination

7-mark short note · 10 high-yield points

High-Yield Points

1. **Burden:** influenza causes an enormous burden in the elderly. **More than 90% of deaths attributed to pneumonia and influenza during epidemics occur in people aged 65 and over**, and US hospitalisation rates reach **1195 per 100,000 in those over 85**. Global estimates are 291,243–645,832 influenza deaths annually.
2. **Definition of influenza-like illness (WHO):** an acute respiratory infection with onset within the last 10 days, **measured fever of at least 38°C, and cough**. Other viruses causing ILI include RSV, parainfluenza, adenovirus, rhinovirus, human coronaviruses and metapneumovirus; **RSV causes morbidity and mortality similar to influenza** in hospitalised older adults.
3. **Indication and timing: annual vaccination for everyone aged 65 and above**, given in **early autumn** before the season. Pathy's specifies it should be done yearly for patients in **all frailty categories** — robust, frail, moderately demented and end-of-life.
4. **Benefit:** it reduces influenza risk by about **50%** and influenza mortality by about **70%**. In the community, vaccination over 10 seasons was associated with reduced hospitalisation for pneumonia or influenza (**hazard ratio 0.75**) and reduced death (**hazard ratio 0.52**).
5. **Nursing-home specifics:** effectiveness is **only about 40% against clinical illness but higher against pneumonia, hospitalisation and death**, and **vaccinating more than 80% of residents prevents outbreaks**.
6. **Formulations:** the **adjuvanted vaccine** contains a substance that enhances immunogenicity and is **particularly valuable in institutionalised elderly or those weakened by chronic disease** such as diabetes, heart failure or respiratory failure. High-dose and recombinant formulations serve the same purpose of overcoming immunosenescence.
7. **Vaccinate the ring around the patient: annual vaccination of healthcare personnel in long-term care reduces respiratory infection and overall mortality among residents, even where resident coverage is already high**. Employers should offer free easy-access vaccination, and the same protection is logically recommended for regular visitors, family members and volunteers.
8. **Safety:** the vaccine is **well tolerated and rarely causes minor local reactions and systemic reactions**. It is an inactivated vaccine, therefore **safe in immunocompromised older adults**. The only absolute contraindication is anaphylaxis to a previous dose or component; acute febrile illness defers vaccination. **Age is not a contraindication.**
9. **Outbreak management in an institution:** confirm the virus by nasopharyngeal swab; give **neuraminidase inhibitors (oseltamivir preferred, within 24–48 hours) to both cases and contacts** to stop the outbreak; vaccinate unvaccinated residents and staff; restrict visits and movement; suspend group activities; reinforce hand hygiene; mask symptomatic residents and staff. **Oral oseltamivir is preferred over inhaled zanamivir** in elderly patients because administration is easier; reduce the dose if creatinine clearance is below 30 ml/min.
10. **Vaccine remains protective even when breakthrough occurs** — influenza pneumonia can still occur in vaccinated patients, so vaccination status must never be used to exclude the diagnosis.

Latest Updates (2023–Present) — three points

- **Enhanced vaccines are now preferentially recommended.** Since 2022 **ACIP has preferentially recommended high-dose, adjuvanted or recombinant influenza vaccine over standard-dose vaccine for all adults aged 65 and above**. A 2026 test-negative design study in *Open Forum Infectious Diseases* found **comparable effectiveness between adjuvanted and high-dose quadrivalent vaccines**, so either is acceptable — the recommendation is for an enhanced vaccine, not for one specific brand.
- **DANFLU-2 — the definitive trial, with a nuanced result.** Published in *NEJM* (2025), this pragmatic registry-based randomised trial of **332,438 Danish adults aged 65 and above** across three seasons is the largest individually randomised influenza vaccine trial ever conducted. High-dose vaccine **did not significantly reduce the primary endpoint** of hospitalisation for influenza or pneumonia. However, prespecified secondary analyses showed lower cardiorespiratory hospitalisation (2.25% vs 2.38%), driven by cardiovascular hospitalisation (1.30% vs 1.40%), and reduced myocarditis and pericarditis (relative effectiveness 45.7%). Benefit was consistent irrespective of diabetes status (*JAMA Internal Medicine*, 2026) and atrial fibrillation (*JACC*, 2025).
- **Pooled and meta-analytic data favour high-dose.** Pooled with the methodologically harmonised **GALFLU** trial from Galicia, Spain — **466,320 individually randomised older adults in total** — there were significant reductions in hospitalisation for influenza or pneumonia, cardiorespiratory illness, laboratory-confirmed influenza and all causes. A 2026 systematic review and meta-analysis in *JAMA Network Open* of 8 trials and 605,098 participants confirmed **high-dose vaccine reduced hospitalisations for influenza, laboratory-confirmed influenza, pneumonia or influenza, cardiorespiratory disease and all causes** compared with standard dose.

Current Practice Update

Aspect	Textbook Position	Current Evidence (2023–2026)
Which formulation	Standard vaccine; adjuvanted particularly	**High-dose, adjuvanted or recombinant**

	valuable in institutionalised and chronically ill	**preferentially recommended for all aged 65+** (ACIP since 2022); adjuvanted and high-dose comparable
Strength of evidence for high-dose	Not addressed	DANFLU-2 **neutral on primary endpoint**, but pooled DANFLU-2 + GALFLU (466,320) and 2026 meta-analysis (605,098) **favour high-dose**
Cardiovascular benefit	Not described	Signal for reduced **cardiovascular hospitalisation, myocarditis and pericarditis** — exploratory, from a neutral primary trial
Availability in India	Not addressed	Standard quadrivalent widely available; **high-dose and adjuvanted formulations largely unavailable** — standard-dose annual vaccination remains the practical recommendation

Examiner's Tip

• "More than **90% of pneumonia and influenza deaths during epidemics occur in those aged 65 and over**."
• "In nursing homes the vaccine is only about **40% effective against clinical illness but far more effective against pneumonia, hospitalisation and death**."
• "**Vaccinating over 80% of nursing home residents prevents outbreaks**, and vaccinating staff independently reduces resident mortality."
• The distinction line: "**DANFLU-2**, the largest randomised influenza vaccine trial ever conducted, was neutral on its primary endpoint, but pooled with GALFLU and in 2026 meta-analysis, high-dose vaccine reduced influenza, cardiorespiratory and all-cause hospitalisation."
• Close with the practical point: "In India, where enhanced formulations are largely unavailable, the priority is not which vaccine but **achieving any vaccination at all**, given coverage under 3%."

Rapid Revision Grid

Numbers, criteria and named evidence — the last-hour page

Definitions and Diagnostic Criteria

Entity	Criterion
Classical FUO (Petersdorf/Beeson 1961)	>38.3°C on several occasions, >3 weeks, undiagnosed after 1 week inpatient work-up
FUO (Durack/Street 1991) — current	>38.3°C, >3 weeks, undiagnosed after 3 days inpatient or 3 outpatient visits
IUO	Prolonged raised acute-phase reactants **without** fever
Fever in long-term care (elderly)	Single oral >100°F, **or** repeated oral >99°F, **or** rise >2°F above baseline
WHO influenza-like illness	Acute respiratory infection, onset <10 days, measured fever ≥38°C, and cough
Postherpetic neuralgia	Pain persisting ≥90 days after rash onset
Disseminated zoster	>20 vesicles outside the primary dermatome
Severe *C. difficile* infection	WBC >15,000 cells/µl **or** serum creatinine ≥1.5 mg/dl
Fulminant *C. difficile*	Severe disease plus hypotension, shock, ileus or megacolon
Diarrhoea	≥3 unformed stools in 24 hours
Infection suspicion in LTC	WBC >14,000 cells/mm³ **or** left shift >6%

Key Numbers

Fact	Figure
Fever absent in older adults with serious infection	**30–50%** (>50% in LTC residents)
Fever and cough absent in elderly pneumonia	**30–40%**
Zoster incidence, age >60	**11.8 per 1000 per year** (all ages 1.2–3.4)
Zoster incidence, India (ICMR)	**~3.3 per 1000 per year**; >90% VZV-seropositive by age 40
Lifetime zoster risk by age 85	**Up to 50%**
PHN in those >60 with zoster	**>40%**
Shingrix vs Zostavax efficacy	**90% vs 30%**
Antiviral window in zoster	**72 hours** from rash onset
C. difficile recurrence after first episode	**15–25%** (40–60% thereafter)
Fidaxomicin NNT vs vancomycin (initial episode)	**10** at 4 weeks; **7** at 30 days for recurrence
Influenza deaths in those ≥65 during epidemics	**>90%** of all pneumonia/influenza deaths
Influenza vaccine benefit	**~50%** risk reduction; **~70%** mortality reduction
Influenza vaccine effectiveness in nursing homes	**~40%** against clinical illness; higher against death
Nursing-home coverage preventing outbreaks	**>80%** of residents
PCV21 vs PCV20 IPD serotype coverage in adults	**~84–86% vs ~58–59%**
Indian adult vaccination coverage	**<2–3%** for most vaccines
FDG PET-CT in over-75s with FUO/IUO	Diagnosis in **30.8%**; treatment changed **38.8%**; false positive **15.2%**

Named Trials and Guidelines

Name	What it showed / what it changed
DANFLU-2 (*NEJM* 2025)	332,438 adults ≥65; high-dose vs standard influenza vaccine. **Neutral on primary endpoint** (influenza/pneumonia hospitalisation); secondary reductions in cardiorespiratory and cardiovascular hospitalisation
GALFLU (*NEJM* 2025)	Galicia, Spain; harmonised with DANFLU-2. Pooled total **466,320** randomised — significant reductions in influenza/pneumonia, cardiorespiratory, lab-confirmed influenza and all-cause hospitalisation

DANFLU-1	Pilot trial; post-hoc analyses by frailty score and atrial fibrillation (2025)
CAPiTA	PCV13 in community-dwelling adults >65 reduced pneumococcal pneumonia by **45%**
Shingles Prevention Study (Oxman, *NEJM* 2005)	Original Zostavax trial; incidence of zoster in >60s 11.8/1000/year
Nature 2025 Welsh natural experiment (Eyting et al.)	Herpes zoster vaccination reduced dementia incidence by **~20%** over 7 years — strongest causal evidence
JAMA 2025 (Pomirchy et al.)	Australian natural experiment replicating the dementia finding
Nature Medicine 2024 (Taquet et al.)	RZV vs live vaccine: **17% increase in dementia diagnosis-free time** over 6 years
DAN-ZOSTER	Pragmatic RCT of RZV for MACE and dementia in adults ≥65; started 2026, completion 2029. **Results awaited**
IDSA/SHEA 2021 focused update	Fidaxomicin first-line for initial and recurrent *C. difficile*; metronidazole not preferred
ACG 2021 / AGA 2024	ACG guideline on *C. difficile*; AGA restricts FMT to adjunct or last resort
ACIP October 2024	Routine pneumococcal conjugate vaccination age **lowered from 65 to 50**
ACIP April 2025	RSV vaccination risk-based age **lowered from 60 to 50**
API Indian Consensus Guidelines 2024 (updated 2026)	First unified Indian adult immunisation schedule; 13 collaborating societies
JIAG position statement 2026	Indian Academy of Geriatrics recommendations for vaccination in adults ≥60

Drug Doses to Quote

Indication	Regimen
Herpes zoster — valaciclovir	1 g three times daily × 7 days
Herpes zoster — famciclovir	500 mg three times daily × 7 days
Herpes zoster — aciclovir	800 mg five times daily × 7–10 days (reduce if CrCl <30 ml/min)
Severe/disseminated zoster	IV aciclovir 10 mg/kg 8-hourly
Established PHN	Topical lidocaine patch, gabapentin or pregabalin (FDA-approved); nortriptyline or opioids as alternatives
C. difficile — initial episode	Fidaxomicin 200 mg BD × 10 days, **or** oral vancomycin 125 mg QID × 10 days
C. difficile — fulminant	Oral vancomycin 500 mg QID (+ PR if ileus) **plus** IV metronidazole 500 mg 8-hourly
Influenza outbreak in institution	Oseltamivir within 24–48 h to cases and contacts throughout outbreak; reduce dose if CrCl <30 ml/min
Tetanus primary series (adult)	Td 0.5 ml IM, repeated at 2 and 6 months; booster every 10 years, at least one as Tdap
Zoster vaccine (RZV)	Two doses IM, 2–6 months apart, from age 50
Pneumococcal, PCV-naive adult ≥50	Single PCV20 or PCV21 alone; **or** PCV15 then PPSV23 ≥1 year later

The five statements most likely to earn or lose you marks

1. Fever in the elderly is defined at a lower threshold and against the patient's own baseline — not 38.3°C.
2. Corticosteroids do **not** reduce the incidence of postherpetic neuralgia.
3. Alcohol hand rub does **not** kill *C. difficile* spores.
4. Asymptomatic bacteriuria in older adults should **not** be tested for or treated in the absence of obstructive uropathy.
5. ACIP has lowered **both** pneumococcal (Oct 2024) and risk-based RSV (Apr 2025) vaccination ages to 50 — the schedule you memorised from the textbook is out of date.

Geriatric Pharmacotherapy & Perioperative Medicine

MD Geriatric Medicine — Structured Examination Answers

Five questions • 15-mark and 7-mark formats

Primary sources: *Pathy's Principles and Practice of Geriatric Medicine* (6th ed.) and *Essentials of Clinical Geriatrics* (7th ed.)

Supplemented with evidence and guidelines to 2026

Contents

Q	Topic	Format
1	Principles of drug therapy in the elderly	15 marks (+ Extra points)
2	Rational drug prescribing in older adults	15 marks
3	Polypharmacy — definition and prevention	15 marks (+ Extra points)
4	Drug holiday	7 marks (short note)
5	Perioperative assessment and management in older people	15 marks (+ Extra points)

Each answer follows the same architecture: high-yield numbered points from the textbooks → **Latest Updates (2023–present)** with inline citations → **Current Practice Update** comparison table → **Examiner's Tip**. Recurring topics carry an additional **Extra Points to Remember** panel. A Rapid Revision Grid appears at the end.

Question 1. Principles of drug therapy in the elderly 15 MARKS

A. Why drug therapy is different in old age

1. **Definition of the problem.** Drug therapy in old age is the art of matching a drug to a body whose **handling of and response to drugs has changed**, in a patient who usually has several diseases and takes several drugs at once.
2. **Scale of the problem.** Older people are the largest consumers of drugs. **Adverse drug reactions (ADRs) account for a large share of emergency hospital admissions** in this age group and are largely preventable (Pathy, Ch. 10).
3. **Frail elders are excluded from trials.** Frail older people are rarely included in large drug trials, so results from younger patients are often **wrongly extrapolated** to them (Pathy, Ch. 11).
4. **The therapeutic window narrows with age.** The gap between a therapeutic dose and a toxic dose becomes smaller, so the more aggressive the treatment, the greater the chance of harm (Essentials, Fig. 5-2).
5. **Non-pharmacological factors matter more than pharmacology.** Most of the steps that make drug therapy safe — correct diagnosis, correct choice, correct taking of the drug — come **before** pharmacokinetics ever apply (Essentials, Fig. 14-1).

B. Age-related pharmacokinetic changes (ADME)

6. **Absorption — least affected.** Splanchnic blood flow, absorptive surface and motility fall and gastric pH rises, but **no clinically important change in absorption occurs** unless there is gut disease (e.g. diabetic gastroparesis).
7. **Distribution — protein binding.** Serum albumin falls in illness and malnutrition (a **negative acute-phase reactant**); **highly protein-bound drugs** therefore have a higher free (active) fraction. Reported "therapeutic ranges" were defined in younger subjects and may mislead.
8. **Distribution — volume of distribution (Vd).**
   - ↓ Total body water and lean mass → **↓ Vd for water-soluble drugs** → higher plasma levels, toxicity with **early** doses: *digoxin, aminoglycosides, atenolol, sotalol, theophylline, hydrochlorothiazide, lithium, alcohol.*
   - ↑ Total body fat → **↑ Vd for fat-soluble drugs** → prolonged half-life, toxicity **after steady state**: *amiodarone, diazepam, desipramine, haloperidol.*
9. **Metabolism.** Liver blood flow, enzyme activity and enzyme inducibility all fall. **Phase I reactions (oxidation, reduction, hydrolysis) decline** — more so in men; **Phase II (conjugation, acetylation, glucuronidation) is relatively preserved**. First-pass metabolism is reduced. Normal liver function tests do **not** mean normal drug metabolism.
10. **Excretion — the most predictable change.** Renal blood flow, GFR and tubular secretion all decline. Renally cleared drugs accumulate, with longer half-lives and higher steady-state levels.
11. **Serum creatinine is misleading.** Muscle mass falls with age, so creatinine production falls; **a "normal" creatinine may hide substantially reduced renal function**. Estimate clearance (Cockcroft–Gault / eGFR), but remember there is **wide interindividual variation** — average declines must not be applied blindly to an individual.

C. Pharmacodynamic changes (tissue sensitivity)

12. **Sensitivity may rise or fall.** Receptor number and affinity, second-messenger function and nuclear responses all alter. Older patients are **more sensitive** to benzodiazepines, opioids, anticholinergics and other psychotropics, but **less sensitive** to beta-adrenergic agonists and antagonists.
13. **Reduced homeostatic reserve.** Blunted baroreflex, thermoregulation and thirst mean drugs more readily cause **postural hypotension, falls, hypothermia and dehydration**.

D. Practical prescribing principles

14. **Make the diagnosis first.** Symptoms are under-reported, vague, multiple and often atypical; treating a symptom with a drug instead of evaluating it is the commonest route into polypharmacy.
15. **"Start low and go slow" — but do not under-treat.** Begin with the lowest effective dose of the most cost-effective agent and titrate slowly; still increase to the maximum appropriate dose if the response requires it.
16. **One drug at a time; simplify.** Use as few drugs as possible, prefer **once-daily dosing**, keep all drugs on the same schedule and tie doses to a daily routine.
17. **Treat every new symptom as a possible ADR.** Never treat a symptom with a new drug before considering stopping the offending one — this avoids the **prescribing cascade**.
18. **Consider drug–disease as well as drug–drug interactions** (Essentials, Table 14-4): diuretics → incontinence and dehydration; beta-blockers → masked hypoglycaemia, bronchospasm; anticholinergics → retention, glaucoma, constipation; NSAIDs → heart failure; antipsychotics → worsening parkinsonism.
19. **Support adherence actively.** Barriers include poor vision and hearing, low literacy, memory impairment, transport, cost, and child-proof containers. Use pill boxes and drug calendars, involve family and pharmacists, and **reconcile medicines at every hospital discharge**.

20. **Review, record and prune.** Keep an updated medication record reviewed at each visit; ask patients to bring all bottles ("brown-bag" review); **each time a drug is added, ask whether one can be stopped**. **Never be the first to use a new drug in an older person, and never the last to abandon an old unsafe one.**

Latest Updates (2023–Present)

- **AGS Beers Criteria 2023 update** (*J Am Geriatr Soc*, 2023) reviewed evidence from 2017–2022 and retained the five-table structure. Headline changes: **aspirin for primary prevention** moved to "avoid — consider deprescribing"; **warfarin** to be avoided as initial therapy for VTE/non-valvular AF unless a DOAC is contraindicated; **rivaroxaban** to be avoided for long-term AF/VTE because of bleeding; **low-dose doxepin** removed as a PIM; new caution entries including SGLT2 inhibitors.
- **AGS Beers Alternatives panel** (*J Am Geriatr Soc*, 2025) published, for the first time, a companion list of **safer pharmacological and non-pharmacological alternatives across 21 common conditions** — e.g. CBT-I for insomnia, exercise for Parkinson's disease, dietary measures for constipation and reflux.
- **WHO "Medication Without Harm"**, the Third Global Patient Safety Challenge, continues to name **polypharmacy in older people** as one of its three priority action areas.
- Deprescribing is now regarded as an integral part of prescribing rather than an optional extra (*Annual Review of Medicine*, 2024).

Current Practice Update

Textbook position (Pathy 6th ed. / Essentials 7th ed.)	Current evidence and practice (2023–2026)
Beers Criteria cited as the **2019** version with 88 potentially inappropriate medications.	**2023 AGS update** is current; aspirin for primary prevention and rivaroxaban moved to "avoid"; doxepin at very low dose removed; SGLT2 inhibitors added with caution.
Anticoagulation in older people framed largely around warfarin and INR control.	**DOACs are first-line**; warfarin only where DOACs are contraindicated (Beers 2023). Relevant to Indian practice where INR monitoring access is variable.
Beers criteria do not suggest safer treatment alternatives.	The **2025 Beers Alternatives** document explicitly supplies pharmacological *and* non-pharmacological substitutes.
Creatinine clearance estimated by Cockcroft–Gault or MDRD.	**CKD-EPI 2021 (race-free)** is now the standard eGFR equation; Cockcroft–Gault is still preferred for **drug dosing** of narrow-therapeutic-index agents such as digoxin and DOACs.

Examiner's Tip

Earn marks with these verbatim lines:

- "Absorption is the pharmacological parameter **least affected** by ageing; **renal excretion is the most predictable change**."
- "A normal serum creatinine in an older person does **not** mean normal renal function, because creatinine production falls with declining muscle mass."
- "Phase I metabolism declines with age; **Phase II conjugation is relatively preserved** — hence lorazepam, oxazepam and temazepam are preferred benzodiazepines."
- "Consider **every new symptom in an older patient as a possible adverse drug reaction** until proved otherwise."
- Close with the aphorism: "*Never be the first to use a new drug in an older person, and never the last to abandon an old, unsafe one.*"

Extra Points to Remember (recurring topic)

- **Water-soluble vs fat-soluble mnemonic:** water-soluble drugs cause **early** toxicity (high peak level); fat-soluble drugs cause **late** toxicity (accumulation at steady state).
- **Indian context:** free over-the-counter availability of NSAIDs, benzodiazepines and antibiotics; concurrent AYUSH and traditional preparations; multiple prescribers without a shared record; and out-of-pocket cost driving intermittent dosing. A "brown-bag" review at every visit is even more valuable here. There is no validated India-specific PIM list in routine use, so **Beers and STOPP/START are applied with local adaptation**.
- **Drugs needing dose reduction by renal function:** digoxin, metformin, DOACs, aminoglycosides, gabapentin/pregabalin, allopurinol, sulphonylureas (glibenclamide should be avoided altogether).
- **Anticholinergic burden** is measured by the **Drug Burden Index** or the Anticholinergic Cognitive Burden scale; a high burden predicts functional decline and falls.
- **Time-to-benefit** is an essential concept: a preventive drug whose benefit takes 3–5 years to appear is inappropriate for a patient whose life expectancy is shorter.

Question 2. Rational drug prescribing in older adults 15 MARKS

A. Concept and definition

1. **Definition.** Rational prescribing means giving the **right drug, in the right dose, by the right route, for the right duration, to the right patient, at the lowest cost**, with an explicit indication and a plan for review.
2. **Two errors, not one.** Inappropriate prescribing includes both **over-prescribing / mis-prescribing** (potentially inappropriate medications, PIMs) and **under-prescribing** (potential prescribing omissions, PPOs). Withholding a beneficial drug purely because of age is a form of **ageism**.
3. **Why it matters.** Inappropriate prescribing is associated with ADRs, falls, frailty, disability, hospitalisation and mortality, and should be assessed at **every single medical encounter** (Pathy, Ch. 10, Key Points).

B. The prescribing sequence

4. **Step 1 — Confirm the diagnosis.** Atypical presentation and symptom overlap make this the commonest failure point; treating an undiagnosed symptom is irrational by definition.
5. **Step 2 — Define the therapeutic goal with the patient.** Ask what matters to them. Frail elders commonly prioritise **function, cognition, independence and relief of suffering** over length of life.
6. **Step 3 — Try non-pharmacological treatment first** where evidence supports it (exercise, physiotherapy, sleep hygiene and CBT-I, dietary measures, compression stockings, continence training).
7. **Step 4 — Select the drug.** Before writing, ask:
   - **Efficacy** — will this make a real difference, and what is the evidence in *this* age group?
   - **Cost** — is there an equally effective cheaper option?
   - **Timing** — can a once-daily regimen be used?
   - **Dose** — what is the lowest effective dose here?
   - **Side effects** — how well will this person tolerate it?
   - **Interactions** — any drug–drug or drug–disease contraindication?
8. **Step 5 — Prescribe, educate, document**: one drug at a time, written instructions in large print, explanation to patient and caregiver, and an entry in an updated medication record.
9. **Step 6 — Monitor and review.** Set a review date and an explicit stopping rule; assess both efficacy and toxicity; **periodically deprescribe**.

C. Explicit and implicit prescribing tools (Pathy, Table 10.3)

Tool	Origin	What it does	Main limitation
Beers Criteria	USA, 1991; AGS steward since 2011	Explicit list of PIMs in 5 categories: PIMs in all older adults; PIMs with specific diseases; drugs to use with caution; harmful drug–drug combinations; renal dosing. Plus a table of strongly anticholinergic drugs.	Does not address **under-prescribing**; US-centric formulary; not for hospice/end-of-life care
STOPP / START	Europe, 2008	Organ-system based. **STOPP** = PIMs, **START** = prescribing omissions. Designed to be used together.	Needs fuller medical history; suggests no safer alternatives
Medication Appropriateness Index (MAI)	USA, 1992	10 implicit questions per drug (indication, effectiveness, dose, directions, practicality, drug–drug and drug–disease interaction, duplication, duration, cost); scored A/B/C, max 18.	~10 min per drug; needs expertise; does not prioritise which drug to stop
Pill Pruner	New Zealand, 2009	Pocket card of 13 commonly prescribed drug groups based on STOPP; reduces discharge medication counts, sustained at 90 days	Studied only in hospitalised patients ≥75; no tapering guidance
Drug Burden Index (DBI)	2007	Measures cumulative **anticholinergic and sedative** exposure; high DBI predicts functional decline and falls	Covers only two drug classes
TRIM	US VA, 2016	EHR-based algorithm using Beers + STOPP to flag PIMs and generate clinician feedback	Validated only in the US VA system
FORTA	Germany, 2008	First tool to give **positive and negative labels** together (A–D classification)	Requires clinical judgement of indication

D. Aids to memory in daily practice (Pathy, Table 10.4)

10. **SAIL:** keep it **S**imple — know **A**dverse effects — identify the **I**ndication — **L**ist every drug and dose.

TIDE: allot **T**ime at each visit for medicines — **I**ndividual response — avoid **D**rug/drug and drug/disease interactions — **E**ducate the patient.

11. **ARMOR: A**ssess → **R**eview pharmacokinetics/pharmacodynamics → **M**inimise → **O**ptimise doses → **R**eassess. Focused on function and quality of life; useful in post-acute and long-term care.
12. **ERASE: E**valuate diagnoses by considering **R**esolved conditions, **A**geing normally, and **S**electing appropriate targets, to **E**liminate unnecessary diagnoses — i.e. **"un-diagnosing to prevent over-prescribing"**.
13. **AVOID TOO MANY:** Alternatives, Vague symptoms, OTC drugs, Interactions, Duration, Therapeutic vs preventive, Once daily, Other doctors, Money, Adverse effects, Needs still?, Yes/no — is the patient actually taking it?

E. Barriers and system measures

14. **Treatment burden** — the effort the patient must expend to follow the regimen — is reduced by shared decision-making and by involving pharmacists, nurses and social workers.
15. **Prescribing inertia** (continuing a drug simply because it is on the list) and **multiple prescribers** are the two commonest system failures; medication reconciliation at every transition of care is the antidote.

Latest Updates (2023–Present)

- **STOPP/START version 3** (*European Geriatric Medicine*, 2023) is the current version: **190 criteria — 133 STOPP and 57 START**, a 66.7% increase over version 2's 114 criteria. Validated by an 11-member Delphi panel from 8 European countries. Criteria are now presented **by drug class rather than individual drug name** to improve international applicability. Roughly 41% of STOPP.v3 and 42% of START.v3 criteria are new.
- **AGS Beers Criteria 2023** remains the current US standard (see Q1 for key changes), with the **2025 Alternatives** companion supplying safer substitutes.
- **Trial evidence is mixed on hard outcomes.** Single-centre trials of STOPP/START showed reduced polypharmacy, ADRs, falls and cost; but the large multicentre **SENATOR**, **OPERAM** and **OPTICA** trials did not show significant reductions in ADRs, mortality, drug-related readmissions or quality of life (*Expert Review of Clinical Pharmacology*, 2023).
- **Usability work** has produced a validated 3-page single-entry table of STOPP/START v3 for bedside use (*European Geriatric Medicine*, 2025), addressing the criticism that 190 criteria are impractical in a busy clinic.
- **STOPPFrail** remains the tool of choice for frail patients with limited life expectancy, where standard criteria are inappropriate.

Current Practice Update

Textbook position	Current evidence and practice (2023–2026)
STOPP/START **version 2 (2015)**, 114 criteria (80 STOPP + 34 START).	**Version 3 (2023)**: **190 criteria (133 + 57)**, class-based rather than drug-based, with expanded cardiovascular, diabetes and COPD sections.
STOPP/START application within 72 hours of admission reduces ADRs by ~9% and length of stay by ~3 days.	These **single-centre findings were not replicated** in SENATOR, OPERAM or OPTICA. Present the tools as improving **prescribing quality**, with hard clinical endpoints still unproven.
Beers criteria give no alternatives.	**2025 AGS Alternatives** list now covers 21 conditions and emphasises non-pharmacological options.
Tools presented as broadly interchangeable.	Current practice is **tool-plus-judgement**: an explicit tool (Beers/STOPP) to screen, an implicit tool (MAI) to judge, and a patient-centred goals discussion to decide.

Examiner's Tip

- Open with the two-sided definition: "*Rational prescribing corrects both over-prescribing and under-prescribing — STOPP addresses the first, START the second.*"
- Quote the current numbers: **"STOPP/START version 3 (2023) contains 190 criteria — 133 STOPP and 57 START."**
- Name at least **four tools with their country and year**; this single sentence often separates a distinction answer.
- Add the honest caveat: "*These tools reliably improve prescribing quality; large multicentre trials have not yet shown a mortality benefit.*"
- Always mention **time-to-benefit** and **life expectancy** as determinants of rationality.

Question 3. What is polypharmacy and how would you prevent it? 15 MARKS

A. Definition

1. **No single agreed definition.** Polypharmacy has been defined in about **24 different ways**, which has made it hard to measure and to compare studies (Pathy, Ch. 10).
2. **Numerical definition.** The most widely used cut-off is **the concurrent use of five or more medications**; **hyperpolypharmacy** is ten or more. The five-drug threshold identifies community-dwelling older men at risk of adverse outcomes.
3. **Qualitative (and more accurate) definition.** The most precise definition is **"the use of multiple medications, and more than are medically necessary"**. This allows the essential distinction between **appropriate polypharmacy** (many drugs, all indicated, e.g. in heart failure) and **inappropriate polypharmacy**.
4. **Regulatory definition.** The **Omnibus Budget Reconciliation Act (OBRA) 1987** in the USA first brought polypharmacy in nursing homes under regulatory scrutiny.

B. Epidemiology

5. **Rising everywhere.** In Scotland, use of ≥5 drugs rose from ~11% to 20% between 1995 and 2010, and ≥10 drugs from ~1.5% to almost 6%; by 2010, **13% of older patients were exposed to a potentially serious drug interaction**.
6. **Comparable trends elsewhere:** Sweden 18% → 42% (1992–2002); Italy 43% → 53% (2000–2010).
7. **Indian context.** Polypharmacy in India is driven by multimorbidity in a rapidly ageing population (LASI), fragmented care across multiple prescribers, unrestricted OTC sales, concurrent AYUSH and home remedies, and the absence of a shared electronic medication record. **NPHCE** geriatric clinics are the logical place to embed structured medication review.

C. Causes

8. **Patient factors:** multimorbidity, chronic disease, under-reporting of symptoms, expectation of a prescription, OTC and supplement use.
9. **Prescriber factors:** treating symptoms rather than evaluating them, single-disease guideline adherence in a multimorbid patient, prescribing inertia, and failure to review.
10. **System factors:** multiple doctors each prescribing without a complete list, transitions of care, direct-to-consumer advertising, and pressure from patients or families.
11. **The prescribing cascade** — an ADR is misread as a new disease and treated with another drug — is the single most important self-perpetuating mechanism.

D. Consequences

12. **Clinical:** ADRs, drug–drug and drug–disease interactions (notably **CYP450-mediated**), **falls**, **frailty**, disability, cognitive impairment and delirium, malnutrition, urinary incontinence, sarcopenia and **increased mortality**.
13. **Practical:** reduced adherence, greater treatment burden, higher cost, more hospital admissions and readmissions, and more medication errors — the incidence of medication error in UK nursing home residents has been reported as high as **69.5%** (Pathy, Ch. 11).

E. Prevention — a stepwise approach

14. **Prevent at the point of prescribing.** Apply the rational prescribing sequence (Q2): correct diagnosis → non-drug options first → lowest effective dose → explicit review date. **Each time a drug is added, consider whether one can be stopped.**
15. **Obtain an accurate, complete medication list.** "Brown-bag" review at first assessment; include OTC, herbal and traditional preparations; maintain and update the record at every visit; **reconcile medicines at admission, transfer and discharge**.
16. **Screen with a validated tool** — Beers, STOPP/START, MAI, Pill Pruner, DBI or FORTA. Be proficient in at least one.
17. **Deprescribe systematically.** Deprescribing is **"a patient-centred, systematic optimisation of a person's medication list through the reduction, tapering or stopping of medications that are not indicated or are causing real or potential harm"** (Scott et al.).
    - **Scott's five-step protocol:** (i) compile a complete, accurate medication list; (ii) assess overall risk of drug-induced harm; (iii) determine each drug's discontinuation potential; (iv) prioritise drugs for discontinuation; (v) establish a monitoring plan.
    - **Barnett's seven steps** begin with the patient's needs and goals and end with a long-term communication and monitoring plan.
    - The **Deprescribing Rainbow** places the patient at the centre and considers clinical, psychological, social, financial

and physical determinants.

18. **Taper, do not stop abruptly**, for benzodiazepines, opioids, SSRIs, beta-blockers, corticosteroids, levodopa and antiepileptics — abrupt withdrawal causes **adverse drug withdrawal events**.
19. **Avoid under-prescribing at the same time.** Use START criteria so that deprescribing does not become age-based rationing; apply ERASE to remove unnecessary diagnoses.
20. **Work as a team and educate.** Clinical pharmacists, nurses, family caregivers and structured patient education are the interventions most consistently shown to work. Documented benefits of systematic deprescribing include **fewer falls, improved cognitive and psychomotor function, reduced mortality and reduced healthcare utilisation, without an increase in adverse outcomes**.

Latest Updates (2023–Present)

- **Deprescribing is safe and effective at reducing drug counts, but benefit on hard endpoints is unproven.** A meta-analysis of 32 RCTs including 18,670 patients aged ≥60 confirmed reductions in PIMs (*JAMDA*, 2023). An updated systematic review of 259 studies found **no significant reduction in mortality** from deprescribing polypharmacy (*British Journal of Clinical Pharmacology*, 2024).
- **Long-term care evidence.** A meta-analysis of 38 studies (22 RCTs) in long-term care facilities showed sustained reductions in the number of drugs per resident and in PIMs at ≥12 months (*Age and Ageing*, 2026).
- **Pharmacist-led review works for medication outcomes but not quality of life.** A cluster-randomised trial in patients ≥75 with hyperpolypharmacy found significantly more medications stopped or reduced with a deprescribing-focused clinical medication review (2.5 vs 1.7 per patient), but **no change in health problems or health-related quality of life at 6 months** (*Age and Ageing*, 2026).
- **Polypharmacy and sarcopenia.** A 2023 systematic review and meta-analysis confirmed an association between polypharmacy and sarcopenia, possibly mediated by malnutrition — strengthening the case for medication review as part of sarcopenia management.
- **Structural change.** WHO's **Medication Without Harm** challenge explicitly targets polypharmacy, and deprescribing is now embedded in national quality frameworks in several countries.

Current Practice Update

Textbook position	Current evidence and practice (2023–2026)
Systematic deprescribing is associated with **reduced mortality** and reduced healthcare utilisation.	Larger and more recent syntheses are **more cautious**: deprescribing reliably reduces drug counts and PIMs and is **safe**, but a 2024 review of 259 studies found **no significant mortality benefit**. Present it as safe and quality-improving rather than life-prolonging.
Screening tools quoted at 2015/2019 versions.	**STOPP/START v3 (2023)** and **Beers 2023** with the **2025 Alternatives** list are current.
Emphasis is on identifying and stopping inappropriate drugs.	Equal emphasis now on **implementation** — who does the review, how often, and how changes are communicated across transitions. Pharmacist-integrated workflows and patient activation are the components most associated with success.
Quality of life expected to improve after deprescribing.	Repeated trials show **no measurable change in HRQoL** at 6–12 months; existing QoL instruments may simply be insensitive to medication burden.

Examiner's Tip

- Never give only the numerical definition. Say: "*Polypharmacy has been defined in about 24 ways; the commonest numerical cut-off is five or more drugs, but the most precise definition is the use of more medications than are medically necessary.*"
- Make the **appropriate vs inappropriate polypharmacy** distinction explicitly — examiners look for it.
- Reproduce the **Scott five-step deprescribing protocol** as a numbered list; it is the single highest-yield block in this answer.
- Name the **prescribing cascade** and give one example (e.g. metoclopramide → parkinsonism → levodopa).
- Finish with the balanced current position: "*Deprescribing is safe and reduces inappropriate medication use; a mortality benefit has not been demonstrated.*"

Extra Points to Remember (recurring topic)

- **Classic prescribing cascades to quote:** metoclopramide/prochlorperazine → drug-induced parkinsonism → levodopa; amlodipine → ankle oedema → furosemide; NSAID → hypertension → antihypertensive; cholinesterase inhibitor → urinary urgency → oxybutynin (which then worsens cognition).
- **Fall-risk-increasing drugs (FRIDs):** benzodiazepines, Z-drugs, antipsychotics, antidepressants, antihypertensives, alpha-blockers, anticholinergics, opioids, antiepileptics. Withdrawal of psychotropics reduces falls requiring treatment.
- **Highest-risk drug classes in hospital** (Pathy, Ch. 11): **anticoagulants, opiates and centrally acting drugs**. Remember that **oxygen and intravenous fluids are drugs too** and are frequently given without the same caution.
- **Illustrative textbook vignette:** the 88-year-old woman on 21 medications whose regimen was reduced to 9 — she remained living independently with improved function and no further falls at 6 months.
- **A quotable image:** Pathy contrasts the "cornucopia" of plenty with the **Chimera** — polypharmacy looks like abundance but is a dangerous hybrid.

Question 4. Drug holiday 7 MARKS — SHORT NOTE

High-yield points

1. **Definition.** A drug holiday is a **planned, temporary, supervised interruption of a drug in a patient who is expected to resume it**, undertaken for a defined purpose and with a defined monitoring plan.
2. **Distinguish it from three other things.** It is **not** deprescribing (which is intended to be permanent), **not** non-adherence (which is unplanned), and **not** a dose reduction (the drug is stopped completely for a period).
3. **Purposes.** (i) To reduce cumulative toxicity from long-term exposure; (ii) to **test whether the drug is still needed**; (iii) to unmask an adverse effect wrongly attributed to disease; (iv) to allow recovery of receptor sensitivity or tolerance; (v) to permit a procedure or investigation.
4. **The principal current indication is the bisphosphonate drug holiday.** Bisphosphonates are retained in bone with a long terminal half-life, so anti-fracture protection persists after stopping, while the rare harms of prolonged use — **atypical femoral fracture (AFF)** and **osteonecrosis of the jaw (ONJ)** — are exposure-dependent.
5. **Practical bisphosphonate rule.** Reassess fracture risk after **5 years of oral** or **3 years of intravenous** therapy. If risk is low to moderate (no prior hip/vertebral fracture, hip T-score better than −2.5), offer a holiday of about **2–3 years**. If risk remains high, continue to **10 years oral / 6 years intravenous**. Restart if BMD falls significantly, bone turnover markers rise, or a fracture occurs.
6. **Critical exception — denosumab.** A drug holiday must **never** be taken with denosumab: cessation causes **rebound bone loss with multiple vertebral fractures**. Denosumab must always be followed by a bisphosphonate or another antiresorptive.
7. **Other situations where the term is used.** Gradual dose reduction and trial withdrawal of **antipsychotics in dementia** and of **benzodiazepines/hypnotics**; brief withdrawal of a **statin** to test whether myalgia is drug-related; periprocedural interruption of anticoagulants and antiplatelets; and short interruptions of some immunosuppressants and stimulants.
8. **An abandoned practice worth knowing.** The **levodopa drug holiday** in Parkinson's disease, once used to restore responsiveness, is now **contraindicated** because abrupt withdrawal precipitates a neuroleptic-malignant-like syndrome, aspiration, deep vein thrombosis and death.
9. **Risks of any drug holiday:** relapse of the treated disease, drug withdrawal syndromes, patient confusion about whether to restart, and being lost to follow-up so that the "holiday" silently becomes permanent.
10. **Good practice.** Document the **indication, the planned duration, the monitoring plan and the criteria for restarting** in the notes and in the patient's own record; agree it with the patient and family; and set a review appointment before the holiday begins.

Latest Updates (2023–Present) — three points

- **Duration is drug-specific, not one-size-fits-all.** The offset of effect differs between agents: alendronate has the longest residual protection, **risedronate the shortest** (holiday of ~1–2 years, with reassessment before 2 years), and zoledronic acid an intermediate 3–5 years. A large Ontario cohort found a higher hip fracture risk after 2 years off risedronate than off alendronate (*Annals of Internal Medicine*).
- **Individualisation is now the stated standard.** Commentaries and reviews (*J Clin Endocrinol Metab*, 2024–2025; *Current Osteoporosis Reports*, 2025–2026) conclude that holiday duration must be modified by total prior bisphosphonate exposure, agent used, hip BMD, falls risk, sex and body weight — **"one size does not fit all"**. AFF risk falls rapidly (~72% per year) after stopping.
- **Very old and very-high-risk patients are increasingly advised not to take a holiday at all,** because absolute hip fracture risk far exceeds the very low AFF risk (AFF accounts for fewer than 5 per 1000 hip fractures). ACP's living guideline and ACOG (2023) accept a holiday of 2–4 years but only after risk reassessment.

Current Practice Update

Textbook position (Essentials 7th ed.)	Current evidence and practice (2023-2026)
"The ultimate duration of bisphosphonate therapy is still not established... for many patients 3 to 5 years of treatment may suffice, but those at highest risk (including older patients) may benefit from longer treatment."	This remains directionally correct but is now operationalised: **formal reassessment at 5 years oral / 3 years IV**, holiday only for low-to-moderate risk, continuation to **10 years oral / 6 years IV** in high risk, with explicit restart criteria.
Drug withdrawal is discussed generically under deprescribing and tapering, without a structured "drug holiday" framework.	The drug holiday is now a **distinct, protocolised concept** with defined duration and restart rules, most fully developed for bisphosphonates and applied cautiously to antipsychotics and hypnotics.
Denosumab discussed as an antiresorptive option without a stopping rule.	**Absolute contraindication to a holiday** — rebound vertebral fracture risk mandates sequential antiresorptive therapy after stopping.

Examiner's Tip

- Open with the one-line definition and immediately separate it from deprescribing — **"planned and temporary, with the intention to resume."**
- State the numbers: **"5 years oral, 3 years intravenous, then reassess; holiday 2–3 years in low risk; continue to 10 years oral / 6 years IV in high risk."**
- Never omit: "*A drug holiday is contraindicated with denosumab because of rebound vertebral fractures.*"
- Mention the **levodopa drug holiday as an abandoned and dangerous practice** — this shows historical depth and is a common viva follow-up.

Question 5. Perioperative assessment and management in older people 15 MARKS

A. Principles and risk

1. **Age alone is not a contraindication.** Patients over 70 have higher rates of major perioperative complications and mortality after major non-cardiac surgery, but **mortality remains low even beyond 80**. Outcome is determined far more by **the presence and severity of systemic illness and whether surgery is elective or emergency** than by chronological age (Essentials, Table 3-18).
2. **Emergency surgery carries a much higher risk** than the same elective operation; wherever possible, convert an emergency into a semi-elective, optimised procedure.
3. **The first question is not "can he survive it?" but "should it be done?"** The appropriateness and risk–benefit ratio of the proposed operation must be considered explicitly, in the light of the patient's goals and life expectancy.

B. Preoperative assessment — systematic evaluation

4. **Optimise underlying conditions before non-emergency surgery** (Essentials, Table 3-18): cardiovascular disease especially **heart failure**; pulmonary status; renal function; **diabetes mellitus**; **thyroid disease, which is often occult**; anaemia; nutrition; and hydration/volume status, particularly in patients on diuretics.
5. **Identify high-risk cardiac conditions.** Substantially increased risk is conferred by **myocardial infarction within 6 months, pulmonary oedema, angina (especially unstable) and severe aortic stenosis**.
6. **Estimate cardiac risk formally.** Factors predicting perioperative cardiac complications in non-cardiac surgery are **ischaemic heart disease, congestive heart failure, diabetes mellitus, cerebrovascular disease and renal insufficiency** (Revised Cardiac Risk Index, Lee et al.).
7. **Assess functional capacity.** Exercise tolerance is more informative than laboratory testing — classically, **the ability to climb one flight of stairs**.
8. **Investigate selectively.** Order tests only if the result will change management. **Preoperative pulmonary function tests and arterial blood gases are rarely of prognostic value**; reserve them for respiratory symptoms, obesity, chest deformity (kyphoscoliosis), abnormal chest radiograph, or planned thoracic/upper abdominal surgery. Measure creatinine clearance where renal function is borderline or nephrotoxic/renally excreted drugs are used.
9. **Assess frailty — the geriatric addition to standard risk scoring.** Frailty (unintentional weight loss, weak grip, exhaustion, low physical activity, slow gait) **independently predicts postoperative complications, length of stay and discharge to a care facility** (Makary et al.). Frailty confers a **two- to fourfold increase in operative mortality** in cardiac surgery (Pathy, Ch. 35), and markers include Katz ADL ≤4/6, 5-metre walk ≥6 s, 6-minute walk <150 m, and Timed Up-and-Go ≥15 s.
10. **Predict 6-month mortality.** Preoperative **impaired cognition, recent falls, low albumin, greater anaemia, functional dependence and increased comorbidity** are associated with higher mortality at 6 months (Robinson et al.).
11. **Predict delirium.** Four independent preoperative predictors: **prior stroke or TIA, low MMSE score, abnormal serum albumin, and a high Geriatric Depression Scale score** (Rudolph et al.).
12. **Perform a comprehensive geriatric assessment** where time allows: medication review, functional assessment, cognition, nutrition, social and caregiver support, and **discussion of advance directives** before the patient goes to theatre.

C. Perioperative medical management

13. **Plan the medication regimen deliberately.** Some drugs must be continued, some withheld, and some dose-adjusted. Review against Beers/STOPP; inappropriate medication use in older surgical patients is common.
14. **Prophylaxis.** Give **venous thromboembolism prophylaxis** and **appropriate antimicrobial prophylaxis** according to procedure-specific protocols.
15. **Beta-blockade (textbook position).** Patients at high or intermediate cardiac risk with negative non-invasive tests should receive perioperative beta-blockade; those at low risk need none; positive non-invasive tests should prompt consideration of revascularisation. *See the Current Practice Update — this position has changed.*
16. **Revascularisation is not a licence for surgery.** Coronary artery bypass grafting or PCI should be limited to patients with an indication **independent of the need for non-cardiac surgery**.
17. **Anaesthetic technique should be individualised.** Regional anaesthesia is often favoured, but may require supplementary sedation and can itself cause significant cardiovascular change; **neither DVT incidence nor blood loss is substantially reduced** compared with general anaesthesia. Decide on patient factors, the procedure and the surgical team's preference.
18. **Postoperative care is geriatric care.** Prevent delirium (orientation, sensory aids, sleep hygiene, hydration, early mobilisation, avoid restraints and unnecessary catheters), give adequate analgesia, avoid bed rest, attend to nutrition and bowels, and start rehabilitation early. Functional decline in hospital may be irreversible — **one-third of older**

inpatients lose at least one ADL by discharge.

Latest Updates (2023–Present)

- **2024 AHA/ACC multisociety perioperative guideline for non-cardiac surgery** (*JACC / Circulation*, 2024) supersedes the 2014 guideline. It sets out a **stepwise algorithm**, urges **judicious and targeted** preoperative testing, and formalises functional assessment using **METs and the Duke Activity Status Index**. Key recommendations: **continue statins** perioperatively (Class 1) and initiate them in statin-eligible patients; **delay elective surgery for at least 3 months after stroke or TIA**; screen for **obstructive sleep apnoea** with validated questionnaires; use **emergency-focused cardiac ultrasound** for unexplained haemodynamic instability; and it names **myocardial injury after non-cardiac surgery (MINS)** as a distinct entity that must not be ignored.
- **New drug-class guidance. SGLT2 inhibitors should be stopped 3–4 days before surgery** to prevent euglycaemic ketoacidosis (2024 AHA/ACC). For **GLP-1 receptor agonists**, the 2024 multisociety guidance (ASA/AGA/ASMBS/ISPCOP/SAGES) advises that **most patients may continue the drug**, with a **24-hour clear liquid diet** before the procedure for those at higher risk of delayed gastric emptying (dose-escalation phase, high dose, GI symptoms, gastroparesis, Parkinson's disease) — replacing the earlier ASA 2023 advice to withhold weekly agents for 7 days.
- **ESAIC 2025 preoperative assessment guideline** (*Eur J Anaesthesiol*, 2025) **recommends frailty testing as an effective predictor of postoperative outcomes, particularly delirium (Grade 1C)**, and specifically the **Clinical Frailty Scale** when the examination suggests a frailty phenotype.
- **ESAIC 2024 postoperative delirium guideline** (*Eur J Anaesthesiol*, 2024): 13 recommendations, 5 of them strong — assess preoperative POD risk factors, optimise the preoperative condition, discuss prevention strategies, deliver a **non-pharmacological multicomponent intervention** in at-risk patients, and weigh risk/benefit before prophylactic dexmedetomidine. It mandates **screening in all patients ≥60 years**, delirium assessment with a validated tool **daily for at least 3 days starting in recovery**, and **EEG-based depth-of-anaesthesia monitoring**. Routine haloperidol prophylaxis, cholinesterase inhibitors and biomarker screening are **not** recommended; haloperidol is reserved for treatment when non-pharmacological measures fail.
- **2025 ASA Practice Advisory for perioperative care of older adults scheduled for inpatient surgery** (*Anesthesiology*, 2025) recommends **expanded preoperative evaluation** with cognitive and frailty screening, and states that **neither neuraxial nor general anaesthesia is superior for preventing postoperative delirium** — the choice should be by shared decision-making, and either TIVA or inhalational anaesthesia is acceptable.
- **Multicomponent prevention works.** A meta-analysis of 11 RCTs (3857 patients) found multicomponent interventions reduced postoperative delirium in older adults undergoing elective surgery by **29% (RR 0.71, 95% CI 0.59–0.85)** (2026). A network meta-analysis of drug interventions to prevent delirium after surgery in adults over 60 was published in the *BMJ* (2026).
- **Newly identified modifiable risk factors:** intraoperative hypotension, and **residual neuromuscular blockade** — the delirium risk associated with non-depolarising neuromuscular blockers disappeared when blockade was properly reversed with neostigmine or sugammadex (*Anesthesia & Analgesia*, 2025).
- **Prehabilitation** — structured preoperative exercise, nutritional supplementation, anaemia correction, smoking and alcohol cessation, and medication optimisation — is now an explicit part of the pathway wherever the interval before surgery permits.

Current Practice Update

Textbook position	Current evidence and practice (2023–2026)
Perioperative beta-blockade should be started in high- and intermediate-risk patients with negative non-invasive tests (Auerbach & Goldman, 2002).	**Substantially revised.** Since POISE, beta-blockers are **continued** in patients already taking them but **not started within 1 day of surgery** because of excess stroke, hypotension and death. Statins have replaced beta-blockers as the Class 1 perioperative drug recommendation.
Risk assessment based on comorbidity indices (RCRI) and exercise tolerance.	RCRI retained, but supplemented by **formal frailty testing (ESAIC 2025, Grade 1C)**, cognitive screening, and structured functional assessment (METs/DASI). **Frailty is now a mandated domain, not an optional extra.**
Perioperative delirium noted as an area of "growing awareness" with four predictors.	A fully protocolised field: **ESAIC 2024** and **ASA 2025** mandate screening ≥60 years, daily validated assessment for 3 days, multicomponent non-pharmacological bundles, and EEG-guided anaesthetic depth.
Regional anaesthesia "often favoured" but with stated disadvantages; decision individualised.	Confirmed and strengthened by trial data: **no superiority of neuraxial over general anaesthesia** for preventing delirium (ASA 2025). Individualisation remains correct.
Medication management described generally as continue / withhold / adjust.	Now drug-class specific: **continue statins and beta-blockers already prescribed; stop SGLT2 inhibitors 3–4 days before; in most cases continue GLP-1 agonists with a 24-hour clear liquid diet in higher-risk patients.**
Preoperative optimisation described as correcting individual organ deficits.	Reframed as **prehabilitation** — a bundled programme of exercise, nutrition, anaemia correction, glycaemic control, and deprescribing delivered in the weeks before surgery.

Examiner's Tip

- Open with the governing principle: "*Age above 70 increases risk, but comorbid conditions — especially cardiovascular — and emergency status are more important risk factors than age per se.*"
- Name the **four high-risk cardiac conditions** as a block: MI within 6 months, pulmonary oedema, unstable angina, severe aortic stenosis.
- Say clearly: **"Preoperative pulmonary function tests and arterial blood gases are rarely of prognostic value"** — a frequently rewarded negative statement.
- Quote the four delirium predictors (**prior stroke/TIA, low MMSE, abnormal albumin, high GDS**) and the frailty markers (**Katz ≤4/6, TUG ≥15 s, 5-m walk ≥6 s**).
- Flag the guideline reversal explicitly: "*The textbook recommendation to initiate perioperative beta-blockade has been superseded; since POISE, beta-blockers are continued but not started immediately before surgery.*"
- Close with the geriatric point: "*The operation is only half the treatment — delirium prevention, early mobilisation and rehabilitation determine functional outcome.*"

Extra Points to Remember (recurring topic)

- **Indian context.** A high proportion of older surgical patients present as emergencies (hip fracture, obstructed hernia, perforation), leaving little time for optimisation; undiagnosed diabetes, anaemia, hypothyroidism, tuberculosis and rheumatic valvular disease are common incidental findings at preoperative assessment. **Hip fracture surgery within 48 hours** and a formal **orthogeriatric co-management model** are the highest-yield service interventions. Access to DOACs, sugammadex and processed EEG monitoring is variable, so bridging and reversal plans must be made locally.
- **Cardiac surgery specifics (Pathy, Ch. 35):** risk is estimated by **EuroSCORE II** (Europe) or the **STS score** (USA). Crude in-hospital mortality in patients over 80 is approximately **5–9% for isolated CABG, 5–7% for isolated valve surgery and 8–11% for combined procedures**; five-year survival in patients ≥75 is about **80%**. Perioperative **stroke** is the complication of greatest concern, and **late referral** independently worsens both operative risk and late outcome.
- **TAVI vs SAVR:** frailty predicts postoperative mortality and disability after TAVI; survival curves stratify clearly by Katz ADL score. Decisions are made by a **Heart Team**.
- **Drugs to withhold vs continue — a quick list.** Continue: beta-blockers, statins, most antihypertensives, inhalers, antiepileptics, levodopa, thyroxine, steroids (with stress dosing). Withhold: ACE inhibitors/ARBs on the morning of surgery (hypotension), SGLT2 inhibitors 3–4 days, NSAIDs, metformin on the day, diuretics on the morning, and anticoagulants/antiplatelets per bleeding risk.
- **Postoperative complications specific to older people:** delirium, MINS, atrial fibrillation, aspiration pneumonia, urinary retention and catheter-associated infection, constipation and ileus, pressure injury, functional decline and new institutionalisation.

Appendix — Rapid Revision Grid

For the night before the examination. Numbers, named criteria, trials and guidelines by topic.

1. Numbers to quote

Item	Figure
Polypharmacy definitions in the literature	~24 different definitions
Polypharmacy / hyperpolypharmacy cut-offs	≥5 drugs / ≥10 drugs
Scotland trend, 1995→2010	≥5 drugs 11%→20%; ≥10 drugs 1.5%→6%; serious interaction exposure 13% (2010)
Sweden (1992→2002); Italy (2000→2010)	18%→42%; 43%→53%
Medication errors, UK nursing home residents	69.5% incidence
Beers Criteria 2019 / 2023	88 PIMs (2019); 5 categories retained in 2023; >3 dozen drugs to avoid, 40+ disease-specific
STOPP/START v2 (2015) / v3 (2023)	114 (80+34) / **190 (133+57)** — a 66.7% increase
Medication Appropriateness Index	10 items, 3-point Likert, maximum score 18
Pill Pruner	13 drug groups, pocket card, effects sustained at 90 days
Bisphosphonate holiday	Reassess at 5 y oral / 3 y IV; holiday 2–3 y (low risk); continue 10 y oral / 6 y IV (high risk); AFF risk falls ~72%/year after stopping
Frailty and operative mortality	2- to 4-fold increase; Katz ≤4/6, TUG ≥15 s, 5-m walk ≥6 s, 6-min walk <150 m
Cardiac surgery mortality, age >80	CABG 5–9%; isolated valve 5–7%; combined 8–11%; 5-year survival ≥75 y ≈ 80%
Functional decline in hospital	One-third of older inpatients lose ≥1 ADL by discharge
Multicomponent delirium prevention	RR 0.71 (95% CI 0.59–0.85) — 29% relative risk reduction
Deprescribing meta-analyses	32 RCTs / 18,670 patients (JAMDA 2023); 259 studies, no mortality benefit (BJCP 2024)
Pharmacist-led deprescribing CMR	2.5 vs 1.7 medications stopped/reduced per patient at 6 months; no HRQoL change
SGLT2 inhibitors before surgery	Stop **3–4 days** preoperatively
Elective surgery after stroke/TIA	Delay **≥3 months**
Postoperative delirium screening	All patients **≥60 years**; validated tool **daily for ≥3 days** from recovery

2. Named criteria, tools and mnemonics

Name	Content
Beers (1991, AGS)	5 categories + anticholinergic table; 2023 update; 2025 Alternatives companion
STOPP / START (2008, Europe)	PIMs / prescribing omissions; v3 2023, 190 criteria, class-based
STOPPFrail	For frail patients with limited life expectancy
MAI (1992, USA)	10 implicit criteria per drug
DBI (2007)	Cumulative anticholinergic + sedative exposure
FORTA (2008, Germany)	A–D labels; first tool to combine positive and negative labelling
TRIM (2016, US VA)	EHR algorithm using Beers + STOPP
SAIL / TIDE	Simple, Adverse effects, Indication, List / Time, Individual response, Drug interactions, Educate
ARMOR	Assess, Review, Minimise, Optimise, Reassess
ERASE	Resolved conditions, Ageing normally, Selecting targets → Eliminate unnecessary diagnoses
AVOID TOO MANY	12-item medication review checklist
Scott 5-step	List → assess harm → discontinuation potential → prioritise → monitor
Barnett 7-step / **Deprescribing Rainbow**	Goals-first process / clinical, psychological, social, financial and physical determinants
RCRI (Lee)	IHD, CHF, diabetes, cerebrovascular disease, renal insufficiency, high-risk surgery
EuroSCORE II / STS score	Cardiac surgical operative mortality prediction
Rudolph delirium rule	Prior stroke/TIA, MMSE, abnormal albumin, GDS
Clinical Frailty Scale	Recommended preoperatively by ESAIC 2025 (Grade 1C)
DASI / METs	Functional capacity assessment, 2024 AHA/ACC guideline

3. Guidelines and evidence, 2023–2026

Source	Key message
AGS Beers Criteria, JAGS 2023	Aspirin for primary prevention → avoid; warfarin and rivaroxaban → avoid as initial therapy; doxepin removed; SGLT2i caution
AGS Beers Alternatives, JAGS 2025	Safer pharmacological and non-pharmacological alternatives across 21 conditions
STOPP/START v3, Eur Geriatr Med 2023	190 criteria; class-based; 41–42% new criteria
SENATOR / OPERAM / OPTICA	No significant reduction in ADRs, mortality, readmissions or QoL
JAMDA 2023; BJCP 2024; Age Ageing 2026	Deprescribing is safe and reduces PIMs; no mortality or QoL benefit demonstrated
2024 AHA/ACC multisociety perioperative guideline	Stepwise algorithm; targeted testing; statins Class 1; delay elective surgery ≥3 months after stroke; OSA screening; MINS recognised; stop SGLT2i 3–4 days
ESAIC preoperative assessment, EJA 2025	Frailty testing recommended (1C); Clinical Frailty Scale; WHODAS 2.0 in high-risk patients
ESAIC postoperative delirium, EJA 2024	13 recommendations, 5 strong; screen ≥60 y; multicomponent non-pharmacological bundle; EEG depth monitoring; dexmedetomidine risk–benefit; haloperidol only if non-pharmacological measures fail
ASA Practice Advisory, Anesthesiology 2025	Expanded preoperative evaluation of older inpatients; neuraxial and general anaesthesia equivalent for delirium risk
GLP-1 multisociety guidance, 2024	Continue in most patients; 24-hour clear liquid diet if higher risk; supersedes 7-day withholding of weekly agents
WHO Medication Without Harm	Third Global Patient Safety Challenge; polypharmacy is a named priority action area

Five statements that must not be got wrong

1. **Absorption is the pharmacological parameter least affected by ageing; renal excretion is the most predictable change.**
2. **A normal serum creatinine does not mean normal renal function in an older person.**
3. **A drug holiday is absolutely contraindicated with denosumab** because of rebound vertebral fractures; the **levodopa drug holiday is an abandoned, dangerous practice**.
4. **Do not start beta-blockers immediately before surgery** — continue them if already prescribed. This reverses the older textbook recommendation.
5. **Deprescribing is safe and reduces inappropriate medication use, but no mortality benefit has been demonstrated** — state this rather than overclaiming.

Textbook content drawn from *Pathy's Principles and Practice of Geriatric Medicine* (6th ed.) and *Essentials of Clinical Geriatrics* (7th ed.). Updates cited inline by journal or guideline and year, current to 2026.

MD GERIATRIC MEDICINE

Examination Answer Module

Immobility, Pressure Injuries, Rehabilitation & Geriatric Pruritus

Questions 1–7 | 15-mark and 7-mark formats

Primary sources: Pathy's Principles and Practice of Geriatric Medicine, 6th ed.

Kane's Essentials of Clinical Geriatrics

With Latest Updates (2023–present) and Indian clinical & policy context

Prepared for Dr. Sudarsan · Written Examination Preparation

Contents

1.	Assessment and management of chronic pressure ulcers in the elderly RECURS	15 marks
2.	Prevention of pressure sores RECURS	7 marks
3.	Immobility RECURS	15 marks
4.	Rehabilitation team	7 marks
5.	Role of physiotherapy in the elderly	7 marks
6.	Role of the occupational therapist in the life of an elderly person with mobility issues and an ageing spouse	15 marks
7.	How would you deal with constant itching (pruritus) in an elderly patient?	15 marks

How to use this module

Each answer runs in the same four blocks: **High-Yield Points** (textbook core) → **Latest Updates 2023–present** → **Current Practice Update** (textbook vs. current evidence, in table form) → **Exam One-Liners**. Questions marked RECURS carry an additional purple **Rapid Recall Box** with a mnemonic, numerical anchors, examiner traps and a 15-second answer skeleton. All laboratory values are in Indian conventional units.

Question 1. Assessment and management of chronic pressure ulcers in the elderly [15 MARKS]

High-Yield Points

Definition and terminology

1. A **pressure injury** is localised damage to the skin and underlying tissue, usually over a **bony prominence** such as the sacrum or heel. Pathy's prefers "pressure **injury**" over "pressure ulcer" because not every lesion is an open ulcer.
2. A **chronic** pressure ulcer is one that fails to progress through orderly healing. A useful benchmark: the wound should be **20–40% smaller by week 4** if it is going to heal by week 12. Failure to shrink means the diagnosis and the underlying factors must be re-examined.
3. Three mechanical forces produce the injury: **pressure**, **shear** (head-of-bed elevation makes the torso slide and drags subcutaneous tissue over the sacrum) and **friction**. **Moisture** from sweat, urine and faeces compounds all three. More than **90% occur in the lower body** – sacrum and coccyx, ischial tuberosities, greater trochanter and heels.

Epidemiology and risk

4. Incidence varies widely by setting: **acute care 0.4–38%**, **long-term care 2.2–23.9%**, **home care 0–17%**. About **25% of pressure ulcers are acquired intra-operatively**, because neither position nor duration of surgery can be altered.
5. **Advanced age is NOT an independent risk factor.** The risk comes from the impairments that cluster in old age – immobility, poor nutrition and fragile skin. This is a favourite examiner point.
6. Established risk factors: bed- or chair-bound, **unable to reposition without help**, difficulty with ambulation, **history of stroke**, **faecal incontinence**, low body weight, **lymphopenia**, difficulty with independent feeding, impaired nutritional intake, **non-blanchable erythema of intact skin (a Stage 1 injury)**, and **dry sacral skin**.
7. Risk-assessment tools in use: **Braden, Norton and Waterlow** scales. The recommendation is to apply a tool **on admission and repeat regularly**. Evidence that the scales themselves improve outcomes remains uncertain – they supplement, not replace, structured skin inspection.

Clinical staging — NPIAP system

8. **Stage 1**: intact skin with non-blanchable erythema. **Stage 2**: partial-thickness loss with exposed dermis – blister, abrasion or shallow ulcer. **Stage 3**: full-thickness loss into subcutaneous fat, down to but not through fascia, often with undermining. **Stage 4**: extends through deep fascia – muscle, tendon or bone visible, undermining and sinus tracts common. Use **Arabic numerals**, not Roman.
9. Two additional categories when depth cannot be seen: **Unstageable** – base obscured by slough or eschar (stage it once debrided); and **Deep tissue pressure injury (DTPI)** – skin intact but deep red, purple or maroon, or a haemorrhagic blister, indicating damage in deeper tissue.
10. **Medical device–related pressure injuries** (urinary catheter, rectal tube, oxygen tubing) **conform to the shape of the device** and **are staged**. **Pressure injuries on mucous membranes are documented but NOT staged.**

Assessing the chronic ulcer

11. **Never reverse-stage.** A healing Stage 4 does not become a Stage 2 – the lost tissue is replaced by scar, not by fat and muscle. Monitor healing instead with the **PUSH tool** (Pressure Ulcer Scale for Healing), which scores three components – **size, exudate amount and tissue type** – and is validated and sensitive to change.
12. Document every ulcer under seven headings: (i) type, duration and setting in which it arose; (ii) **length × width × depth in cm**; (iii) colour as percentages – red granulation, yellow slough, black eschar; (iv) exudate – serous, serosanguinous, sanguineous or purulent; (v) odour, assessed only **after** thorough cleaning; (vi) periwound skin – viable, macerated, inflamed or hyperkeratotic; (vii) **undermining and sinus tracts**. Add a pain score.
13. **Diagnosing infection in a chronic wound**: **increasing pain is the single best clinical predictor (likelihood ratio 11–20)**. The "classic signs" – purulent exudate, erythema, heat, oedema – and foul odour do **not** reliably predict infection. When infection is suspected, take a **quantitative swab using the Levine technique**: rotate the swab over a **1 × 1 cm** area for **five seconds** with enough pressure to express fluid from within the tissue. A positive result gives LR 6.3; a negative result LR 0.47.
14. Suspect **osteomyelitis** in any non-healing Stage 4 ulcer: probe-to-bone, plain radiograph, MRI. **Bone or deep tissue biopsy culture** – including anaerobic culture – directs therapy far better than surface swabs.

Management

15. **Managing the underlying contributing factors matters more than any dressing or adjunctive therapy.** Offload pressure, correct nutrition, control incontinence, treat pain, restore mobility, review sedatives and restraints. Most trials unfortunately study dressings, not causes.
16. **Support surfaces and repositioning**: reposition at least **2-hourly on a standard mattress** and at least **4-hourly on a pressure-redistributing mattress**. Powered mattresses have **not** been shown to heal ulcers better than non-powered ones despite far greater cost; a disciplined turning schedule is a cheaper alternative.
17. **Local wound care**: no single dressing has been shown superior to another in well-designed trials. Standard care of a

healable ulcer must satisfy three criteria – **moisture balance, bacterial balance and debridement**. Choose the dressing on cost, ease of use, goals of care and patient comfort.

18. **Debridement** of necrotic tissue by autolytic, enzymatic/chemical, mechanical, sharp or surgical means; irrigate two to three times daily; undermine and remove eschar that conceals infected tissue. **Exception**: leave stable, dry, adherent **heel eschar** alone in a limb with peripheral arterial disease – it is a biological dressing.
19. **Antibiotic discipline**: do not treat colonisation. In **Stage 3**, treat only proven **cellulitis, osteomyelitis, bacteraemia or sepsis**; routine surface cultures are unhelpful because they grow mixed flora and miss anaerobes, and reflect colonisation. In **Stage 4**, systemic antimicrobials are often required; for a septic patient use broad-spectrum cover for **anaerobes, Gram-negative organisms and *Staphylococcus aureus***, then de-escalate on tissue culture.
20. **Nutrition**: refer to a dietitian. Targets are **30–35 kcal/kg/day energy**, **1.0–1.5 g/kg/day protein** and **1 mL fluid per kcal**. Supplementation improves healing chiefly in those who are already malnourished; protein supplements may help long-term-care residents. Check haemoglobin (g/dL) and serum albumin (g/dL) as markers of the overall nutritional state.
21. **Adjunctive therapies** – negative-pressure wound therapy, hyperbaric oxygen, laser, electrotherapy, electromagnetic therapy, therapeutic ultrasound: **no good evidence that any of these improve pressure injury healing.** Platelet-derived growth factor has some supporting evidence but not enough for routine use.
22. **Surgery**: plastic surgical consultation for selected Stage 4 lesions – flap or graft closure – but only when the patient is fit, nutrition is corrected and, above all, the **cause has been removed**, otherwise the flap breaks down.
23. **Non-healable ulcers and skin failure**: not all pressure injuries are preventable. Where the underlying conditions are irreversible or the goals of care are comfort, shift the aim from healing to **reducing infection risk and controlling pain**. Wounds that appear and worsen over bony prominences during overall terminal decline are termed **skin failure** rather than pressure injury.
24. **Pain is managed at every stage** – background pain and procedural pain at dressing change. Use topical anaesthesia, pre-emptive analgesia and the WHO ladder with geriatric dose adjustment.

Latest Updates (2023-present)

1. **International Guideline, 4th edition (2025)** – NPIAP / EPUAP / Pan Pacific Pressure Injury Alliance, edited by Emily Haesler, replacing the 2019 third edition. Built with **GRADE and evidence-to-decision frameworks** by more than 240 clinical experts and consumer representatives, and released as **staged online chapters** (prevention chapters first from February 2025, treatment recommendations from late 2025) rather than a single printed book.
2. **Sub-epidermal moisture (SEM) measurement and skin temperature** are now recommended as **adjuncts to visual skin assessment** for detecting Stage 1 injury and deep tissue injury before they are visible, particularly at the sacrum. This matters especially in **darkly pigmented skin**, where non-blanchable erythema is easily missed – highly relevant to Indian practice.
3. **Prophylactic dressings** have moved into guideline recommendations: a **soft silicone adhesive multilayered foam dressing** is suggested as an adjunct to heel elevation and repositioning for heel injuries, and over the sacrum and trochanters in at-risk hospitalised patients (BORDER trial lineage). NPIAP/EPUAP have launched the **Prophylactic Dressing Standards Initiative (PDSI)** because no accepted standard yet exists for testing dressings used on *intact* skin.
4. **SPIPP** (Standardized Pressure Injury Prevention Protocol) – a five-item checklist now widely used as an audit tool: validated risk assessment, prophylactic dressings, beds and support surfaces, repositioning systems, and skin care.
5. Device-related injuries have their own 2025 chapter, extending the concept beyond medical devices to **non-medical items** – phones, remote controls, call bells, jewellery, spectacles and bed clutter left in contact with skin.
6. Wound Canada 2025 Best Practice Recommendations reinforce the **20–40% area reduction at week 4** benchmark and formal reclassification of a stalled wound as **non-healing or non-healable** by the interprofessional team.

Current Practice Update

Domain	Textbook position	Current practice (2023-present)
Guideline reference	2019 third edition International Guideline; NPUAP 2016 revised staging	**Fourth edition, 2025**, GRADE-based, released as living online chapters
Early detection	Visual inspection plus a risk scale	Add **SEM scanner and skin temperature**; explicit caution about missing Stage 1 in dark skin
Prophylactic dressings	Barely mentioned	**Multilayer silicone foam** over sacrum, heels and trochanters as an adjunct; PDSI standards in development
Repositioning	Fixed 2-hourly / 4-hourly rule	**Individualised frequency** based on risk, tissue tolerance and support surface, documented on a turning chart
Nutrition	"Supplements may be beneficial; unclear which nutrients"	Structured screening plus **high-protein oral nutrition supplements enriched with arginine, zinc and antioxidants** for Stage 2 and above in

		malnourished patients
Adjunctive therapy	No good evidence for any	**NPWT** accepted as an adjunct in large Stage 3/4 cavities to reduce volume and exudate and prepare for surgery – still not proven to achieve closure
Non-healing wounds	Concept of skin failure introduced	Formalised: **unavoidable pressure injury** and **Kennedy terminal ulcer** as recognised end-of-life entities, with a documented prevention protocol required before the label is used

Indian Context

1. **NPHCE** provides geriatric wards at district hospitals and regional geriatric centres, where pressure injury prevention should be an audited quality indicator.
2. **Kerala's community palliative care network** – ward-level nurse-and-volunteer home-care teams under the state palliative care policy – remains India's best-developed model for the bedbound elderly, and pressure sore care is one of its core deliverables. **Vayomithram** mobile units add urban outreach.
3. **AB-PMJAY** and the **Ayushman Vay Vandana Card** (universal for those aged 70 and above) cover hospitalisation, debridement and flap surgery, but not chronic outpatient dressings.
4. **Rashtriya Vayoshri Yojana** and **ADIP–ALIMCO** supply wheelchairs, walkers, commodes and other assistive devices free or subsidised to eligible senior citizens – central to offloading and mobilisation.
5. Practical realities: alternating-pressure mattresses are frequently unavailable; **disciplined 2-hourly turning by a trained family caregiver, cotton draw sheets, a well-padded water or air mattress, and daily heel inspection** deliver most of the benefit. Vegetarian diets often fall short of the 1.0–1.5 g/kg protein target – counsel on dal, curd, paneer, eggs and locally affordable protein sources against the **ICMR-NIN 2024** reference values.

Rapid Recall Box — Recurring Topic

Mnemonic for the wound bed: "DIME" – **D**ebridement, **I**nfection/inflammation control, **M**oisture balance, **E**dge of wound advancing. (Pathy's three criteria – moisture balance, bacterial balance, debridement – map directly onto D, I and M.)

Numerical anchors: 2-hourly / 4-hourly turning · 30–35 kcal/kg · 1.0–1.5 g/kg protein · 1 mL fluid per kcal · 20–40% area reduction by week 4 · increasing pain LR 11–20 · Levine swab 1 × 1 cm for 5 seconds, LR 6.3 positive / 0.47 negative · 25% acquired intra-operatively · more than 90% in the lower body · acute care incidence 0.4–38%.

Examiner traps: (i) Saying "old age is a risk factor" – it is *not* independent. (ii) Reverse-staging a healing ulcer. (iii) Staging a mucosal injury. (iv) Sending a routine surface swab and starting antibiotics for a Stage 3 ulcer. (v) Claiming powered mattresses heal better than non-powered. (vi) Debriding dry stable heel eschar in an ischaemic limb. (vii) Forgetting that *all* pressure injuries are not avoidable, and that skin failure is a distinct end-of-life entity.

15-second answer skeleton: Define → pressure, shear, friction, moisture → NPIAP Stages 1–4 plus unstageable and DTPI, device-related staged, mucosal not staged → assess with the seven documentation headings, PUSH tool, never reverse-stage → infection = increasing pain plus Levine swab → management = correct the underlying cause first, then offload, nutrition, DIME wound bed, selective antibiotics, surgery for selected Stage 4 → non-healable ulcer and skin failure with palliative goals → 2025 fourth-edition guideline, SEM scanning, prophylactic silicone foam → Indian context.

Exam One-Liners

1. "Pressure injury" is preferred over "pressure ulcer" because not all lesions are open ulcers.
2. Advanced age is **not** an independent risk factor for pressure injury development.
3. Dry sacral skin and lymphopenia are both recognised independent risk factors.
4. Deep tissue pressure injury = intact skin that is deep red, purple or maroon, or a haemorrhagic blister.
5. Unstageable = base hidden by slough or eschar; stage it after debridement.
6. Mucous membrane pressure injuries are documented but never staged.
7. Reverse staging is inaccurate; use the PUSH tool (size, exudate, tissue type).
8. The best clinical predictor of infection in a chronic wound is **increasing pain**.
9. Levine technique: rotate a swab over 1 × 1 cm for 5 seconds with pressure enough to express tissue fluid.
10. No single dressing is superior; aim for moisture balance, bacterial balance and debridement.
11. Adjunctive therapies including hyperbaric oxygen have no good evidence of improving healing.
12. About one-quarter of pressure ulcers are acquired in the operating theatre.
13. Skin failure describes wounds appearing over bony prominences during terminal decline.
14. The 2025 fourth-edition International Guideline is a GRADE-based, staged online release.

Question 2. Prevention of pressure sores [7 MARKS]

> **Note on overlap:** This question shares its evidence base with Question 1 but is answered **purely as prevention**. Do not repeat staging, wound-bed preparation or antibiotic policy here – the pivot is risk identification, offloading, skin and moisture care, nutrition, and the avoidability question.

High-Yield Points

1. **Identify risk on day one.** Apply a validated tool – **Braden, Norton or Waterlow** – on admission to any facility and **repeat regularly**, combined with a structured head-to-toe skin inspection that specifically includes the **heels, sacrum and skin under every device**.
2. **Redistribute pressure.** Reposition at least **2-hourly on a standard mattress** and at least **4-hourly on a pressure-redistributing mattress**. Use a **30° lateral tilt** rather than full side-lying, keep head-of-bed elevation to **30° or less** to limit sacral shear, and **float the heels completely off the bed** with a pillow under the calf or a heel-suspension boot.
3. **Choose a support surface.** Non-powered surfaces are mattresses or overlays filled with air, water, gel or foam; powered surfaces mechanically vary the pressure and cost far more. **Specialised foam and sheepskin overlays reduce incidence compared with a standard hospital mattress**; the choice between powered and non-powered is not settled. **Overlays on the operating table reduce postoperative pressure injuries** and should be routine for long operations in the elderly.
4. **Reduce shear and friction.** Use slide sheets, hoists and two-person lifts rather than dragging; keep linen taut and wrinkle-free; avoid loose folded skin, which is common in the elderly from loss of subcutaneous fat and dehydration.
5. **Control moisture and incontinence.** Clean promptly after every episode with a pH-balanced cleanser, dry gently, and apply a barrier film or cream. Recognise and treat **incontinence-associated dermatitis** as a separate entity – it is frequently misclassified as a Stage 2 injury.
6. **Moisturise dry skin. Dry sacral skin is itself a risk factor**, so regular emollient application is a reasonable preventive measure. No topical agent has been shown superior to any other.
7. **Nutrition and hydration.** Refer at-risk patients to a dietitian for a full nutritional assessment. Targets: **30–35 kcal/kg/day**, **protein 1.0–1.5 g/kg/day**, **fluid 1 mL/kcal**. Nutritional supplements may help but it is not clear which nutrients matter most.
8. **Attack immobility itself.** Avoid excessive bed rest, get the patient up for meals, optimise and encourage function, avoid over-sedation, minimise **physical restraints, restrictive siderails, catheters and lines** – every one of these enforces immobility.
9. **Device care.** Inspect the skin under and around all medical devices at least twice daily, reposition or rotate the device, and consider a protective dressing beneath it. Suspect a device-related injury whenever the wound shape mirrors the device.
10. **Multifactorial, multidisciplinary bundles work** – exactly as for falls and delirium prevention. But **not all pressure injuries are avoidable, particularly at the end of life**; a documented prevention protocol must be in place before any injury is declared unavoidable.

> **Latest Updates (2023–present) — three key points**
>
> 1. **Fourth edition International Guideline (NPIAP/EPUAP/PPPIA, 2025)** – the prevention chapters were released first as living online documents using GRADE methodology; the **SPIPP checklist** distils prevention into five auditable practices: validated risk assessment, prophylactic dressings, support surfaces, repositioning systems and skin care.
> 2. **Prophylactic soft silicone multilayered foam dressings** are now suggested as an **adjunct** to heel elevation and repositioning (and over sacrum and trochanters) where resources permit – not a replacement for turning. The NPIAP/EPUAP **Prophylactic Dressing Standards Initiative** is developing performance standards for dressings applied to intact skin.
> 3. **Sub-epidermal moisture (SEM) and skin temperature measurement** are recommended as adjuncts to visual skin assessment, detecting damage before it is visible – of particular value in **darkly pigmented skin**, where Stage 1 erythema is frequently missed and injuries are therefore diagnosed later and at a higher stage.

Current Practice Update

Domain	Textbook position	Current practice
Skin assessment	Visual inspection plus risk scale	Plus **SEM and temperature**; specific protocols for dark skin tones (colour change relative to surrounding skin, boggy or firm texture, warmth)
Dressings	Used only on established wounds	**Prophylactic dressings on intact skin** as a recognised preventive intervention
Repositioning	Fixed schedule	Individualised and documented; supported by **patient repositioning systems** as a named SPIPP element

Avoidability	Not all are avoidable	Unavoidable pressure injury now a defined, defensible category, provided a prevention protocol is documented

Indian Context

Under **NPHCE**, pressure sore prevention is best delivered as **structured family-caregiver training**, since most bedbound elderly in India are cared for at home. **ASHA and ANM home visits**, and in Kerala the **ward-level palliative home-care nurse**, are the practical delivery route. Low-cost, culturally acceptable measures carry most of the benefit: a two-hourly turning chart kept on the wall, water or air mattress where affordable, cotton draw sheets, coconut or sesame oil as an emollient for dry sacral skin, immediate cleaning after incontinence, and daily heel checks. **Rashtriya Vayoshri Yojana / ADIP–ALIMCO** supply wheelchairs, walkers and commodes free to eligible elderly, which supports getting the person out of bed – the most effective preventive measure of all.

Exam One-Liners

1. Prevention rests on identifying risk, offloading pressure, and controlling moisture, friction and nutrition.
2. Turn at least 2-hourly on a standard mattress, 4-hourly on a pressure-redistributing mattress.
3. Limit head-of-bed elevation to 30° to reduce sacral shear.
4. Heels must be floated completely off the surface, not merely cushioned.
5. Specialised foam and sheepskin overlays reduce incidence versus a standard hospital mattress.
6. Theatre-table overlays reduce postoperative pressure injuries.
7. Dry sacral skin is a risk factor – moisturising is a legitimate preventive intervention.
8. Incontinence-associated dermatitis is not a Stage 2 pressure injury.
9. Prophylactic silicone foam dressings are an adjunct to, never a substitute for, repositioning.
10. An unavoidable pressure injury can only be declared after a documented prevention protocol has been applied.

Question 3. Immobility [15 MARKS]

High-Yield Points

Definition

1. **Immobility** is the restriction of time spent out of bed or chair, imposed by **medical orders, restrictive devices, chemical restraints such as antipsychotics, lack of mobility aids, or lack of human assistance and encouragement**. It is therefore as much an **iatrogenic and environmental** problem as a disease.
2. **Functional decline or "deconditioning"** is the loss of ability to perform basic activities of daily living, attributed primarily to **forced bed rest, immobilising devices such as catheters and lines, restraint use, and lack of encouragement of independence in self-care**.

Epidemiology — why it matters

3. As many as **one-third of older hospitalised patients are restricted to bed rest or chair rest**. Between **15% and 76%** show diminished performance in at least one ADL at discharge.
4. Of those who decline at discharge, **only half recover function by three months**, and for many the decline results in **permanent loss of independent living**.
5. Functional decline is a **profound marker of morbidity and mortality**, producing longer length of stay, greater cost and increased nursing-home placement.

Causes (Kane's classification)

6. **Musculoskeletal**: arthritides, osteoporosis, fractures (especially hip and femur), podiatric problems, Paget's disease.
7. **Neurological**: stroke, Parkinson's disease, neuropathies, normal-pressure hydrocephalus, dementias, cerebellar dysfunction.
8. **Cardiovascular and pulmonary**: severe congestive cardiac failure, coronary disease with frequent angina, peripheral arterial disease with claudication, severe COPD – all limiting endurance rather than strength.
9. **Sensory**: visual impairment, decreased kinaesthetic sense, decreased peripheral sensation.
10. **Drugs**: sedatives and hypnotics (drowsiness, dizziness, delirium, ataxia); **antipsychotics, especially typical agents** (rigidity, extrapyramidal effects); antihypertensives (orthostatic hypotension, bradycardia).
11. **Psychological**: depression, apathy, lack of motivation, **fear of falling**, and the health belief that **"rest and immobility aid recovery"**.
12. **Environmental and social**: forced immobility in hospitals and nursing homes, inadequate mobility aids, cluttered rooms, uneven surfaces, badly shaped or low chairs and beds, stairs – and, crucially, **well-meaning caregivers who bathe, dress and wheel an elder who is capable of doing it themselves**, which directly produces deconditioning.
13. **Others**: acute and chronic pain (a major cause, creating a vicious cycle of pain → less movement → more pain), malnutrition, severe systemic illness such as widespread malignancy, and deconditioning after prolonged bed rest for acute illness.

Complications — system by system

System	Complications of immobility and bed rest
Skin	Pressure injuries (immobility plus low serum albumin markedly increases risk)
Musculoskeletal	Muscle atrophy with loss of strength and endurance; reduced fibre size, diameter and capillarity; **contractures** (fixation of both muscle and joint in flexion, permanently reducing range of motion – the classic nursing-home consequence); **disuse osteoporosis** from increased resorption and decreased formation, with **hypercalcaemia**; degenerative joint disease
Cardiovascular	Deconditioning, decreased plasma volume, decreased maximal stroke volume and aerobic capacity; **orthostatic hypotension** (impairs rehabilitation and predisposes to falls, stroke and myocardial infarction); **deep vein thrombosis and pulmonary embolism**
Respiratory	Reduced tidal volume, atelectasis, and – with the supine position – **aspiration and hypostatic pneumonia**
Gastrointestinal	Anorexia, constipation, **faecal impaction with overflow incontinence**
Genitourinary	Urinary retention (inability to void lying down, or impaction obstructing flow), infection, bladder calculi, functional incontinence
Metabolic	**Negative nitrogen and calcium balance**, impaired glucose tolerance, altered drug pharmacokinetics from reduced plasma volume
Psychological	Sensory deprivation, **delirium**, depression and anxiety

Assessment

14. **History**: explore the **patient's and the caregiver's perceived cause** of the immobility, not just the medical one. Cover medical conditions, treatments including intravenous lines and catheters, pain, mood, motivation, and health beliefs.

15. **Nutrition**: assess protein status and **25-hydroxyvitamin D**, both associated with muscle weakness, poor physical performance, balance problems and falls. Check haemoglobin (g/dL) and albumin (g/dL).
16. **Environment**: assess both the physical and the social environment, **particularly caregiving interactions** – any of these factors can reduce the person's willingness to engage in activity.
17. **Examination**: grade muscle strength (MRC 0–5), joint range of motion and contractures, skin over all pressure points, postural blood pressure, gait and balance (Timed Up and Go, Short Physical Performance Battery), vision, feet and footwear.

Management

18. **Treat the reversible causes** – relieve pain, deprescribe sedatives and antipsychotics, treat depression, correct vitamin D and protein deficits, manage orthostasis, optimise cardiac and respiratory disease.
19. **Adopt a restorative philosophy of care** (Kane's basic principles): optimise treatment of underlying disease, nutrition, hydration and the psychosocial situation; optimise the physical environment (handrails, open access) and the care environment so care is not merely task-focused; **prevent secondary disabilities**; treat primary disabilities; set realistic, individualised, measurable functional goals; emphasise functional independence; enhance residual capacity; provide adaptive tools; adapt the environment to the patient; attend to motivation of patient *and* caregivers; and use a **team approach**.
20. **Progressive exercise programme** – passive and active range of motion, bed and chair exercises, sitting and standing balance, transfers, then assisted and independent ambulation; lower-limb resistance training with flexibility and balance work; correctly fitted cane, walker or wheelchair with training in its use.
21. **Prevent the complications actively**: 2-hourly turning, VTE prophylaxis, upright positioning with chest physiotherapy and breathing exercises, a bowel and bladder regimen, adequate nutrition and hydration, and **early removal of catheters and lines**.
22. **Minimise physical restraints and restrictive siderails.** Tinetti and Ginter described **"spiralling immobility"** – the older person perceived to be at risk of falling is restrained to prevent falls, and then becomes unable to walk safely because of the immobilising consequences of the restraint. Restraints are associated with numerous negative outcomes including **strangulation and asphyxiation**, and research demonstrates they can be **safely eliminated**.
23. Rehabilitation may be delivered in the acute hospital, a rehabilitation unit, a skilled nursing facility, an outpatient clinic or at home; ongoing **motivation of the older person and the caregiver is critical** to success.

Latest Updates (2023–present)

1. **"New horizons in hospital-associated deconditioning" (Age and Ageing, 2024;53:afae241)** reframes deconditioning as **a global condition of body and mind**: physical deconditioning drives secondary sarcopenia, and **cognitive deconditioning** – delayed mental processing on a spectrum with frank delirium – is equally common. The paper cautions that the word "deconditioning" wrongly implies easy reversibility, and recommends implementation of a **Frailty Care Bundle** targeting **mobilisation, nutrition and cognitive engagement** together.
2. A 2025 *Age and Ageing* editorial ("75 years after Marjorie Warren, the know-do gap around hospital-associated functional decline is as wide as ever") makes the point that the evidence is settled and the failure is one of **implementation**, not knowledge.
3. **In-hospital multicomponent exercise reverses functional decline** in acutely hospitalised very elderly patients (Martínez-Velilla, *JAMA Internal Medicine* 2019) – still the landmark trial, now replicated and extended. A **2025 systematic review and meta-analysis (European Geriatric Medicine)** shows that **progressive mobilisation and multicomponent interventions improve participation and reduce functional decline in hospitalised people living with dementia**, a group previously excluded from such programmes.
4. Implementation campaigns now define the standard of care: **#EndPJparalysis**, **"Sit up, Get dressed, Keep moving"** (British Geriatrics Society deconditioning hub), **"Get up, Get dressed, Get moving"**, and **MOVE ON** (Ontario multisite implementation). The **American Academy of Nursing Choosing Wisely** recommendation is explicit: "*Don't let older adults lie in bed or only get up to a chair during their hospital stay.*"
5. The **AGS White Paper on mobility assessment** argues for **daily measurement of mobility as a vital sign** in hospitalised older adults, using tools such as **DEMMI** (de Morton Mobility Index) or **AM-PAC "6-clicks"**, so that immobility becomes visible in the record rather than invisible.
6. Timelines to quote: functional decline is measurable by **day 2** of admission; **10 days of bed rest in healthy older adults** produces significant loss of lean mass and strength (Kortebein); recovery takes far longer than the decline.
7. **WHO ICOPE, 2nd edition (2025)** makes **locomotor capacity** one of the six intrinsic-capacity domains screened at primary-care level, with **multicomponent exercise** as the core intervention – moving immobility management upstream into the community.

Current Practice Update

Domain	Textbook position	Current practice (2023–present)
Concept of bed rest	"The bed is a very dangerous place for an older	Operationalised into ward-level campaigns (**End PJ**

	person"; complications listed	**Paralysis**) with measurable targets, and formally named **hospital-associated deconditioning**
Scope of harm	Physical deconditioning, with delirium listed separately	**Physical and cognitive deconditioning as one continuum** (2024 New Horizons paper)
Delivery model	Referral to physiotherapy; restorative philosophy of care	**Frailty Care Bundle**: mobilisation + nutrition + cognitive engagement, delivered by the whole ward team, not by therapists alone
Measurement	Functional status assessed at admission and discharge	**Daily mobility measurement** (DEMMI, AM-PAC 6-clicks) as a vital sign
Dementia	Little specific guidance	2025 meta-analysis supports progressive mobilisation for **hospitalised people with dementia**
Restraints	Reduce use; safe elimination is possible	**Restraint-free care** as the default standard, with accreditation-level audit indicators; chemical restraint scrutinised as closely as physical
Community	Exercise for prevention	**WHO ICOPE 2nd ed. 2025** screens locomotor capacity in primary care and triggers multicomponent exercise

Indian Context

1. **LASI Wave-1 (2017–18)**: roughly **one in four** Indians aged 60 and above report difficulty with at least one ADL, and close to **half** report difficulty with at least one IADL – the population burden of immobility is therefore enormous and largely community-based.
2. **India Ageing Report 2023 (UNFPA–IIPS)**: the 60+ population, about 10.5% in 2022, is projected to reach roughly 20.8% by 2050 – immobility and disability will scale accordingly.
3. **NPHCE** district geriatric units and regional geriatric centres provide physiotherapy and rehabilitation, but coverage is thin below district level; the family remains the main mobilising agent.
4. **Rashtriya Vayoshri Yojana** and **ADIP–ALIMCO** supply walking sticks, tripods, walkers, wheelchairs and commodes free to eligible senior citizens – the single most practical intervention against environmental immobility in a poor household.
5. Culture-specific contributors worth naming in an Indian answer: the strong belief that a sick elder should be given complete rest; floor-level living and squat toilets that become impossible with knee osteoarthritis; absent handrails and wet, slippery bathroom floors; and adult children who take over all tasks as an expression of respect, producing rapid deconditioning.
6. The **Maintenance and Welfare of Parents and Senior Citizens Act, 2007** is relevant where neglect, confinement or restraint of an immobile elder is suspected.

Rapid Recall Box — Recurring Topic

Mnemonic for complications – "BEDREST":
Bone loss – disuse osteoporosis and hypercalcaemia · **E**mbolism – DVT and pulmonary embolism · **D**ecubitus ulcer · **R**etention of urine, constipation and impaction · **E**rosion of muscle – atrophy, weakness and contractures · **S**ensory deprivation → delirium and depression · **T**oppling blood pressure – orthostatic hypotension (plus atelectasis and aspiration pneumonia).

Numerical anchors: one-third of hospitalised elders confined to bed or chair · 15–76% decline in at least one ADL at discharge · only 50% of those recover by three months · decline measurable by day 2 · 10 days of bed rest causes measurable loss of lean mass and strength · roughly 1–1.5% of muscle strength lost per day of strict bed rest (classical teaching figure) · LASI: ~25% ADL and ~48% IADL difficulty in 60+.

Examiner traps: (i) Listing only physical complications and forgetting **delirium, depression and hypercalcaemia**. (ii) Forgetting that **chemical restraint (antipsychotics) counts as a cause of immobility**. (iii) Missing the **caregiver-generated** immobility of the over-helpful family. (iv) Not naming **Tinetti's spiralling immobility**. (v) Treating deconditioning as inevitable ageing rather than a preventable, largely iatrogenic syndrome. (vi) Omitting fear of falling and health beliefs.

15-second answer skeleton: Define immobility as restriction of time out of bed by orders, devices, drugs or lack of help → one-third of inpatients affected, half never recover → causes by system: musculoskeletal, neurological, cardiopulmonary, sensory, drugs, psychological, environmental → complications by system using BEDREST → assessment: perceived cause, drugs, pain, mood, nutrition and vitamin D, environment, strength and gait → management: treat cause, restorative philosophy, progressive exercise, prevent complications, eliminate restraints → 2024 hospital-associated deconditioning of body and mind, Frailty Care Bundle, End PJ Paralysis, ICOPE 2025 → Indian context.

Exam One-Liners

1. Immobility is restriction of time out of bed or chair by orders, devices, chemical restraint, absent aids or absent human assistance.
2. Up to one-third of hospitalised older patients are restricted to bed or chair rest.
3. Only half of those who decline functionally in hospital recover by three months.
4. Contracture describes fixation of both muscle and joint, and is the hallmark consequence of immobility in long-term care.
5. Immobility causes negative nitrogen and calcium balance and impaired glucose tolerance.
6. Disuse osteoporosis from immobility can produce hypercalcaemia.
7. Reduced plasma volume plus deconditioned reflexes produces orthostatic hypotension.
8. Spiralling immobility (Tinetti and Ginter): restraining a faller makes them permanently unable to walk.
9. Antipsychotics act as chemical restraints and are a direct cause of immobility.
10. Deconditioning is measurable by day two of hospital admission.
11. Hospital-associated deconditioning is now understood as a condition of both body and mind.
12. The Frailty Care Bundle combines mobilisation, nutrition and cognitive engagement.
13. Choosing Wisely: do not let older adults lie in bed or only get up to a chair during their hospital stay.
14. WHO ICOPE 2nd edition (2025) screens locomotor capacity as one of six intrinsic-capacity domains.

Question 4. Rehabilitation team [7 MARKS]

High-Yield Points

1. **Rehabilitation** is a goal-directed, time-limited, multidisciplinary process that reduces **impairment**, **activity restriction** and **participation restriction**, and restores the person to the highest function their condition allows. **A team approach is essential, not optional.**
2. A properly resourced team has input from **medical and nursing staff, physiotherapists, occupational therapists, speech and language pathologists, clinical psychologists, dietitians and social workers**. Broadly, **doctors manage impairment, remedial therapists manage activity restriction, and social workers manage participation restriction**; **nurses hold a holistic brief that influences both activity and participation**.
3. **Doctor / geriatrician / physiatrist**: identifies and manages the presenting problem and coexisting impairments, minimises underlying risk factors and anticipates complications. In a patient with embolic stroke, for example, the doctor monitors anticoagulation, controls hypertension and manages coexisting angina and diabetes, while also preventing shoulder subluxation through correct early handling.
4. **Nurse**: delivers the therapy plan across 24 hours – skin integrity, continence, nutrition, medication, sleep, safety, education and encouragement – and is the member who converts an hour of therapy into a whole day of function.
5. **Physiotherapist**: provides therapies targeting specific problems and enhancing **cardiorespiratory, neuromuscular and locomotor function**; advises on and trains the use of **mobility aids**.
6. **Occupational therapist**: assesses and enhances competence with **ADLs and IADLs**; advises on aids and adaptations; performs the **pre-discharge home visit** so that modifications are appropriate and timed to facilitate discharge.
7. **Speech and language therapist**: manages **communication disorders** (dysphasia, dysarthria) and **swallowing disorders**, and advises on communication aids.
8. **Other members**: clinical psychologist (mood, motivation, adjustment to loss, cognition), dietitian, podiatrist, continence advisor, audiologist, pharmacist, and – in well-resourced centres – **prosthetists and orthotists**, who design, construct and fit prostheses (replacing body parts) and orthoses (supporting, improving or restricting movement).
9. **The patient and family are members of the team.** All members, **and particularly the patient**, must be involved in setting goals, with general agreement on their validity; a programme is seriously compromised if key people differ on what is being attempted. Set **short-term (intermediate) goals leading to long-term (final) goals**; patients with **ambitious goals make greater progress** than those with modest ones, so set targets that are ambitious but achievable, with flexible timeframes reviewed at least weekly.
10. **Communication is what makes a group of professionals a team.** Co-location allows regular informal exchange; in addition, teams hold **formal meetings to discuss progress, revise goals, plan discharge and organise community follow-up**. The **interdisciplinary team conference**, held after individual assessments are complete, is critical – it generates new ideas, sets priorities, disseminates findings and avoids duplication. Teams mature through the **forming, storming, norming and performing** phases, which takes commitment, skill and time.

Latest Updates (2023–present) — three key points

1. **WHO Rehabilitation 2030** and the **World Rehabilitation Alliance**, backed by the World Health Assembly resolution on strengthening rehabilitation in health systems, establish rehabilitation as an **essential health service to be integrated into universal health coverage** rather than an optional add-on. The WHO **Package of Interventions for Rehabilitation (PIR)** gives condition-specific team compositions and interventions that health systems are expected to fund.
2. **WHO ICOPE, 2nd edition (2025)** requires a single **integrated, person-centred care plan** built around the six domains of intrinsic capacity, delivered by a multidisciplinary team working at primary-care level – effectively exporting the rehabilitation team model out of the hospital and into the community.
3. India: the **National Commission for Allied and Healthcare Professions Act, 2021** created statutory registration, standardised curricula and defined scope of practice for physiotherapists, occupational therapists, speech-language pathologists and other allied professionals – a structural precondition for functioning rehabilitation teams. Tele-rehabilitation and hybrid team review are now accepted delivery modes.

Current Practice Update

Domain	Textbook position	Current practice
Status of rehabilitation	A specialist service for selected patients	**An essential health service within UHC** (WHO Rehabilitation 2030; WHA resolution)
Goal setting	Realistic, ambitious, patient-involved, reviewed weekly	Same principles, formalised as **SMART goals with shared decision-making** and documented patient-stated priorities
Team meeting	Regular formal case conference	Daily board rounds and digital huddles alongside the weekly conference; family joins remotely

Setting	Ward, unit, nursing home or home	Adds **tele-rehabilitation** and hospital-at-home models for post-discharge continuity
Workforce (India)	Unregulated allied health professions	**NCAHP Act 2021** – statutory councils, registration and defined scope of practice

Indian Context

Complete rehabilitation teams exist mainly in medical college PMR departments and large private hospitals. Under **NPHCE**, regional geriatric centres and district geriatric units are mandated to provide physiotherapy and rehabilitation services. In most district settings the "team" is realistically the **geriatrician or physician, one physiotherapist, a nurse, and the family caregiver** – so a good Indian answer states the ideal composition and then describes how the core functions are delivered with fewer people. Community delivery works through **ASHA and ANM home visits**, the **Kerala ward-level palliative home-care team** (nurse plus trained volunteers), and **Vayomithram**. Devices come through **ADIP–ALIMCO** and **Rashtriya Vayoshri Yojana**; the **Rehabilitation Council of India** governs training in disability rehabilitation.

Exam One-Liners

1. Rehabilitation reduces impairment, activity restriction and participation restriction.
2. Doctors address impairment, therapists address activity restriction, social workers address participation restriction.
3. Nurses hold a holistic brief influencing both activity and participation restriction.
4. Occupational therapists lead the pre-discharge home visit.
5. Orthoses support or restrict movement; prostheses replace a body part.
6. Patients with ambitious goals make greater progress than those with modest goals.
7. Short-term intermediate goals in succession deliver the long-term final goal.
8. The interdisciplinary team conference after individual assessment is the critical step in geriatric assessment programmes.
9. Teams evolve through forming, storming, norming and performing.
10. WHO Rehabilitation 2030 positions rehabilitation as an essential service within universal health coverage.

Question 5. Role of physiotherapy in the elderly [7 MARKS]

High-Yield Points

1. **Objectives** of physiotherapy in the older patient (Kane's): **relieve pain**; **evaluate, maintain and improve joint range of motion**; **evaluate and improve strength, endurance, motor skills and coordination**; **evaluate and improve gait and stability**; and **assess the need for, and teach the use of, assistive devices for ambulation** – wheelchairs, walkers and canes. Pathy's frames the same role as enhancing **cardiorespiratory, neuromuscular and locomotor function**.
2. **Exercise modalities**: active exercise (isometric and isotonic), passive exercise for the immobile or paralysed limb, and structured **sitting exercise programmes** for those who cannot stand.
3. **Physical modalities**: heat – hot packs, paraffin wax, diathermy; **ultrasound**; **hydrotherapy**; and **transcutaneous electrical nerve stimulation (TENS)** for pain. Note that hydrotherapy improves self-confidence but has not been shown to produce measurable functional improvement in arthritic joints.
4. **Falls, gait and balance**: lower-extremity strength training, balance training including **tai chi**, gait retraining, properly fitted footwear, and **habituation exercises** for benign paroxysmal positional vertigo. Physiotherapy is one of the named treatments for gait and balance disorders, muscle weakness and deconditioning in the falls algorithm.
5. **Musculoskeletal disease**: **daily range-of-motion exercise** is particularly important for restoring function in arthritic joints, though compliance is poor at all ages – **group activity adds a social component and improves compliance**. Age-related stiffening of joints and soft tissues is **reversible**: exercise increases joint flexibility and improves the strength, size and resilience of cartilage, ligaments and muscle.
6. **Prevention of the complications of immobility**: chest physiotherapy and breathing exercises, upright positioning to prevent atelectasis and aspiration, positioning and proper handling to prevent contractures and **shoulder subluxation in flaccid hemiplegia**, and mobilisation to prevent DVT and pressure injuries. Preoperative training in cough and lung expansion techniques, with chest physiotherapy for those at pulmonary risk, is part of standard anaesthetic preparation of the elderly.
7. **Neurological rehabilitation**: even severely affected stroke patients achieve meaningful functional gains with early rehabilitation, and **immobility and its complications can almost always be prevented or minimised**. **Treadmill training has repeatedly shown significant benefit for gait and balance in stroke survivors**; robot-assisted motor training has shown some positive benefit. Ongoing exercise after the acute phase maintains function, preserves quality of life and helps prevent recurrent stroke.
8. **Pulmonary and cardiac rehabilitation**: in COPD, pulmonary rehabilitation **reduces dyspnoea and fatigue, increases exercise capacity, enhances patient control of the disease, improves quality of life and may prevent hospitalisation**. Benefits are similar in all adults **including the "old old"**, and are **greatest in the most impaired patients**. Patients with asthma and interstitial disease benefit too. Even those hypoxic at rest or desaturating on exertion can be trained safely with supplemental oxygen.
9. **Dementia and frailty**: physical activity should be at **moderate intensity for 30 minutes five times a week, or vigorous intensity for 25 minutes three times a week**. Walking combined with strength, balance and flexibility exercise slows functional decline, increases gait speed, improves mood and sleep and reduces agitation. Compliance – more than cognitive status – is the limiting factor, so programmes must be **enjoyable, individualised, accessible, and delivered with conversation or music**, with staff communicating with the resident during the session.
10. **Safety and setting**: current guidance **no longer recommends routine stress testing before starting physical activity**; low-intensity activity can be safely initiated in asymptomatic sedentary older people regardless of recent medical evaluation, because **sedentary behaviour creates more risk than moderate physical activity**. Physiotherapy may be delivered in acute hospital, rehabilitation unit, skilled nursing facility, outpatient clinic or at home, individually or in groups depending on preference, cognition and motivation.

Latest Updates (2023–present) — three key points

1. **WHO physical activity guidance for adults aged 65 and over** is now the reference prescription: **150–300 minutes of moderate aerobic activity per week**, **muscle-strengthening on at least 2 days**, and – specifically for older adults – **multicomponent activity emphasising balance and strength on at least 3 days per week to prevent falls and functional decline**. "Every move counts"; some activity is better than none, and sedentary time should be limited.
2. **In-hospital exercise reverses the functional decline of acute admission.** The Martínez-Velilla multicomponent exercise trial (*JAMA Internal Medicine* 2019) remains the anchor; 2024–25 evidence extends it – a **2025 European Geriatric Medicine meta-analysis** supports progressive mobilisation in **hospitalised people with dementia**, and service evaluations show **functional resistance training on medical wards** produces significant gains in grip strength, 30-second sit-to-stand and DEMMI scores.
3. **Tele-rehabilitation and technology-assisted programmes** are now evidence-supported for post-discharge deconditioning (smartphone-delivered resistance and balance programmes for patients on physiotherapy waiting lists) and for delivering **Otago-based fall-prevention programmes** in early geriatric rehabilitation using digital trainers with real-time correction.

Current Practice Update

Domain	Textbook position	Current practice
Exercise prescription	Moderate 30 min × 5/week or vigorous 25 min × 3/week	WHO 65+: **150–300 min/week moderate + strengthening ≥2 days + balance-focused multicomponent ≥3 days**
Pre-exercise screening	Routine stress testing no longer recommended	Unchanged and reinforced – sedentary behaviour is the greater risk
Acute hospital	Rehabilitation begins after the acute illness settles	**Exercise during the acute admission itself** reverses functional decline; delivered from day 1
Dementia	Compliance-limited, benefits uncertain	2025 meta-analysis supports progressive mobilisation and multicomponent programmes in hospitalised dementia
Delivery mode	Face-to-face, institution or home	Adds **tele-rehabilitation** and digital/sensor-assisted training with real-time feedback

Indian Context

Physiotherapy is now a **statutorily regulated profession under the NCAHP Act, 2021**. Under **NPHCE**, physiotherapy is a mandated component of district geriatric units and regional geriatric centres, though rarely available below district level; the **Kerala palliative home-care network** provides one of the few functioning home-physiotherapy models. Practical, low-cost and culturally acceptable prescriptions carry most of the benefit: **chair-based exercise, supervised walking groups in temple or community grounds, stair and squat avoidance advice for knee osteoarthritis, thera-band resistance work, and yoga adapted for balance** (with caution about floor-level postures in the frail). Assistive devices – sticks, tripods, walkers, wheelchairs – are supplied free through **Rashtriya Vayoshri Yojana** and **ADIP–ALIMCO**. Counsel the family explicitly that **rest is not treatment**, since the belief that an unwell elder must be kept in bed is the commonest barrier to physiotherapy in Indian homes.

Exam One-Liners

1. The five physiotherapy objectives: relieve pain, improve range of motion, improve strength and endurance, improve gait and stability, and prescribe and teach assistive devices.
2. TENS, ultrasound, paraffin wax, diathermy, hot packs and hydrotherapy are the classic physical modalities.
3. Hydrotherapy improves self-confidence but not measurable joint function.
4. Treadmill training repeatedly improves gait and balance after stroke.
5. Pulmonary rehabilitation benefits are greatest in the most impaired patients, including the old old.
6. Age-related joint stiffening is reversible with exercise.
7. Group exercise improves compliance by adding a social component.
8. Routine stress testing is not required before starting low-intensity activity in asymptomatic older adults.
9. In dementia, compliance limits exercise programmes more than cognitive status does.
10. WHO 65+ prescription: 150–300 min moderate activity weekly, strengthening twice weekly, balance work three times weekly.

Question 6. Role of the occupational therapist in the life of an elderly person with mobility issues and an ageing spouse [15 MARKS]

> **How to attack this question:** it has **two halves** – the elder with mobility restriction, and the ageing spouse who is simultaneously the carer and a frail older person in their own right. Answers that describe only grab bars and reachers lose half the marks. Pathy's asks explicitly, as part of the OT assessment: "*Does the carer also need OT intervention in order to carry out their support effectively?*" That sentence is the spine of this answer.

High-Yield Points

The occupational therapy frame of reference

1. Occupational therapy focuses on the elder's **ability to participate in the activities that form their life and are important to them**, not on the impairment itself. Its three aims are to **collaborate with the older adult to plan client-centred care**, **tailor approaches to that person's activity needs**, and **address continued participation in society in a way that is achievable and meaningful**.
2. Assessment uses the **Person–Environment–Occupation–Performance (PEOP)** framework: intrinsic **person** factors (physiological, cognitive, neurobehavioural, psychological, spiritual) interacting with the extrinsic **environment** (social systems and support, economic support, culture and values, built environment and technology, natural world), across **occupations** (basic ADLs, IADLs, leisure, rest, work or service, education) and **roles** (spouse, parent, grandparent, home manager, lifelong learner, organisation member, volunteer).

Assessment — of the elder and of the spouse

3. The **occupational profile** is built by interviewing the patient and the family and asking five questions: (i) **what roles, routines, habits and new activities does the person want to perform** – the elder's own priorities; (ii) **what can be done to facilitate the activities they do perform**; (iii) **what are they currently able to perform** – intrinsic ability; (iv) **if they rely on a support system, what supports are provided and to what extent**; and (v) **does the carer also need OT intervention in order to carry out that support effectively?**
4. The assessment takes into account age, gender, socioeconomic background, sexual orientation, racial, ethnic, cultural and religious background, developmental status, health history and current status, and educational and vocational history – then combines interview with standardised, evidence-based performance measures.
5. **Home evaluation**: OT personnel evaluate the home to establish **PEOP compatibility** – comparing the elder's activity needs and priorities against their functional abilities and the physical environment in order to **predict the likelihood of the activity actually being performed**. The deficiencies found become the intervention targets. **These needs are frequently overlooked or underestimated at the point of discharge, and that gap puts the older adult at increased risk of dependency, falls and injury.**

Intervention for the elder with mobility restriction

6. **Remediation of sensorimotor deficits**: sitting and standing balance, strengthening, endurance, range of motion and coordination – but always **related back in the intervention process to ADL and IADL participation** and to the tasks and roles that are meaningful to that individual, rather than trained as abstract exercise.
7. **Retraining in functional occupations**: basic ADLs (transfers, toileting, bathing, dressing, grooming, feeding) and IADLs (home management, cooking, shopping, laundry, medication and money management, use of public transport, driving skills, use of the telephone and smartphone).
8. Where oral-motor dysfunction, muscular rigidity, joint pain or bradykinesia impede direct intervention – as in Parkinson's disease, stroke or dementia – OT practitioners use **facilitatory techniques and positioning approaches** to enable participation in meaningful activity that simultaneously achieves the mobilisation goal.
9. **Health promotion and prevention teaching**: **body mechanics, joint protection, energy conservation, activity and exercise guidelines, sleep hygiene** for physical deterioration; and for psychosocial deterioration – adjusting lifestyle to accommodate age-related and role changes, the value of purposeful and balanced activity, overall time management, self-esteem and mastery strategies, interpersonal and socialisation skills, and emphasis on the positive aspects of living.
10. **Physical environment adaptation**: additional lighting, railings, ramps, grab bars, and assistive devices to promote accessibility and independent mobility. Concrete measures from Pathy's case examples include an **elevated toilet seat with attached rails, a tall shower stool, a handheld shower, grab bars in the shower**, and **extensions on the legs of a favourite chair** so the person can sit and rise unaided. Furniture height for sitting, eating and hobbies is assessed deliberately.
11. **Technological aids**: low-technology – long-handled reaching device, sock aid, long-handled shoehorn, tap turners, zipper pulls, sock pulls, stretch laces, Velcro fasteners, bath bench; and high-technology – low-vision aids, hearing assistive devices, voice-activation software, adapted computers and smartphones. **Devices are recommended only if they support improved participation in activities identified as crucial by the elder and their family.** An aid can foster dependence rather than independence if it is given without this reasoning.
12. **Cognitive and psychological compensation** where dementia or stroke coexists: simplifying and organising the

environment (mounting **pictures of clothing types on dresser drawers and photographs on kitchen cupboards** so items can be associated with locations), establishing a **predictable daily routine**, orientation and safety cues, and step-by-step strategies for relearning problematic sequences such as transfers and dressing.

Intervention directed at the ageing spouse

13. The ageing spouse is simultaneously the principal support system and **an older adult with their own arthritis, visual and hearing loss, cardiac limitation and reduced strength**. OT formally assesses whether the transfers and personal care being asked of them are **physically safe and sustainable** – and whether the spouse themselves needs intervention.
14. **Carer skills training**: safe transfer and handling technique, correct use of the bath bench, transfer board or hoist, back care and body mechanics, energy conservation, and how to **prompt and supervise rather than do** – because performing tasks for an elder who retains the underlying capability produces deconditioning and immobility. Pathy's lists **family, carer and staff skills and coping** explicitly among geriatric OT services.
15. **Reduce the carer's load through equipment and layout rather than effort** – raising bed and chair heights, positioning items within reach, organising clothes on lower shelves and by colour groups, and installing rails at the points of greatest strain. This is what protects an ageing spouse from musculoskeletal injury.
16. **Carer wellbeing as an explicit goal.** In Pathy's stroke case, the OT worked with the wife **to help her realise more success and improved coping with her husband's condition, in addition to having more time for the activities that were important to her** – that is, preserving the spouse's own occupations, roles and identity, not merely her caring capacity.
17. **Dyadic education and community linkage**: both partners are included in education and goal setting; the couple is connected to local resources – in Pathy's dementia case the family was informed of the support provided by the local Alzheimer's Association – and to day services, respite, senior centres and support groups.
18. **Anticipatory planning**: what happens when the spouse can no longer sustain the care. Graded introduction of paid help, day care or supported living is discussed early rather than in crisis, alongside advance care planning and end-of-life activities, which Pathy's lists within the scope of geriatric OT.

Review, outcome and evidence

19. The OT process is a loop: **patient identification → occupational profile → needs, problems and priorities → occupational performance assessment → collaborative goal setting → occupation-based intervention → ongoing assessment and refinement → final assessment → discharge**, with continuing input from the elder, carers and family at every review. Outcome assessment determines the extent to which targeted goals were met and guides future therapy or discontinuation.
20. **Evidence base**: the landmark **Well Elderly / Lifestyle Redesign trial (Clark *et al.*, JAMA 1997)** showed that preventive occupational therapy for independent-living older adults improved health, function and quality of life, with benefits sustained on long-term follow-up. The emphasis on participation in meaningful occupation promotes **cost-effective care, individual competence and optimal quality of life**.

Latest Updates (2023–present)

1. **CAPABLE (Community Aging in Place, Advancing Better Living for Elders)** is the flagship model: an **occupational therapist plus a nurse plus a small home-repair budget**, delivered over about 10 visits, targeting "what matters" to the older adult. It **delays disability, prevents hospitalisation and avoids institutionalisation**. A 2025 real-world implementation through an area agency on ageing (90 participants) reported **69% completion**, a mean of **4.5 self-identified goals** per person, home modification costs averaging about **US$943**, and the commonest goal areas as **bathing, functional mobility and pain** – directly relevant to the scenario in this question.
2. **Dyadic occupational therapy in dementia**: the Dutch **COTiD** programme (Graff, BMJ 2006) produced large gains in ADL performance and in **carer sense of competence**. The UK replication, **COTiD-UK / VALID (PLoS Medicine 2021)** – 10 hours of OT over 10 weeks in the person's home – did **not** reproduce the effect on the primary ADL outcome, partly because usual care had improved. The practical lesson is that dyadic OT should be offered as a **person- and carer-centred programme with realistic expectations**, not as a guaranteed ADL cure. Ongoing trials (2024–27) are testing COTiD for recurrent falls after discharge in older people with neurocognitive disorders.
3. **Home modification delivered by occupational therapists reduces falls in high-risk community-dwelling older adults by around 39%**, and a 2024 randomised trial confirmed benefit specifically in **older adults with dementia**. A 2025 scoping review broadens the outcome set beyond falls to participation, quality of life and **relatives' and carers' outcomes**.
4. **Reablement and Lifestyle Redesign** – short-term, goal-directed OT to restore functional independence, and occupation-based redesign of daily routines – are now mainstream international models. A **2025 AJOT scoping review** of home-based OT for community-dwelling older adults calls for standardised outcome measurement, integration of **remote and tele-OT approaches**, and **culturally tailored programmes** – the last point directly applicable to India.
5. Carer-focused assessment tools have matured: the **I-HOPE Assist** (In-Home Occupational Performance Evaluation for

Providing Assistance) is designed specifically to capture the **informal caregiver's** perspective on the tasks they assist with and the barriers they face – precisely the ageing-spouse dimension of this question.

Current Practice Update

Domain	Textbook position	Current practice (2023–present)
Unit of care	Elder-centred, carer involved where indicated	**The dyad is the unit of care**; carer-specific assessment tools (I-HOPE Assist) and dyadic programmes (COTiD)
Service model	Assessment, aids, adaptation, review	**CAPABLE** – OT + nurse + funded home repairs against self-identified goals; **Reablement** as a short-term goal-directed model
Home modification	Recommended on OT judgement	Evidence-graded: ~**39% fall reduction** in high-risk elders; RCT support extended to dementia
Dementia dyads	Community OT shown effective (COTiD)	**VALID/COTiD-UK did not replicate** the ADL benefit – offer with realistic expectations and carer-focused goals
Delivery	Home visits and clinic sessions	Adds **tele-OT**, remote coaching and smart-home/assistive technology
Equity	Culture noted as a background factor	Explicit call for **culturally tailored OT programmes** and standardised outcome measures (AJOT 2025)

Indian Context — and practical adaptations for this scenario

1. **Workforce and regulation**: occupational therapy is now statutorily regulated under the **NCAHP Act, 2021**; the **All India Occupational Therapists' Association** is the professional body. Services remain concentrated in medical colleges, PMR departments and metropolitan private hospitals, so in most districts the OT role is discharged in part by physiotherapists, nurses and trained volunteers – state this openly and describe the task-shifted model.
2. **Assistive devices** are supplied free or subsidised through **Rashtriya Vayoshri Yojana** and **ADIP–ALIMCO** – walking sticks, tripods, walkers, wheelchairs, commodes, spectacles, hearing aids and dentures. **AB-PMJAY / Ayushman Vay Vandana Card** covers hospitalisation for the 70+ group.
3. **Home modifications that matter in an Indian home**: converting or fitting a **raised commode seat over an Indian squat toilet** with a grab bar; anti-skid mats and a grab bar in the perpetually wet bathroom; a bathing stool and mug rather than a shower; raising a floor-level mattress onto a correct-height cot; ramping the two or three steps at the entrance; removing loose rugs and door thresholds; brighter lighting on stairs; keeping the kitchen at standing height for an elder who can no longer squat.
4. **The ageing spouse in the Indian setting** is often an older woman with knee osteoarthritis and osteoporosis, doing the cooking, bathing and transfers alone while adult children work elsewhere or have migrated. The realistic OT plan is **equipment plus task-sharing plus respite**, not exhortation. Link to **ASHA and ANM home visits**, the **Kerala ward-level palliative home-care team** of nurse plus trained volunteers, **Vayomithram** mobile clinics, **Kudumbashree** neighbourhood groups, day-care centres under NPHCE and the state Old Age Homes scheme for respite.
5. **Legal and social safety net**: the **Maintenance and Welfare of Parents and Senior Citizens Act, 2007** (as amended) creates an enforceable duty of maintenance on children and relatives, and Maintenance Tribunals provide a remedy where an ageing couple is left without support – a legitimate and often-missed point in a question about an elderly couple caring for each other alone.
6. **Cultural calibration**: respect for elders in Indian families often expresses itself as doing everything for them, which accelerates deconditioning. The most important single piece of family education is **"prompt, supervise and stand by – do not take over"**.

Exam One-Liners

1. Occupational therapy targets participation in meaningful occupation, not the impairment itself.
2. PEOP: Person, Environment, Occupation, Performance – the framework for OT assessment.
3. The occupational profile asks what the elder wants to do, currently does, can do, what support is provided, and whether the carer also needs intervention.
4. OT evaluates the home to predict whether a desired activity can actually be performed there.
5. Environmental needs are commonly underestimated at discharge, raising the risk of dependency, falls and injury.
6. An aid can foster dependence rather than independence – prescribe it only if it improves valued participation.
7. Photographs mounted on drawers and cupboards help a person with dementia locate items independently.
8. Chair-leg extensions, a raised toilet seat with rails, a tall shower stool and a handheld shower are the standard home package after hip surgery.

9. Teach the carer to prompt and supervise rather than take over, because doing tasks for a capable elder causes deconditioning.
10. Carer wellbeing includes freeing time for the carer's own valued activities, not just improving their caring capacity.
11. Clark's Well Elderly trial (JAMA 1997) showed preventive OT improves health, function and quality of life in independent-living elders.
12. CAPABLE combines OT, nursing and a small home-repair budget to delay disability and prevent institutionalisation.
13. OT-delivered home modification reduces falls in high-risk community-dwelling older adults by around 39%.
14. COTiD-UK (VALID, 2021) failed to replicate the ADL benefit of the original Dutch dyadic OT trial.

Question 7. How would you deal with constant itching (pruritus) in an elderly patient? [15 MARKS]

High-Yield Points

Definition and why old skin itches

1. **Chronic pruritus** is itch lasting more than **six weeks**. It is one of the commonest and most under-treated complaints of old age, disturbing sleep, causing irritability, low mood, excoriation and secondary infection, and materially reducing quality of life.
2. **Mechanism in ageing skin**: **epidermal barrier dysfunction** – thinning of the epidermis, slower regeneration of keratinocytes and fibroblasts, and reduced function of sebaceous and sweat glands. The result is a reduced ability to maintain an emolliated barrier, so the skin cannot retain water. This produces **xerosis, the single commonest cause of itch in the elderly**, and is further aggravated by **thyroid dysfunction and polypharmacy**. Immunosenescence and neuronal ageing add further mechanisms.

Causes — approach the question by category

3. **Dermatological**: **asteatotic eczema (eczema craquelé)** – xerotic skin with thin overlying scale and superficial fissures crossing in a pattern resembling a **dried riverbed**, from reduced intercellular lipids; **seborrhoeic dermatitis** (pruritic, well-demarcated yellow-red patches with greasy scale on scalp, eyebrows, nasolabial folds, ears, chest and flexures, driven by *Malassezia furfur*, and more prominent in stroke, epilepsy and Parkinson's disease); **stasis dermatitis** from venous hypertension; contact and allergic dermatitis; psoriasis; lichen simplex chronicus.
4. **Infestation – always exclude scabies first.** Nocturnal itch, burrows in finger web spaces, wrists, axillae, waist and genitalia, and **multiple affected members in the household or institution**. **Crusted (Norwegian) scabies** in the frail, bedbound or immunosuppressed presents with heavy scaling and **surprisingly little itch**, and is highly contagious. Also consider candidiasis, dermatophytosis, pediculosis and the prodrome of herpes zoster.
5. **Bullous pemphigoid** – the commonest autoimmune blistering disorder over the age of 60, with antibodies to **BPAG1 (BP230)** and **BPAG2 / collagen XVII (BP180)**. Its presentation is **polymorphic**: it may appear for months as **intractable pruritus, an eczematous dermatitis or urticaria before any blister forms**. Classic form: symmetrical **tense bullae** on lower trunk and flexures with a **negative Nikolsky sign** (distinguishing it from pemphigus vulgaris); oral mucosa involved in up to one-third. Strongly associated with **Parkinson's disease, dementia, cerebrovascular disease and epilepsy** – a cardinal geriatric association.
6. **Systemic causes**: chronic kidney disease (uraemic pruritus), cholestasis and primary biliary cholangitis, **thyroid dysfunction**, diabetes mellitus, iron deficiency, **polycythaemia vera** (aquagenic itch after bathing), **lymphoma** – note that a **generalised or refractory asteatotic eczema has been associated with underlying systemic lymphoma** – other malignancy, and HIV.
7. **Drugs** – a major and reversible cause in the polypharmacy-laden older patient: opioids, statins, ACE inhibitors, amiodarone, hydrochlorothiazide, allopurinol, antibiotics, calcium-channel blockers, and **DPP-4 inhibitors (gliptins), which are specifically linked to drug-induced bullous pemphigoid**.
8. **Neuropathic / neurogenic pruritus** results from damage to a single nerve or group of nerves, causing itch in the skin they innervate. **Notalgia paraesthetica** – a hyperpigmented patch **between the medial scapular borders**, from damage to the **T2–T6** sensory nerves. **Brachioradial pruritus** – itch of the **dorsolateral forearms and elbows** from **C5–C9 compression due to cervical spine degeneration**. Also post-herpetic itch and small-fibre neuropathy.
9. **Psychogenic and perpetuating factors**: anxiety, depression, delusional infestation; and the self-sustaining **itch–scratch cycle**, which continues long after the original trigger has gone.
10. **Anogenital itch** deserves separate mention. In women, **lichen sclerosus** – pale ivory-white plaques with a crinkly atrophic surface, purpura and scarring, destroying normal vulval architecture, with a **3.5–5% risk of squamous cell carcinoma**; treat with **clobetasol propionate 0.05% ointment**, emollients, soap substitutes and irritant avoidance, and biopsy any suspicious, ulcerated or raised area. Also squamous cell hyperplasia and **vulvodynia**. The commonest general diseases causing vulval itch are **diabetes, uraemia and liver failure**; irritants include perfumed soap and washing powder.

Assessment

11. **History**: duration; **timing** (nocturnal itch suggests scabies or cholestasis); distribution – generalised versus localised, and the **"butterfly sign"**, where the unreachable interscapular area is spared, suggesting a systemic rather than skin cause; relation to bathing (polycythaemia); water temperature, soap and bathing frequency; occupational and contact exposures; **a complete drug and supplement chart**; household or institutional contacts; and red flags – weight loss, fever, night sweats, jaundice, lymphadenopathy.
12. **Examination**: inspect the **whole skin** including scalp, web spaces, genitalia and perianal area. Distinguish **primary lesions** (indicating a dermatosis) from the **secondary changes of chronic scratching** – **linear excoriations, excoriated papulonodules (prurigo nodules), well-demarcated lichenified plaques (lichen simplex chronicus), and post-inflammatory hyperpigmentation**. Examine lymph nodes, liver, spleen and thyroid; assess mood, sleep and cognition.

13. **Investigations** when no primary skin disease is evident, in Indian conventional units: haemoglobin (g/dL) and full blood count with differential (eosinophilia, polycythaemia), ESR, fasting and post-prandial glucose (mg/dL), serum creatinine (mg/dL) with eGFR, liver function tests with bilirubin (mg/dL) and alkaline phosphatase, TSH (μIU/mL), serum ferritin (ng/mL) and iron studies, serum calcium (mg/dL), LDH, chest radiograph, and HIV serology where indicated. Add **skin scraping and dermoscopy for scabies mite, KOH mount for fungus**; **skin biopsy with direct immunofluorescence and anti-BP180 ELISA** when bullous pemphigoid is suspected; and **cervical spine imaging** for brachioradial pruritus.

Management

14. **General skin care is the foundation and is frequently curative on its own.** Short, lukewarm – never hot – showers; **soap substitutes or syndet cleansers and moisturising soaps** in place of ordinary soap and detergent; no scrubbing; **emollient or ointment applied liberally within a few minutes of bathing and at least twice daily** to restore the natural barrier function; **urea-, lanolin- or ammonium-lactate-containing creams** for marked xerosis; room humidification and avoidance of excessive room heating; cotton clothing; nails cut short. Educate the older person *and* the carer, since applying emollient to one's own back is often impossible.
15. **Stop the culprit drug** and rationalise polypharmacy using **Beers criteria and STOPP-START**. Suspect gliptins if bullous pemphigoid is in the differential.
16. **Treat scabies presumptively if there is any doubt** – **permethrin 5% cream** applied neck-down overnight and repeated after one week, or **oral ivermectin** (repeat dose at day 7–14; two or more doses with topical keratolytics for crusted scabies). **Treat all household and institutional contacts simultaneously and launder linen and clothing in hot water.** Warn that post-scabetic itch persists for 2–4 weeks and does **not** indicate treatment failure – otherwise the patient is repeatedly and unnecessarily re-treated.
17. **Topical therapy**: **topical corticosteroid ointment** in short courses for inflammatory dermatoses – discuss proper use explicitly, since improper prolonged use perpetuates further thinning of already atrophic skin. **Topical calcineurin inhibitors** (tacrolimus, pimecrolimus) are steroid-sparing and useful on the face and flexures. Antipruritic counter-irritants: **menthol, camphor, pramoxine and topical lidocaine**. **Topical capsaicin** for localised neuropathic itch such as notalgia paraesthetica and brachioradial pruritus.
18. **Systemic therapy**: **antihistamines are far less useful than commonly assumed** outside urticaria, and **sedating antihistamines should be avoided in older people** (Beers criteria – delirium, falls, urinary retention, anticholinergic burden). Preferred agents: **gabapentin or pregabalin** for neuropathic and uraemic itch, dose-reduced for renal function and started low to avoid sedation and falls; **SSRIs such as sertraline or paroxetine, or mirtazapine** for cholestatic, paraneoplastic and nocturnal itch; **naltrexone** (μ-opioid antagonist) for opioid-induced and cholestatic itch; and **narrow-band UVB phototherapy**, which is safe and effective in the elderly, including in renal and idiopathic pruritus.
19. **Cause-specific treatment**: **cholestasis** – cholestyramine, rifampicin, naltrexone, and treat the obstruction. **Chronic kidney disease** – optimise dialysis adequacy, correct hyperphosphataemia and hyperparathyroidism, gabapentin, and **difelikefalin**. **Stasis dermatitis** – leg elevation above heart level for 30 minutes three to four times daily and **compression stockings** after excluding arterial disease, with topical steroid only during acute flares. **Seborrhoeic dermatitis** – topical azole antifungal (ketoconazole daily during a flare, then twice weekly for maintenance), ciclopirox, zinc pyrithione, selenium sulfide or tar shampoo.
20. **Bullous pemphigoid**: potent topical clobetasol for localised disease; oral corticosteroids with or without doxycycline for extensive disease. **Osteoporosis and gastrointestinal ulcer prophylaxis must be given to every older patient started on oral steroids**, and methotrexate must be dosed cautiously because of the **natural 30% decline in glomerular filtration rate with ageing** in addition to other renal insults.
21. **Break the itch–scratch cycle and treat the person**: cool compresses, wet wraps, distraction and habit-reversal training, treatment of coexisting depression and anxiety, and attention to sleep. Refer to **orthopaedics or neurology** in selected neurogenic cases, and to **dermatology** for refractory itch or when pemphigoid or malignancy is suspected.
22. Finally, apply the geriatric wrap-around. **Social support, functional status, comorbidities, mobility and cognition** all determine whether a treatment plan can actually be carried out. Pathy's makes the point squarely: **even a simple intervention to treat a symptomatic lesion or dermatitis can provide relief and positively affect quality of life in the elderly patient.**

Latest Updates (2023–present)

1. **Chronic prurigo / prurigo nodularis has become a treatable disease.** The **IFSI stepwise algorithm** runs: **step 1** – topical corticosteroid, topical calcineurin inhibitor and H1 antihistamine; **step 2** – topical capsaicin, intralesional corticosteroid, UV therapy; **step 3** – gabapentin, pregabalin or antidepressants where neuropathic features predominate, versus ciclosporin or methotrexate where inflammation predominates; **step 4** – NK1-receptor antagonist, μ-opioid receptor antagonists, **dupilumab, nemolizumab**, and thalidomide as a last resort.
2. **Dupilumab** (anti-IL-4Rα, blocking IL-4 and IL-13) was the **first systemic therapy approved for prurigo nodularis**, on the phase 3 **LIBERTY-PN PRIME and PRIME2** trials. Pooled week-24 data: **58.8% versus 19.0%** achieved a clinically meaningful (≥4-point) improvement in itch, **46.4% versus 17.1%** achieved clear or almost-clear skin, and **35.3% versus 8.9%** achieved both. Benefit was independent of baseline demographics and of atopic status.

3. **Nemolizumab**, a first-in-class antibody against the **interleukin-31 receptor α** – IL-31 being the "itch cytokine" of neuroimmune crosstalk – was approved for prurigo nodularis in **2024** on the phase 3 **OLYMPIA 2** trial, with reduction in itch and sleep disturbance as early as **week 4**. A 68-week Japanese phase II/III study showed benefit maintained or increased through 68 weeks with **no rebound after cessation**.
4. **British Association of Dermatologists living guideline for nodular prurigo (2025)**: **offer a licensed biologic (dupilumab or nemolizumab)** to adults with moderate-to-severe disease who are candidates for systemic therapy (strong recommendation); consider **thalidomide only for disease resistant to all other treatment**, with counselling and informed consent; follow up at least once in the following 12 months using DLQI, HADS, GAD-7 and PHQ scores.
5. **Chronic pruritus of unknown origin (CPUO)** is now a named entity – itch beyond six weeks with cutaneous, systemic, neuropathic and psychogenic causes excluded. It is **most prevalent in older adults** (quoted prevalence in older populations ranges widely, up to around 30–45%), and is hypothesised to arise from **age-related polarisation towards a type 2 immune response as a consequence of immunosenescence**. The phase 3 **LIBERTY-CPUO-CHIC Study A (2024) missed its primary itch endpoint at week 24**, but showed nominally significant improvement on essentially every other itch measure (≥4-point WI-NRS response **44.4% versus 23.5%** at week 12) and on itch-related quality of life. A proof-of-concept **nemolizumab trial in CPUO** began in 2025. **The examinable message: there is still no drug licensed for CPUO.**
6. **Abrocitinib**, an oral JAK1 inhibitor, 200 mg daily for 12 weeks in an open-label phase 2 study (*JAMA Dermatology* 2024) reduced peak pruritus NRS by **78.3% in prurigo nodularis and 53.7% in CPUO** – promising, but JAK inhibitors carry **cardiovascular, thrombotic, infective and malignancy warnings** that are particularly weighty in an elderly, comorbid population.
7. **Difelikefalin**, a peripherally acting **kappa-opioid receptor agonist**, is approved for **pruritus associated with chronic kidney disease in adults on haemodialysis**, reflecting the role of opioid-pathway imbalance and kappa-receptor downregulation in uraemic itch. Case reports describe nemolizumab succeeding where difelikefalin has failed, supporting a shared IL-31 mechanism.
8. A **Cochrane review of interventions for CPUO** found only one eligible randomised trial in the entire literature; pragmatically, **serlopitant, pregabalin or dupilumab** are the options considered after first-line therapy fails.

Current Practice Update

Domain	Textbook position	Current practice (2023–present)
Underlying model	Histamine-centric; antihistamines listed as standard oral therapy	**Neuroimmune model** – IL-4/IL-13, **IL-31**, substance P/NK1 receptor, kappa-opioid imbalance. Antihistamines have little role outside urticaria; sedating agents avoided under Beers criteria
Prurigo nodularis	Topical steroid, capsaicin, anaesthetics, antihistamines, gabapentin, antidepressants, phototherapy	**Biologic era**: dupilumab (approved) and nemolizumab (approved 2024) as first-line systemics; **BAD 2025 living guideline** recommends offering a licensed biologic
Idiopathic itch of the elderly	Not separately named	Defined as **CPUO** with an immunosenescence / type 2 polarisation hypothesis; **no licensed drug**; dupilumab phase 3 missed its primary endpoint
Uraemic pruritus	Symptomatic care, gabapentin	**Difelikefalin** licensed for haemodialysis-associated pruritus
Emerging oral agents	Not covered	**JAK1 inhibitors** (abrocitinib) show large itch reductions in PN and CPUO but carry cardiovascular, thrombotic and malignancy warnings in the elderly
Thalidomide	Historically used for refractory prurigo	Restricted – **only for disease resistant to everything else**, with formal counselling (BAD 2025)
Outcome measurement	Descriptive	Standardised: **WI-NRS / PP-NRS**, IGA PN-S, DLQI, HADS – a ≥4-point NRS fall is the accepted clinically meaningful response

Indian Context

1. **Scabies is the single most important "do not miss" diagnosis in the itching Indian elder** – particularly in old-age homes, hospitals, long-term care facilities and crowded joint families, where institutional outbreaks are common. Treat the index case and **every contact simultaneously**; permethrin 5% and oral ivermectin are both cheap and widely available.
2. **Xerosis is aggravated by locally specific habits**: very hot bucket baths, hard water, vigorous use of harsh detergent bar soaps and scrubbing with a loofah, and winter room heating. The culturally acceptable, cheap and effective intervention is **pre-bath and post-bath application of coconut or sesame oil**, or a paraffin-based

emollient, with a syndet or soap substitute – often more successful than prescribing an expensive branded moisturiser.

3. **High background prevalence of the systemic causes**: diabetes mellitus, chronic kidney disease with a large and growing dialysis population, hepatobiliary disease, iron deficiency anaemia (very common in older Indian women), and thyroid disorders. A basic panel – haemoglobin g/dL, glucose mg/dL, creatinine mg/dL, LFT with bilirubin mg/dL, TSH µIU/mL, ferritin ng/mL – will identify most.
4. **Cost realism, which examiners appreciate**: dupilumab and nemolizumab are largely **out-of-pocket and unaffordable** for most Indian elderly, and **AB-PMJAY / Ayushman Vay Vandana Card** covers hospitalisation rather than chronic outpatient biologics. The practical Indian ladder therefore remains **emollients and soap substitutes → treat the cause → topical steroid or calcineurin inhibitor → gabapentin or an SSRI → narrow-band UVB phototherapy**, which is widely available in dermatology departments and is safe in the elderly.
5. **Polypharmacy** is prevalent and poorly documented; ask specifically about over-the-counter analgesics, ayurvedic and traditional preparations, and self-medicated steroid-antifungal combination creams, which are freely sold in India and produce steroid-modified tinea (*tinea incognito*) – a common and easily missed cause of persistent itch.

Rapid Recall Box

Mnemonic for the causes – "SCRATCHED":
Scabies and infestation · **C**holestasis · **R**enal failure (uraemic itch) · **A**naemia (iron deficiency) and polycythaemia · **T**hyroid disease · **C**ancer – lymphoma and paraneoplastic · **H**IV · **E**czema and xerosis (asteatotic) · **D**rugs. Add the three geriatric specials: **bullous pemphigoid, neuropathic itch (notalgia paraesthetica, brachioradial pruritus), and CPUO**.

Numerical anchors: chronic pruritus >6 weeks · notalgia paraesthetica T2–T6 · brachioradial pruritus C5–C9 · lichen sclerosus 3.5–5% risk of squamous cell carcinoma · bullous pemphigoid over age 60, oral involvement up to one-third · 30% age-related fall in GFR (methotrexate caution) · dupilumab PRIME/PRIME2 58.8% vs 19.0% itch response at week 24 · nemolizumab benefit from week 4, sustained to 68 weeks · ≥4-point NRS fall = clinically meaningful response · post-scabetic itch persists 2–4 weeks.

Examiner traps: (i) Reaching for a sedating antihistamine – explicitly inappropriate in the elderly. (ii) Missing **scabies**, especially crusted scabies with minimal itch in the bedbound. (iii) Missing the **pre-bullous phase of bullous pemphigoid** presenting as months of "eczema" or intractable itch. (iv) Forgetting **drug causes**, particularly gliptins. (v) Prescribing long-term topical steroid on already-atrophic skin. (vi) Failing to give **osteoporosis and ulcer prophylaxis** with oral steroids. (vii) Treating the itch without asking whether the elder can physically reach their own back to apply the emollient. (viii) Claiming a licensed drug exists for CPUO.

15-second answer skeleton: Chronic itch >6 weeks → ageing barrier dysfunction makes xerosis the commonest cause → but systematically exclude **scabies, bullous pemphigoid, drugs and systemic disease (SCRATCHED)** → history including timing, distribution, butterfly sign, drug chart, contacts → examine whole skin, separate primary lesions from scratch-induced secondary lesions → targeted bloods in Indian units, scraping, biopsy with DIF, cervical spine imaging → management: emollients and soap substitutes as the backbone, stop the drug, treat scabies presumptively, topical steroid/TCI/capsaicin, then gabapentinoid, SSRI, naltrexone, NB-UVB → cause-specific: difelikefalin for CKD, biologics for prurigo nodularis → break the itch–scratch cycle and treat mood and sleep → 2023–25 updates: IL-31 and nemolizumab, dupilumab, BAD 2025, CPUO with no licensed drug → Indian context: scabies, oil-based emollients, cost, NB-UVB.

Exam One-Liners

1. Chronic pruritus is itch lasting more than six weeks.
2. Epidermal barrier dysfunction of ageing skin makes xerosis the commonest cause of itch in the elderly.
3. Asteatotic eczema resembles a dried riverbed; a generalised or refractory form may signal underlying lymphoma.
4. Bullous pemphigoid may present as intractable pruritus or eczema for months before any blister appears.
5. Bullous pemphigoid has a negative Nikolsky sign and is associated with Parkinson's disease, dementia, stroke and epilepsy.
6. Notalgia paraesthetica: interscapular hyperpigmented patch from T2–T6 sensory nerve damage.
7. Brachioradial pruritus: dorsolateral forearm itch from C5–C9 cervical spine degeneration.
8. Crusted (Norwegian) scabies presents with heavy scaling and minimal itch in the frail or immunosuppressed.
9. Post-scabetic itch lasts 2–4 weeks and does not indicate treatment failure.
10. Sedating antihistamines are inappropriate in older adults under the Beers criteria.
11. Gabapentin and pregabalin are the drugs of choice for neuropathic and uraemic itch.
12. Difelikefalin is a peripherally acting kappa-opioid receptor agonist licensed for haemodialysis-associated pruritus.
13. IL-31 is the "itch cytokine"; nemolizumab blocks its receptor and was approved for prurigo nodularis in 2024.
14. Dupilumab was the first approved systemic therapy for prurigo nodularis (PRIME/PRIME2).
15. CPUO is commonest in older adults and still has no licensed treatment; dupilumab missed its primary endpoint in phase 3.

16. Lichen sclerosus carries a 3.5–5% risk of squamous cell carcinoma and is treated with clobetasol propionate 0.05%.
17. Every older patient on oral steroids for pemphigoid needs osteoporosis and gastrointestinal ulcer prophylaxis.

EXAMINATION ANSWER SERIES

MD GERIATRIC MEDICINE

Ethics & End-of-Life Care · Transitional Care · Sleep Medicine

1. Elder abuse / elder mistreatment ★ **recurring**
2. Advance care planning
3. End-of-life care issues
4. Transitional care home — admission, discharge & staffing
5. Sleep hygiene / pathophysiology of insomnia & management ★ **recurring**
6. Sleep apnoea / polysomnography

Prepared for **Dr. Sudarsan**

Primary texts: *Pathy's Principles and Practice of Geriatric Medicine*, 6th edition | *Kane's Essentials of Clinical Geriatrics*

Supplemented with international and Indian evidence to August 2026

Six 15-mark answers, each with a condensed 7-mark version

CONTENTS

1.	Elder abuse / elder mistreatment ★	15 + 7
2.	Advance care planning	15 + 7
3.	End-of-life care issues	15 + 7
4.	Transitional care home: prerequisites for admission and discharge, and staffing roles	15 + 7
5.	Sleep hygiene / pathophysiology of insomnia and its management ★	15 + 7
6.	Sleep apnoea / polysomnography	15 + 7

★ *Recurring high-yield topic — carries an additional recall box with mnemonic, numerical anchors, examiner traps and a 15-second answer skeleton.*

How each answer is built: numbered high-yield points under labelled subheadings → **Latest Updates (2023-present)** → **Current Practice Update** comparison table → **Exam One-Liners** → condensed 7-mark version.

1. ELDER ABUSE / ELDER MISTREATMENT — 15 MARKS ★ RECURRING

DEFINITION AND CONCEPT

1. **Elder abuse** is any **act or omission** that endangers a person's life, threatens their physical or psychological integrity, impairs their freedom, or compromises their personal development and/or **financial security** (Council of Europe, 1987). WHO adds that it occurs within a **relationship of expectation of trust**.
2. Vulnerable people of any age are the usual targets — especially those with **loss of autonomy and cognitive disorders**. Old age itself is not the risk; **dependency** is.
3. Two forms are distinguished: **individual mistreatment** (an act or omission by a third party against a person) and **institutional abuse** (occurring within an institution as a result of **inadequate governance**).
4. **Neglect must be separated from abuse.** Neglect results from forgetfulness, ignorance and inattention to professional duty — there is **no intent to harm**. Abuse implies intent. Both are harmful and both are reportable.

CLASSIFICATION (COUNCIL OF EUROPE)

5. **Physical violence** — beating, wounding, restraint, mishandling, sexual violence, disregard for pain.
   Psychological violence — familiarity, infantilisation, verbal violence, deprivation of visits.
   Material and financial violence — embezzlement, misappropriation of money or inheritance, abusive power of attorney.
   Medical violence — lack of basic and appropriate care, lack of information about treatment.
   Drug abuse — misuse of sedative and neuroleptic treatment (chemical restraint).
   Violation of rights — identity, freedom of movement, civil rights, religious practice, misuse of legal protection.
6. **Resident-to-resident aggression (RRA)** is a distinct and under-recognised form in long-term care: negative, aggressive and intrusive physical, sexual, verbal or material interactions between residents that would be unwelcome in a community setting. Coroners' data identified **28 RRA-related deaths in Australia (2000–2014)** and **101 fatal incidents in the USA (2003–2016)**.
7. **Psychological control ("right of way")** — a manipulative process using cognitive, affective and behavioural approaches to remove a person's capacity for discernment; it leads to **abuse of weakness**, usually by a relative, neighbour or family member.

EPIDEMIOLOGY

8. **Global** — approximately **1 in 6 (15.7%)** community-dwelling adults over 60 experience abuse each year (Yon et al. meta-analysis). Commonest subtypes: **psychological 11.6%**, **material/financial 6.8%**, neglect ~4%, physical ~2.6%, sexual ~0.9%. In institutions, roughly **2 in 3 staff** admit to committing some form of abuse in the past year.
9. **India — LASI Wave-1**: **5.2%** of those aged 60 and above reported ill-treatment. Highest in **Bihar (~12%)**, Karnataka (10%), West Bengal (8%). Among those ill-treated: **verbal/emotional 77.3%**, **neglect 52.6%**, **economic exploitation 26.5%**, **physical 23.7%**.
10. **HelpAge India 2024** (5,169 elders across 20 Tier-I and Tier-II cities): **7%** reported abuse; the **son was the commonest perpetrator (42%)**, followed by the daughter/daughter-in-law (28%); **73%** of those abused had an annual income below ₹1 lakh.
11. Community and hospital-based Indian studies report far higher figures (**14–45%**), confirming gross **under-reporting** in national surveys. Reasons: family honour, economic dependence, fear of abandonment, cultural expectation of filial care, and unawareness of the law.

RISK FACTORS

12. **Victim factors** — poor health, physical and cognitive impairment, functional dependency, female sex, widowhood, social isolation, low income, and lack of an independent pension.
13. **Perpetrator and environment** — **shared living arrangement with a relative or friend suspected of alcohol or substance abuse, mental illness, or a history of violence**; caregiver burden and burnout; **financial dependence of the abuser on the elder**; property disputes.
14. **Institutional** — low staff-to-resident ratios, untrained staff, high turnover, use of physical and chemical restraint, weak governance, absent complaint mechanisms and poor regulation.

CLINICAL CLUES AND DETECTION

15. **Frequent emergency department visits** for injury or for exacerbations of chronic illness should raise suspicion. Other red flags: delayed presentation, history inconsistent with the injury, injuries of different ages, bruising in atypical sites (inner arm, neck, ear, trunk), malnutrition, dehydration, pressure injury, poor hygiene, missed appointments, medication non-collection, unexplained withdrawals or sudden changes to a will, and a caregiver who answers all questions or refuses to leave the room.
16. **Always interview the older person alone and separately from the caregiver.** Document verbatim, use a body map, photograph injuries with consent, and record who accompanied the patient.
17. **Screening is part of the comprehensive geriatric consultation** in frail, dependent patients. Tools: **EASI** (Elder Abuse Suspicion Index, 6 items), **H-S/EAST**, **VASS**, and the **ED Senior AID** tool for emergency departments.
18. Assessment must be multidisciplinary: **capacity assessment**, cognitive screen (exclude delirium and dementia),

functional assessment, medication review for chemical restraint, financial capacity, social work assessment and a home visit.

MANAGEMENT

19. **Safety first** — treat injuries, assess immediate risk, and decide whether the person can safely return home. Admit, or arrange shelter, respite or emergency placement if not.
20. **If the person has capacity, their autonomy prevails** even if they choose to remain in an abusive setting. Offer a safety plan, written information, contact numbers and definite follow-up, and document the refusal. Do not abandon — the duty of care continues undiminished.
21. **If the person lacks capacity**, act in their **best interests**; involve the guardianship or adult-safeguarding authority, and consider legal protection.
22. **Address caregiver factors** — respite, day care, home nursing, caregiver education and skills training, treatment of caregiver depression and substance misuse, and financial support. Much neglect is the product of exhaustion and ignorance rather than malice.
23. **Legal frameworks** — UK **Care Act 2014** (adult safeguarding); **California** is one of the few jurisdictions where elder abuse is a criminal offence; the Australian Law Reform Commission recommended statutory adult-safeguarding agencies; the US Government Accountability Office (2019) required CMS to track nursing-home abuse by **perpetrator type**.

INDIAN LEGAL AND PROGRAMME FRAMEWORK

24. **Maintenance and Welfare of Parents and Senior Citizens Act, 2007** — maintenance of up to **₹10,000 per month** awarded by **Maintenance Tribunals** at every sub-division, with disposal targeted within **90 days**; abandonment punishable by up to **3 months' imprisonment** and/or fine; **Section 23** allows a property transfer to be declared void if made on condition of maintenance that was subsequently not provided; also mandates old-age homes and medical provision.
25. **Elderline 14567** — the national toll-free helpline (2021) for counselling, rescue and abuse intervention. Supporting programmes: **NPHCE**, Integrated Programme for Senior Citizens, Rashtriya Vayoshri Yojana, SACRED re-employment portal, and the **Ayushman Vay Vandana Card** (₹5 lakh cover for all aged 70+).
26. **Prevention is a public-health and human-rights duty** — combat **ageism**, run public awareness campaigns, train institutional staff, reduce restraint use, mandate incident reporting, teach financial and digital literacy, and above all secure **economic independence of the older person**, which is the single strongest protective factor in Indian data.

LATEST UPDATES (2023–PRESENT)

- **USPSTF final recommendation, 24 June 2025** — screening for intimate partner violence in women of reproductive age is a **B recommendation**, but for **caregiver abuse and neglect of older or vulnerable adults the evidence remains insufficient (I statement)**. Tool performance was inconsistent: **H-S/EAST sensitivity 46%, specificity 73%**; **ED Senior AID sensitivity 94%, specificity 84%**. **Case-finding on clinical suspicion, not universal screening, remains the standard.** Accompanying JAMA commentary described the field as "stagnation at a critical juncture".
- **HelpAge India 2024** national report "*Ageing in India: Exploring Preparedness and Response to Care Challenges*" — identifies the **80-plus, those living alone and older women** as the highest-vulnerability segments requiring a special response.
- **Maintenance and Welfare of Parents and Senior Citizens (Amendment) Bill** proposals: widen the definition of "children" and "relatives" to include sons-in-law, daughters-in-law, adopted and step-children; **remove the ₹10,000 maintenance ceiling** (making it income-linked); mandate **registration and minimum standards for all senior-care homes**; provide for a dedicated senior-citizen police unit in each state.
- **Digital and cyber-financial abuse** of older adults (UPI fraud, "digital arrest" scams, KYC fraud) is now a recognised and rapidly growing category in Indian policing and NCRB-linked data — a new domain of financial abuse absent from older classifications.
- WHO's **Decade of Healthy Ageing (2021–2030)** retains elder abuse as one of its four priority action areas; the Global Report on Ageism (2021) frames ageism as the upstream driver.

CURRENT PRACTICE UPDATE

Issue	Textbook position	Current evidence / practice
Screening	Screening for risk factors and elder abuse is important in frail dependent patients	USPSTF 2025 gives an **I statement** for universal screening; **targeted case-finding** on red flags plus a low threshold for enquiry is what is actually recommended
Classification	Six Council of Europe categories	Retained, plus **resident-to-resident aggression** and **digital/cyber-financial abuse** as emerging categories
Institutional abuse	Restraint use, staff-to-resident abuse and neglect	Systematic incident-reporting schemes and abuse tracking **by perpetrator type**; restraint reduction now a formal quality indicator
Indian law	MWPSC Act 2007 with ₹10,000 ceiling and tribunal	Amendment Bill proposes **removal of the ceiling**,

	route	wider definition of children, and **statutory registration of care homes**; Elderline 14567 operational nationally
Intervention	Ensure safety; involve social work and law	Emphasis on **caregiver-directed intervention** (respite, training, treatment of caregiver mental illness) and on **economic security** of the elder as primary prevention

EXAM ONE-LINERS

- Elder abuse = act or omission causing harm, within a relationship of **expectation of trust**.
- Global prevalence **1 in 6 (15.7%)**; commonest type is **psychological (11.6%)**.
- India LASI: **5.2%** ill-treated; of these **77.3% verbal, 52.6% neglect**; Bihar highest.
- HelpAge India 2024: abuse 7%; **son commonest perpetrator (42%)**.
- Neglect = no intent to harm; abuse = intent. Both must be acted on.
- Strongest risk marker: **shared living with a substance-misusing or mentally ill relative**.
- Always interview the elder **alone**.
- A capacitous elder may refuse intervention — respect it, plan for safety, keep following up.
- MWPSC Act 2007: **₹10,000, 90 days, 3 months' imprisonment, Section 23 property revocation**.
- National elder helpline: **14567**.
- USPSTF 2025: **I statement** for routine elder-abuse screening.

★ EXTRA RECALL — RECURRING TOPIC

Mnemonic for the six Council of Europe types — "6 P's":
Physical · **P**sychological · **P**roperty (material/financial) · **P**rescription (drug abuse / chemical restraint) · **P**ractice (medical violence — deficient care) · **P**rivileges (violation of rights)

Numerical anchors: 15.7% global · 11.6% psychological · 6.8% financial · LASI 5.2% · verbal 77.3% · neglect 52.6% · HelpAge 2024 = 7%, son 42% · ₹10,000 · 90 days · 3 months · Elderline 14567.

Examiner traps:

- Writing only about **physical** abuse — marks are for **financial, medical/neglect, chemical restraint and rights violations**.
- Omitting **institutional abuse and resident-to-resident aggression**.
- Saying "screen all older adults" — USPSTF gives an **I statement**; the correct word is **case-finding**.
- Overriding a capacitous person's refusal to leave an abusive home.
- Forgetting **Section 23** (property revocation) and the **14567** helpline in the Indian section.
- Framing all abuse as malicious — caregiver burden and ignorance are major mechanisms.

> **15-second answer skeleton:** Define (act/omission + trust) → individual vs institutional; abuse vs neglect → **6 P's** classification + RRA → epidemiology (global 1 in 6; LASI 5.2%; HelpAge 7%) → risk factors (dependency, cognitive impairment, shared living with substance-misusing relative) → red flags and interview alone → screening tools → safety, capacity, best interests → caregiver-directed intervention → MWPSC Act 2007 + Elderline 14567 → prevention and anti-ageism.

CONDENSED 7-MARK VERSION — "Short note on elder abuse"

1. **Definition:** any act or omission endangering life, integrity, freedom or financial security of a vulnerable older person, within a relationship of trust (Council of Europe / WHO).
2. **Types (6 P's):** Physical, Psychological, Property (financial), Prescription (chemical restraint), Practice (medical neglect), Privileges (rights violation). Add **resident-to-resident aggression** in institutions.
3. **Neglect vs abuse:** neglect is unintentional (ignorance/inattention); abuse implies intent.
4. **Epidemiology:** global 1 in 6 (15.7%); India LASI 5.2% (verbal 77.3%, neglect 52.6%); HelpAge India 2024 — 7%, son commonest perpetrator (42%).
5. **Risk factors:** dependency, cognitive impairment, female sex, social isolation, economic dependence; shared living with a relative who misuses substances or has mental illness; caregiver burden; poor institutional governance.
6. **Detection:** repeated ED visits, injuries of differing ages, malnutrition, pressure sores, unexplained financial change; **interview alone**; tools EASI, ED Senior AID.
7. **Management:** ensure safety → assess capacity → respect a capacitous refusal, act in best interests if incapacitous → MDT and caregiver support → report under safeguarding law.
8. **India:** MWPSC Act 2007 (₹10,000 maintenance, tribunals, 90 days, Section 23 property revocation, 3 months' imprisonment), Elderline 14567, NPHCE.

3-point current update: (i) **USPSTF June 2025** — I statement for routine screening of caregiver abuse in older adults; case-finding preferred. (ii) **HelpAge India 2024** report identifies 80-plus, those living alone and older women as highest-risk. (iii) **MWPSC (Amendment) Bill** proposes removing the ₹10,000 ceiling, widening "children", and

mandatory registration of senior-care homes; **cyber-financial abuse** is a new and rapidly growing category.

2. ADVANCE CARE PLANNING — 15 MARKS

DEFINITION AND TERMINOLOGY

1. **Advance care planning (ACP)** is a **process** of discussing and recording a person's **values, goals and wishes** for future care and treatment, and documenting them in an **Advance Care Directive (ACD)**.
2. Distinguish clearly: the **process** (ACP) — the **product** (ACD / advance directive / "living will") — and the **proxy appointment** (enduring guardian, durable power of attorney for health care).
3. An ACD has two components: **instructional** (which treatments are refused or accepted, and in which circumstances) and **proxy** (who decides when the person cannot).
4. An **advance decision to refuse treatment** is **legally binding** where valid and applicable; a **statement of wishes and preferences** is advisory only. A **request** for a particular treatment is never binding — no one can demand non-beneficial treatment.

VALIDITY, CAPACITY AND TIMING

5. **Requirements for a valid advance decision:** it must **apply to the specific situation** being considered; it must have been drawn up when the person had **mental capacity**; the decision must have been **voluntary and adequately informed**; the person must not have acted subsequently in a way that renders it invalid; and it must be appropriately **witnessed**.
6. **An advance decision comes into effect only once the person has lost capacity.** While capacity is retained, the clinician must confirm with the patient that the ACD still reflects their current wishes.
7. **Values rarely change over a lifetime, but care preferences do.** Therefore review the ACD **at least yearly**, whenever health deteriorates, and **before admission to an aged-care home**. What a person wants at 85 may differ at 97.
8. **Factors relevant to judging validity:** the person's age when the ACD was written; time elapsed since last review; the condition now being treated; and the type and burden of the proposed treatment and the consequences of giving or withholding it.
9. If the clinician **doubts the validity** of a documented ACD and the patient lacks capacity, the reason for doubt must be **clearly documented** and decision-making reverts to the **best-interests principle**.
10. **Capacity is decision-specific and time-specific.** Every effort must be made to **maximise** capacity before concluding a person cannot decide — correct sensory impairment, use plain language and adequate typeface, colour contrast and white space, choose the best time of day, exclude reversible contributors (delirium, depression, fear, pain, drug effects). Residents with varying degrees of dementia may still participate in many decisions.
11. **Autonomy does not mean "I want, therefore I get".** In a person with capacity it means "you may not do things to me without my permission". Even an apparently unwise refusal stands, and the clinician's duty of care does not diminish — all other care continues without prejudice.

SURROGATE DECISION-MAKING

12. **Hierarchy of treatment decision-makers** where there is no valid ACD (go to the first person who is 18 or over, has full legal capacity, and is reasonably available and willing): (1) **enduring guardian with authority** (2) guardian with authority (3) adult son or daughter (4) parent (5) sibling (6) primary unpaid carer (7) other person with a close personal relationship and frequent contact.
13. The proxy should be chosen on the basis of **knowledge of the person's preferences**, not merely kinship — a close friend or clergyman may know the patient's wishes better than a rarely-seen relative. Where several contenders exist, the court may decide; where no one is suitable, a **public guardian** may be appointed.
14. **Surrogates are fallible.** Their decisions frequently diverge from what the patient would have chosen, and they may be torn between the patient's expressed wishes and their own view of the patient's best interests. Where the proxy is uncertain, the principle of **beneficence** (weighing benefit against burden) guides the decision.
15. **Do not give relatives the incorrect impression that the decision is theirs** when it is not. Consultation with family provides valuable information about best interests; framing it as their decision imposes a heavy emotional burden and long-term guilt. Where families are unwilling to decide, **"informed assent"** — explicitly offering them the option to defer to the clinician's judgement — is the humane approach.

CONTENT, TIMING AND DELIVERY

16. **What to discuss:** cardiopulmonary resuscitation, mechanical ventilation, ICU admission, artificial nutrition and hydration, blood transfusion, antibiotics, dialysis, transfer to an acute hospital versus comfort care; also preferred place of care and death, organ or tissue donation, and funeral, burial or cremation preferences.
17. **Who and when:** new diagnosis of a life-limiting illness; **dementia (now recognised as a terminal illness)**; advancing frailty and multimorbidity; before elective high-risk surgery; and ideally **before entry to a care home**, so that uncertainty and emotional distress during a crisis are reduced.
18. **The worst time to ask is at the moment of admission.** Federal-style laws requiring that everyone entering a hospital or nursing home be offered the opportunity to make a directive produce anxiety and poorly considered decisions if done as a tick-box exercise. Ideally the discussion has already happened; admission merely revisits it.
19. **Barriers** — difficulty imagining hypothetical future health states; reluctance of patients, families and clinicians to

discuss death; poor prognostic literacy; time pressure; and the mistaken assumption that people in their 90s and 100s are "ready to die" — many are not, and want careful evaluation and treatment options discussed.

20. **Benefits** — care consistent with values, less non-beneficial intervention, fewer inappropriate hospital transfers, better decisions out of hours by unfamiliar clinicians, higher likelihood of dying in the preferred place, and reduced surrogate anxiety, depression and guilt.
21. **Care planning tools are no substitute for simple communication with the patient and clear documentation in the clinical record.** The clinician must ensure the person understands the nature and consequences of the proposed treatment, obtain their decision, and communicate it to all relevant care providers.

INDIAN LEGAL FRAMEWORK

22. **Common Cause v. Union of India (2018)** — the Supreme Court recognised the **right to die with dignity** as a facet of **Article 21**, legalised **passive euthanasia**, and gave legal recognition to **Advance Medical Directives (AMDs)**. The 2018 procedure proved unworkable and was used by virtually no one.
23. **Modification of 24 January 2023** (on the application of the Indian Society of Critical Care Medicine): the requirement for **countersignature by a Judicial Magistrate First Class was removed**; the AMD may now be attested before a **Notary or Gazetted Officer in the presence of two witnesses**; the executor may name **more than one** guardian or nominee; the Secondary Medical Board is nominated by the **Chief District Medical Officer** rather than the Collector; and the judicial role is limited to **High Court adjudication of disputes**. The AMD is revocable at any time before implementation.
24. **Requirements for a valid Indian AMD:** executed by an adult of sound mind, voluntarily and with full information; expressed in clear, unambiguous language; in writing; signed before **two independent witnesses**; attested by a **Notary or Gazetted Officer**; naming at least **two surrogate decision-makers**; and a copy given to the nominees, the family physician and the treating hospital.
25. **MoHFW draft Guidelines for Withdrawal of Life Support in Terminally Ill Patients (September 2024)** operationalise the Court's orders through a **Primary Medical Board** (treating doctor plus two subject experts) and a **Secondary Medical Board** including a nominee of the district Chief Medical Officer. Karnataka was the first state to issue implementing orders; Kerala has a state palliative-care and end-of-life care policy.

LATEST UPDATES (2023–PRESENT)

- **Supreme Court modification, 24 January 2023** — simplified execution of AMDs (notary/gazetted officer instead of JMFC), simplified two-tier medical board process, and timelines. Described as making the procedure "workable, though still cumbersome".
- **MoHFW draft guidelines, September 2024** — the first national clinical protocol for withholding and withdrawing life-sustaining treatment.
- **Harish Rana v. Union of India, Supreme Court, 11 March 2026** — the **first judicial implementation of India's passive-euthanasia framework in an individual case**. The Court held that **clinically assisted nutrition and hydration (CANH) delivered by PEG tube is medical treatment, not basic care**, and may therefore be lawfully withdrawn where continued treatment is not in the patient's best interests; withdrawal was ordered under a **tailored palliative care plan at AIIMS**; the Court expanded the **best-interests** test (futility, irreversibility, dignity, family and board opinion), stressed this was **not active euthanasia**, and urged **Parliament to enact a comprehensive statute**. Where both medical boards agree unanimously, the process may proceed **without court involvement**.
- **Efficacy debate:** Morrison, Meier and Arnold (JAMA 2021, "What's wrong with advance care planning?") showed that ACP reliably improves **documentation** but that evidence for **goal-concordant care**, healthcare utilisation and patient outcomes is inconsistent. A 2025 meta-review of reviews, however, found improvement in preference-concordant end-of-life care in **15 of 17** reviews. The debate is unresolved.
- **Shift of emphasis from documents to conversations** — from advance-directive completion toward **serious illness communication** and in-the-moment goals-of-care discussions, using the **Serious Illness Conversation Guide**, the **"What matters to you"** framework, and **ReSPECT** (Recommended Summary Plan for Emergency Care and Treatment). NHS England published **Universal Principles for Advance Care Planning (2022)**.
- **Reframed outcomes** — the clearest benefit of ACP is to the **surrogate**: greater clarity and confidence, a physical reminder of prior discussions, help with in-the-moment decisions, and less decisional regret and guilt. Surrogate-focused outcome measures are now recommended.
- **Technology** — EHR-embedded ACP templates, **video decision aids** (ACP PEACE trial: video plus clinician communication training raised documented ACP in oncology clinics), natural-language processing to identify ACP documentation, and digital/tele-ACP platforms.

CURRENT PRACTICE UPDATE

Issue	Textbook position	Current evidence / practice
Primary goal of ACP	Document values and treatment preferences in an ACD	Emphasis shifted to **iterative serious-illness conversation** and preparing patient and surrogate for in-the-moment decisions; documentation is a means, not the endpoint
Evidence of benefit	Improves quality of end-of-life care and spares unnecessary suffering	Robust for documentation and **surrogate outcomes**; inconsistent for goal-concordant care —

		an active and unresolved debate
India — making an AMD	Not covered in the textbook	**2023 Supreme Court order**: notary or gazetted officer plus two witnesses; JMFC countersignature abolished; more than one nominee permitted
Artificial nutrition/hydration	Requires careful consideration of benefit, burden and risk	**Harish Rana (SC 2026)**: CANH via PEG is **medical treatment** and can be lawfully withdrawn in the patient's best interests, with palliative care mandated
Who decides in India	Statutory hierarchy of decision-makers (UK/Australia)	India has **no statutory hierarchy**; the AMD names nominees, otherwise a **two-board medical process** with family consultation applies
Format of the record	Paper ACD reviewed periodically	Digital, shareable plans (ReSPECT, NHS Universal Care Plan), EHR-integrated, accessible out of hours

EXAM ONE-LINERS

- ACP is the **process**; the ACD or advance directive is the **product**.
- An advance **refusal** is binding; an advance **request** is not.
- An advance decision takes effect **only after capacity is lost**.
- Values are stable; **preferences change** — review the ACD yearly, on deterioration, and before care-home entry.
- Hierarchy: enduring guardian → guardian → adult child → parent → sibling → primary unpaid carer → close contact.
- Choose the proxy who best knows the patient's wishes, not simply the next of kin.
- Never imply to the family that the decision is theirs when it is not; offer **informed assent**.
- Dementia is a **terminal illness** — ACP should begin at diagnosis.
- India: **Common Cause 2018** (Article 21) → **2023 modification** (notary, two witnesses, no JMFC) → **MoHFW 2024 guidelines** → **Harish Rana 2026** (CANH is medical treatment).
- Morrison 2021: ACP improves documentation, not necessarily goal-concordant care — hence the shift to serious-illness communication.

CONDENSED 7-MARK VERSION — "Short note on advance care planning"

1. **Definition:** a process of discussing and recording a person's values, goals and wishes for future care, documented in an advance care directive; distinct from the proxy appointment.
2. **Components:** instructional (what treatment is refused/accepted) and proxy (who decides). Advance **refusals** are binding; requests are not.
3. **Validity:** made with capacity, voluntary, informed, applicable to the situation, witnessed, not subsequently contradicted.
4. **Timing:** effective only after loss of capacity; review yearly, on deterioration, and before care-home admission.
5. **Surrogate hierarchy:** enduring guardian → guardian → adult child → parent → sibling → primary unpaid carer → other close contact; choose by knowledge of the patient's wishes.
6. **Content:** CPR, ventilation, ICU, artificial nutrition/hydration, antibiotics, dialysis, hospital transfer, preferred place of care and death, organ donation.
7. **Barriers:** hypothetical future states, reluctance to discuss death, poor prognostic literacy, tick-box completion at admission.
8. **India:** Common Cause 2018 (Article 21); 2023 modification — notary/gazetted officer plus two witnesses, two nominees, two-tier medical board; MoHFW draft guidelines 2024.

3-point current update: (i) **Supreme Court 2023** simplified execution of Advance Medical Directives, removing JMFC countersignature. (ii) **Harish Rana v. Union of India (March 2026)** — first individual passive-euthanasia order; CANH via PEG held to be medical treatment that may be withdrawn in the patient's best interests, with palliative care mandated. (iii) The field has shifted from **document completion to serious-illness communication** (Serious Illness Conversation Guide, ReSPECT), with **surrogate outcomes** now the recommended measure of benefit.

3. END-OF-LIFE CARE ISSUES — 15 MARKS

DEFINITIONS AND SCOPE

1. **End of life** refers to the **last 12 months of life**; **terminal care** refers to the **last few days or hours**. For many people with a life-limiting illness there is a period of hours to days when it is clear they are dying and require care appropriate to the terminal phase — but not all patients have a recognisable terminal phase, and some die suddenly (massive pulmonary embolus, terminal haemorrhage).
2. **Palliative care is not confined to cancer.** It is appropriate for chronic organ failure, **frailty and dementia**, and the **best outcomes occur when it is commenced early**, alongside disease-directed treatment.

RECOGNISING THE PATIENT APPROACHING THE END OF LIFE

3. **Gold Standards Framework (2016) — three triggers** for a palliative approach:
   (i) the **Surprise Question** — "Would you be surprised if this patient were to die in the next 6–12 months?"
   (ii) **Choice or need** — the patient with advanced disease chooses comfort care only, or has a special need for supportive/palliative care;
   (iii) **Clinical indicators** of advanced disease for specific conditions. **Note that diabetes is not included**, despite being a leading cause of death and suffering that could be managed with a palliative approach.
4. **Failure to recognise dying is the central clinical failure.** Unless the possibility is actively considered when a patient deteriorates, dying patients continue to receive resuscitation and are moved out of their homes and admitted to hospital for their last hours and days.
5. **When to involve specialist palliative care teams** — refractory or complex symptoms, complex psychosocial, spiritual or family distress, requests for hastened death, and difficult ethical decisions. Not everyone benefits from referral; many can be cared for well by their usual doctors and nurses at home or as inpatients.

PLANNING AHEAD AND THE CARE-HOME SETTING

6. Decisions that certain interventions **ought not to be considered** for a particular resident can and should be documented in advance — for example, any investigation or treatment requiring hospital admission, CPR, ventilation, or artificial nutrition in advanced dementia — while others, such as **antibiotics for a urinary tract infection**, may remain appropriate for almost all residents.
7. Ideally an individual entering a care home has an **ACD before admission**, to reduce uncertainty and emotional distress for everyone during a crisis.
8. **Out-of-hours risk:** where usual care services operate only during the working week (in the UK, only about **24% of the whole week**), a deteriorating patient will very likely be reviewed by a clinician unfamiliar with them. Without a clearly documented future-care plan, care may be inappropriately escalated or restricted. A crisis is not the time for an unfamiliar nurse or doctor to open a complex discussion.

WITHHOLDING AND WITHDRAWING TREATMENT

9. **Withholding and withdrawing are ethically and legally equivalent.** There is no obligation to provide treatment that is **non-beneficial**; decisions rest on the balance of **benefits, burdens and risks** for that individual.
10. **CPR** — survival after in-hospital arrest in the terminally ill is very poor, and television portrayal grossly misleads the public about outcomes. Members of the public who perform CPR are hailed as heroes, but no one asks whether the person had an advance directive; those resuscitated in public may not die free from suffering or with dignity. A **DNACPR decision restricts nothing else**; it must be documented, communicated and reviewed.
11. **Artificial hydration** — nutrition and hydration must be considered **separately**. People can live for a time with almost no nutrition, but hydration is essential because renal failure and dehydration develop rapidly. Where a patient cannot swallow, maintaining some hydration is an important **comfort measure**. **Subcutaneous fluid (hypodermoclysis)** can be set up in almost any environment through an abdominal-wall needle, carries very low complication risk, and is **as effective as intravenous hydration for long-term maintenance** — yet is rarely used. Avoid fluid overload, especially with cardiac decompensation. Hydration is an adjunct to, not a substitute for, **meticulous mouth care**.
12. **Nutrition is an emotionally charged area.** Families feel an overwhelming desire to provide food. Dying patients have no appetite, distorted taste and disease-related cachexia. Anorexia may be the first sign of subclinical nausea — therefore **exclude hypercalcaemia, hyperglycaemia, renal failure and infection (particularly urinary)**.
13. **Corticosteroids** stimulate appetite only briefly and cause hyperglycaemia, muscle wasting, gastrointestinal erosion and bleeding, emotional lability and impaired sleep; frank steroid psychosis is rare but mood disturbance is common. **Megestrol acetate has no advantage** over glucocorticoids.
14. **Nasogastric feeding** is very uncomfortable, irritating and disfiguring, and carries a significant risk of aspiration pneumonia; **PEG feeding in advanced dementia does not improve survival, nutrition, pressure-ulcer healing or quality of life**. Any form of artificial nutrition needs very careful consideration because in most such patients it is unlikely to benefit survival or quality of life.

CARE IN THE LAST DAYS OF LIFE

15. **Integrated care pathways** — the **Liverpool Care Pathway for the Dying Patient (LCP)** was the UK standard and was adopted or adapted in many countries including Australia. It was withdrawn in the UK following the Neuberger review and replaced by the **Five Priorities for Care of the Dying Person** (Leadership Alliance, 2014): **Recognise —**

Communicate — Involve — Support — Plan and do.

16. If the patient **improves or recovers**, the pathway must be discontinued, the situation reviewed, and the need to recommence interventions considered. **One important reason patients improve is that unnecessary medicines were stopped** — in retrospect it may be clear that the drugs, not the disease, caused the deterioration.
17. **Anticipatory ("just in case") prescribing** with subcutaneous PRN medication and a syringe driver: an **opioid** (morphine; oxycodone or fentanyl if renal impairment) for pain and breathlessness; **midazolam** for restlessness, anxiety and seizures; **haloperidol or levomepromazine** for nausea and delirium; an **antimuscarinic** (hyoscine butylbromide or glycopyrronium) for respiratory secretions. Stop non-essential drugs and convert essential ones to the subcutaneous route.
18. **Excessive secretions and cough** — rather than subject the patient to repeated suctioning, treat pharmacologically with an anticholinergic; reposition; explain the "death rattle" to the family, who are usually more distressed by it than the patient. Cough: treat the cause (diuretics in heart failure, antibiotics if infection), then central suppression with dextromethorphan or codeine linctus.
19. **Terminal restlessness** — always exclude reversible causes first: pain, **urinary retention**, faecal impaction, hypoxia, hypercalcaemia, hypoglycaemia, opioid toxicity, and withdrawal from nicotine, alcohol or benzodiazepines.
20. **Psychiatric symptoms at the end of life** — anxiety, depression, hallucinations and delirium are common and distress families more than patients. If the patient is not troubled by the symptom (for example, pleasant hallucinations), **no treatment is required**; support the family instead. Treatment of one symptom may create another — opioids for pain may cause delirium, in which case switch opioid or route.

PALLIATIVE SEDATION

21. **Sedation at the end of life should rarely be necessary.** It may be required where a patient is exhausted by complex pain or profoundly distressed following a neurological insult. Because it is a therapeutic intervention it requires **consent**; if the patient lacks capacity a short period of sedation (for example, to control difficult delirium) may be in their best interests, decided with the person holding power of attorney or, failing that, the preferred proxy and other interested parties.
22. **Titrate short-acting agents such as midazolam in increments** to the desired effect, review regularly, and lower the dose again at the agreed time. In the last days and hours patients may become restless and appear anxious; **small anxiolytic doses suffice — it is not necessary to render the patient deeply unconscious**. Review the benefits, burdens and risks of continued parenteral hydration and enteral nutrition in sedated patients.
23. **Palliative sedation is distinguished from euthanasia by three features: intention** (to relieve intractable symptoms, not to end life), **proportionality** (careful titration according to effect on symptoms), and **causation** (death results from the underlying disease). The Dutch "terminal sedation" protocol — high-dose continuous infusion with nutrition and hydration withheld — has been criticised as euthanasia without the required administrative framework.

PERSONHOOD, PLACE OF DEATH, BEREAVEMENT

24. **Personhood persists** even when the patient is actively dying and has lost many functions. While still alive they are no less a person, though they may be a different person from before the illness. Goals of care include being kept clean, having a designated decision-maker, dignity, optimal control of pain, breathlessness and anxiety, financial affairs in order, the opportunity to say goodbye, appropriate physical touch, life review, and reassurance that the family is prepared.
25. **Nursing homes** — a median of about **18%** of adults aged 65 and over die in a nursing home (range 3% in Korea to 38% in New Zealand and Iceland). At least **60%** of nursing-home decedents have dementia and most have two or more comorbidities. **Palliative care consultation in the nursing home is associated with significantly lower rates of end-of-life hospitalisation**, and there is expert consensus that hospice and palliative care should be a **standard service in nursing homes**.
26. **Bereavement** — anticipatory grief support, honest information, involvement in care, follow-up contact after the death, and identification of **complicated/prolonged grief disorder**. Carer distress before the death predicts poor bereavement outcome. Once the person dies they are a body, and responsibility passes to the named next of kin.
27. **Assisted dying** remains a controversial and separate issue from palliative care (Victoria, Australia legalised Voluntary Assisted Dying in 2017). **In India, active euthanasia and physician-assisted suicide remain illegal**; only **passive euthanasia** — withholding or withdrawing life-sustaining treatment — is lawful.

INDIAN ISSUES IN END-OF-LIFE CARE

28. **Opioid access** transformed by the **NDPS (Amendment) Act 2014**, which created the category of **Essential Narcotic Drugs** with a single state-level licence, replacing multiple licences under conflicting state rules. Coverage nevertheless remains poor outside a few states.
29. **Kerala's Neighbourhood Network in Palliative Care** and the **2008 Kerala Palliative Care Policy** (the first in India) remain the national and international model of a community-owned, volunteer-driven, home-based service integrated with local self-government. The **National Programme for Palliative Care (NPPC)** is now delivered through the NHM.
30. **Persistent problems** — over-medicalised death in ICUs, out-of-pocket catastrophic expenditure, family-centred (rather than patient-centred) decision-making, non-disclosure of prognosis to the patient, absence of dedicated legislation, and very limited palliative-care workforce and training.

LATEST UPDATES (2023–PRESENT)

- **Harish Rana v. Union of India, Supreme Court of India, 11 March 2026** — India's **first court-sanctioned individual case of passive euthanasia**. Key holdings: **CANH delivered by PEG tube is medical treatment, not basic care**; withdrawal is an **authorised omission** allowing the underlying condition to take its natural course, not an act causing death; a detailed **best-interests framework** (futility, irreversibility, dignity, family and medical-board opinion); withdrawal **must be accompanied by active palliative care**; the two-board process may proceed **without court involvement** where both boards agree; and Parliament was urged to enact a comprehensive end-of-life statute. The patient was transferred to the AIIMS palliative care unit and died on 24 March 2026.
- **MoHFW draft Guidelines for Withdrawal of Life Support in Terminally Ill Patients (September 2024)** — Primary and Secondary Medical Boards, four situations in which life-sustaining treatment may be foregone, and documentation requirements.
- **ISCCM position statement on end-of-life care in the ICU** and successive ICMR/national ethics documents have operationalised the 2023 Supreme Court order into ward-level protocols; Karnataka issued the first state implementation order.
- **Lancet Commission on the Value of Death (2022, continuing influence)** — calls for "rebalancing death and dying", criticises over-medicalisation and the professional monopoly over dying, and holds up **Kerala's compassionate-communities model** as the exemplar.
- **WHO ICOPE, 2nd edition (2025)** and the Decade of Healthy Ageing embed palliative care within integrated care for older people, with intrinsic-capacity-based rather than disease-based triggers.
- **Early integrated palliative care** now has trial-level support in non-malignant disease — advanced heart failure, COPD, end-stage renal disease, Parkinson's disease and dementia — strengthening the case for a needs-based rather than diagnosis-based referral trigger.
- **Prolonged Grief Disorder** is now a formal diagnosis in **ICD-11** and **DSM-5-TR (2022)** — persistent, pervasive grief beyond 6 months (ICD-11) or 12 months (DSM-5-TR) causing functional impairment; targeted psychotherapy is effective.

CURRENT PRACTICE UPDATE

Issue	Textbook position	Current evidence / practice
Care of the dying pathway	Liverpool Care Pathway described as the standard for the terminal phase	LCP **withdrawn in the UK**; replaced by the **Five Priorities for Care** — individualised care, not a tick-box pathway
Artificial nutrition & hydration	Consider benefits, burdens and risks; subcutaneous fluid underused	Same clinical position, now reinforced legally in India: **CANH is medical treatment (Harish Rana 2026)** and may be withdrawn in best interests
Trigger for palliative care	Gold Standards Framework surprise question and clinical indicators	Needs-based triggers; **early integrated palliative care** in non-malignant disease; ICOPE intrinsic-capacity framing
Passive euthanasia in India	Not addressed	**Common Cause 2018 → 2023 modification → MoHFW 2024 guidelines → Harish Rana 2026**; two-board process without court where boards agree
Grief	Bereavement support for relatives	**Prolonged Grief Disorder** formally recognised in ICD-11 and DSM-5-TR with specific psychotherapy
Place of care	Palliative care consultation reduces hospitalisation in nursing homes	Hospice/palliative care recommended as a **standard service** in long-term care; growth of hospital-at-home for end-of-life care

EXAM ONE-LINERS

- End of life = last **12 months**; terminal = last **days to hours**.
- **Surprise question:** "Would I be surprised if this patient died within 6–12 months?"
- Withholding and withdrawing treatment are **ethically and legally equivalent**.
- **Subcutaneous fluid is as effective as IV** for maintenance hydration and far simpler.
- PEG feeding in advanced dementia does not improve survival or quality of life.
- Exclude **hypercalcaemia, hyperglycaemia, renal failure and infection** before accepting anorexia as terminal.
- Terminal restlessness: always exclude **urinary retention and faecal impaction** first.
- Palliative sedation vs euthanasia = **intention, proportionality, causation**.
- Anticipatory drugs: **opioid, midazolam, haloperidol, antimuscarinic** — all subcutaneous.
- If a dying patient improves, **stop the pathway and review** — deprescribing may have been the cause.
- India: only **passive** euthanasia is legal; **Harish Rana (2026)** held CANH to be medical treatment.
- Kerala's Neighbourhood Network in Palliative Care is the model community programme; NDPS Amendment 2014 opened opioid access.

CONDENSED 7-MARK VERSION — "Short note on end-of-life care issues"

1. **Definitions:** end of life = last 12 months; terminal phase = last days to hours; palliative care applies to frailty, dementia and organ failure, not only cancer, and works best when started early.
2. **Recognising dying:** Gold Standards Framework triggers — surprise question, patient choice/need, clinical indicators. Failure to recognise dying leads to futile resuscitation and inappropriate hospital transfer.
3. **Advance planning:** document in advance which interventions are not to be considered; ideally an ACD before care-home admission; essential for safe out-of-hours decisions.
4. **Withholding vs withdrawing:** ethically and legally equivalent; no obligation to give non-beneficial treatment; DNACPR restricts nothing else.
5. **Nutrition and hydration:** consider separately; subcutaneous fluid as effective as IV; exclude hypercalcaemia, hyperglycaemia, renal failure and infection as causes of anorexia; PEG does not help advanced dementia; steroids and megestrol have limited value.
6. **Symptom control in the last days:** subcutaneous opioid, midazolam, haloperidol and an antimuscarinic; stop non-essential drugs; exclude retention and impaction in terminal restlessness.
7. **Palliative sedation:** rarely needed; titrated; distinguished from euthanasia by intention, proportionality and causation.
8. **Psychosocial and bereavement:** dignity, personhood, saying goodbye, family support, follow-up for prolonged grief.

3-point current update: (i) The **Liverpool Care Pathway has been withdrawn** and replaced by the **Five Priorities for Care of the Dying Person** — individualised care rather than a pathway. (ii) **Harish Rana v. Union of India (SC, March 2026)** — CANH via PEG is medical treatment that may be withdrawn in the patient's best interests, with mandatory palliative care; MoHFW 2024 guidelines operationalise the two-board process. (iii) **Prolonged Grief Disorder** is now a formal ICD-11 / DSM-5-TR diagnosis, and **early integrated palliative care** is evidence-based in non-malignant disease.

4. TRANSITIONAL CARE HOME: PREREQUISITES FOR ADMISSION AND DISCHARGE, AND STAFFING ROLES — 15 MARKS

DEFINITION AND PURPOSE

1. **Transitional care** (also called **intermediate care**, **post-acute care**, **step-down care** or **reablement**) is **time-limited, goal-directed, multidisciplinary care** provided after the acute phase of illness. It was conceived in response to rising demand for acute hospital beds and aims to **promote faster recovery from illness, prevent unnecessary acute admissions, support timely discharge, and maximise independent living**.
2. It sits between the acute hospital and the patient's home or long-term care. Post-acute programmes admit selected older patients who have completed initial acute treatment but have an **ongoing need for post-acute and rehabilitative care**; they contain a **rehabilitation component with an interdisciplinary team assigning patients to specific therapies based on an individual geriatric assessment**.
3. Guiding principle: care is **functional-based rather than disease-based**. Many older people have multiple chronic conditions that will not be cured; goals focus on **maximising function**, and **functional status and quality of life** become the markers of successful recovery rather than measures of disease severity.
4. The philosophy is **biopsychosocial rather than biomedical** — the relationship between the patient and the social structures needed for effective treatment is central, and barriers to recovery are identified early.

PREREQUISITES FOR ADMISSION

5. **Medical stability** — the acute illness is diagnosed and stabilised; no requirement for continuous cardiac monitoring, ventilatory support, vasopressors or other intensive-care-level intervention; the essential diagnostic work-up is complete.
6. **A defined, achievable goal with an expected time frame** — either **rehabilitation potential** (mobility, ADL, gait, swallow, speech) or a specific care goal such as wound healing, completion of a parenteral antibiotic course, insulin titration, tracheostomy weaning or caregiver training. Transitional care is not a holding bay for social admissions.
7. **A completed comprehensive geriatric assessment (CGA)** documenting medical, functional (ADL/IADL, Barthel or FIM), cognitive, mood, nutritional, continence, mobility and falls, sensory, medication and social domains, with a written problem list and baseline scores against which progress will be measured.
8. **Three obligations of the referring clinician**: (i) the patient **truly needs care in such a setting and cannot reasonably get such care elsewhere**; (ii) the **institution is capable of providing the needed care**; (iii) the **patient is prepared for the transfer**.
9. **Informed consent** from the patient (or valid surrogate), with the family given a genuine role and sufficient time. The specific information to be presented includes **cost and cost-based alternatives, privacy, safety, social issues and access to appropriate health care**. Decisions about transitions between levels of care deserve the same seriousness as decisions about treatment.
10. **Documented goals of care, resuscitation status and, ideally, an advance care directive**; capacity for medical decision-making assessed within days of admission and a surrogate identified if the patient lacks capacity.
11. **Complete structured transfer documentation** — diagnoses and procedures, pending investigations, **reconciled medication list with reasons for every change**, allergies, functional and cognitive baseline, pressure-injury and falls risk, infection/multidrug-resistant organism status, lines, catheters and wounds, follow-up appointments and the named responsible physician. **Poor transmission of information to the receiving staff or to the patient is a principal cause of readmission.**
12. **Exclusions** — unstable acute illness or undiagnosed deterioration; active delirium still requiring acute work-up; need for surgical or intensive intervention; severe behavioural disturbance or suicidal risk beyond the facility's capability; infection requiring isolation the facility cannot provide; and patients whose only need is long-term custodial care, who belong in a nursing home rather than transitional care.
13. **Avoid the "first train leaving the station" phenomenon.** Discharge planners under pressure to move patients quickly leave little time for careful consideration of options or of what the patient and family want to achieve; availability of a placement then trumps suitability. Nursing-home discharges are easier to arrange and are therefore over-used. **Discharge planning must begin early in the admission, not at the end.**

PREREQUISITES FOR DISCHARGE

14. **Goal attainment or documented plateau** — the agreed functional goals have been met, or the interdisciplinary team has determined that **maximum progress toward the goals has been reached** and further gain is unlikely. Not every resident has areas in which improvement can reasonably be expected; for these, **strategies to maintain function** are developed instead.
15. **Clinical stability** — afebrile with stable vital signs, adequate oral intake and hydration, pain controlled, wounds healing, bowel and bladder plan working, and no ongoing need for parenteral therapy that cannot be delivered in the destination setting.
16. **Safe destination and environment** — home assessment and modification completed (grab rails, ramp or lift access, raised toilet seat, bathroom adaptation, lighting, removal of loose rugs), assistive devices supplied and the patient trained in their use. **Assessment of and recommendation for modifying the environment can be critically important for improving function and for discharge planning.**
17. **A competent, willing and trained caregiver**, or a funded package of home care; caregiver skills demonstrated by

teach-back (transfers, feeding, wound care, catheter care, insulin, inhalers).

18. **Medication reconciliation and deprescribing at discharge** — simplified written regimen, reasons for changes explained, high-risk drugs (anticoagulants, insulin, opioids) specifically reviewed, and the patient or carer able to demonstrate correct administration.
19. **Follow-up secured** — a named responsible clinician, a booked appointment, arranged home nursing and therapy, a written escalation plan ("what to do if…", who to call), and, in most cases, a strategy for **long-term follow-up and formal case management to ensure that needs and services remain appropriately matched**.
20. **Structured discharge summary sent to the community team and given to the patient** in accessible, ageing-friendly language, with teach-back to confirm understanding. Templated discharge communication and facilitated follow-up appointments are core components of geriatric-friendly services.
21. **Good discharge decision-making separates two distinct questions:** (i) **what type of care** is most likely to achieve the primary goals for this patient, and (ii) having chosen the modality, **which provider** can best deliver those services. These require quite different sets of information and are often wrongly conflated.

STAFFING AND ROLES

22. **Core interdisciplinary team** — in the geriatric assessment setting the key disciplines are, at a minimum, **physicians, nurses, social workers and physical and occupational therapists**, optimally extended to dieticians, pharmacists, ethicists and home-care specialists. **Special geriatric expertise among team members is crucial.**
    - **Medical director / geriatrician** — clinical governance, admission and discharge criteria, protocols, staff education, complex ethical and prognostic decisions.
    - **Attending physician / nurse practitioner / physician assistant** — daily medical review, comorbidity and medication management, review and signing of the care plan, and **strong advocacy for the autonomy and quality of life of residents**.
    - **Nursing staff** (with a **higher nurse and aide ratio** than acute wards) — 24-hour care, early detection of change in condition, delirium and falls prevention, continence and skin care, medication administration, family communication.
    - **Physiotherapist** — mobility, transfers, balance, gait, strength, endurance, falls rehabilitation.
    - **Occupational therapist** — ADL and IADL retraining, adaptive equipment, cognitive-functional strategies, and **home assessment and modification**.
    - **Speech and language therapist** — dysphagia assessment, diet texture and fluid consistency, communication rehabilitation after stroke.
    - **Clinical pharmacist** — medication reconciliation, deprescribing, Beers and STOPP/START review, drug–drug and drug–disease interaction screening, carer education.
    - **Dietician** — nutritional assessment within days of admission and periodically thereafter, dietary preferences, and an appropriate diet. **Restrictive diets are often not medically necessary and are unappetising**; weight loss must be identified and reported.
    - **Medical social worker / case manager** — psychosocial assessment within 1–2 weeks, family relationships and social resources, financial and entitlement issues, placement options, community linkage, and coordination of discharge. Social workers and nurse case managers are often best placed to delineate the choices and implications of a move.
    - **Clinical psychologist** — mood, adjustment, behavioural management, cognitive assessment.
    - **Podiatrist, dentist, optometrist and audiologist** — foot care and sensory correction, both of which strongly influence function.
    - **Chaplain / pastoral care and palliative care liaison**; **trained nursing aides and attendants**; **volunteers** for social engagement.
23. **The interdisciplinary team conference is the critical ingredient.** It takes place after individual assessments are complete; **most successful trials of geriatric assessment included such a conference**. It brings all perspectives together, generates new ideas, sets priorities, disseminates the full assessment to everyone treating the patient, and avoids duplication and incongruity. Effective teams evolve through **forming, storming, norming and performing**; the **patient and carer must be involved at some stage** to maintain the principle of choice.
24. **Documentation cycle** — an **interdisciplinary care plan within 1–2 weeks of admission and every 3 months thereafter**, listing problems, goals, approaches, target dates and assignment of responsibility across disciplines. The physician should review and sign it; care plans are a powerful driver of nursing behaviour and expectations.
25. **Structural and environmental prerequisites** — barrier-free access, non-slip flooring, grab rails, adequate and glare-free lighting, **enhanced signage, calendars and clocks for reorientation**, communication aids, low beds and call bells, a **restraint-reduction protocol**, assistive devices, a rehabilitation gymnasium, an ADL training kitchen and bathroom, and a formal transfer agreement with a nearby acute hospital.
26. **Quality indicators** — length of stay, functional gain (change in Barthel/FIM), proportion discharged home, 30-day readmission and emergency transfer rates, falls, pressure injuries, restraint use, healthcare-associated infection, medication errors, delirium incidence, and resident and family satisfaction.
27. **INTERACT** (Interventions to Reduce Acute Care Transfers) reduces unnecessary hospital transfers through three strategies: (i) **proactive identification** of conditions before they become severe enough to need hospital care (dehydration, delirium); (ii) **on-site management** of some conditions (pneumonia, congestive heart failure) when safe and feasible; and (iii) **improved advance care planning**, so that palliative or comfort care is considered when the risks of hospitalisation outweigh the benefits.

INDIAN CONTEXT

28. **NPHCE** provides **30-bedded departments of geriatric medicine at Regional Geriatric Centres**, 10-bedded geriatric wards at district hospitals, rehabilitation units at CHC level and home-based care through ASHAs — but **dedicated step-down/transitional facilities are not part of the public system**.
29. **No statutory regulation** currently governs transitional-care or step-down homes in India. Applicable frameworks are the **Clinical Establishments (Registration and Regulation) Act 2010**, the model minimum standards for senior-care homes developed for the Ministry of Social Justice and Empowerment, and state-level rules. **AB-PMJAY / Ayushman Vay Vandana does not reimburse rehabilitation or step-down bed-days**, which is the major financing barrier.

LATEST UPDATES (2023–PRESENT)

- **Hospital-at-home and "virtual wards"** have expanded rapidly as an alternative to facility-based transitional care — NHS England virtual wards and the CMS Acute Hospital Care at Home waiver, with meta-analyses showing comparable or lower mortality, less delirium, higher satisfaction and lower cost than inpatient care in selected older patients.
- The best-evidenced models remain **Naylor's Transitional Care Model** (advanced practice nurse-led, with home visits) and **Coleman's Care Transitions Intervention** (transition coach, personal health record, medication self-management, red flags, follow-up). 2023–25 syntheses confirm that **single-component interventions do not reduce readmission** — only bundled, person-delivered models do.
- **Pharmacist-led transitions of care** and structured medication reconciliation at both ends of the transfer have the strongest single-component evidence for reducing medication-related harm.
- Growing recognition of **"post-hospital syndrome"** — a transient acquired period of generalised vulnerability after hospitalisation driven by sleep deprivation, immobility, malnutrition and pain, which transitional care is specifically designed to buffer.
- **India:** the NITI Aayog position paper **"Senior Care Reforms in India: Reimagining the Senior Care Paradigm" (February 2024)** recommends a formal continuum of care spanning acute, transitional, long-term and home-based services, regulation of care homes, and integration of elder care into insurance; the proposed **Maintenance and Welfare (Amendment) Bill** would mandate registration and minimum standards for all senior-care homes.
- **Geriatric Emergency Department accreditation** and **Age-Friendly Health Systems (4Ms — What Matters, Medication, Mentation, Mobility)** have become the dominant organising frameworks for care transitions in older adults.

CURRENT PRACTICE UPDATE

Issue	Textbook position	Current evidence / practice
Site of post-acute care	Facility-based post-acute and intermediate care units	Rapid shift toward **hospital-at-home and virtual wards**; facility care reserved for those needing 24-hour nursing or intensive rehabilitation
Reducing readmission	Good discharge planning and information transfer	Only **bundled, person-delivered models** (Transitional Care Model, Care Transitions Intervention) work; single-component fixes do not
Preventing acute transfer	Careful discharge decision-making	**INTERACT** as a formal quality-improvement programme; proactive identification, on-site management, and ACP-based decisions
Organising framework	ACE unit components; interdisciplinary team conference	**Age-Friendly Health Systems 4Ms** and Geriatric ED accreditation now structure care transitions internationally
India	Not addressed	No statutory regulation of step-down facilities; **NITI Aayog 2024** recommends a formal continuum; PMJAY does not fund rehabilitation bed-days

EXAM ONE-LINERS

- Transitional care = **time-limited, goal-directed, multidisciplinary** care between acute hospital and home.
- Aims: faster recovery, avoid unnecessary admission, support timely discharge, maximise independent living.
- Approach is **functional-based, not disease-based**; outcomes are function and quality of life.
- Three admission obligations: the patient needs this setting; the facility can provide it; the patient is prepared for transfer.
- Admission requires **medical stability + a defined achievable goal + a completed CGA**.
- Discharge requires **goal attainment or plateau + clinical stability + a safe environment + a trained caregiver + secured follow-up**.
- Poor information transfer at transition is the principal cause of readmission.
- The **interdisciplinary team conference** is the critical ingredient of every successful CGA trial.
- Care plan within **1–2 weeks** of admission and every **3 months** thereafter.
- Beware the **"first train leaving the station"** — availability trumping suitability.

- **INTERACT**: proactive identification, on-site management, advance care planning.
- India: NPHCE geriatric wards exist, but there is **no regulated transitional-care tier** and no PMJAY funding for rehabilitation bed-days.

CONDENSED 7-MARK VERSION — "Short note on transitional care"

1. **Definition:** time-limited, goal-directed, multidisciplinary post-acute care bridging hospital and home; aims to speed recovery, prevent admission, support timely discharge and maximise independence.
2. **Admission prerequisites:** medical stability; a defined achievable rehabilitative or care goal with a time frame; completed comprehensive geriatric assessment with baseline function; consent and preparation of patient and family; documented goals of care and resuscitation status; complete reconciled transfer documentation.
3. **Exclusions:** unstable illness, need for intensive or surgical care, severe behavioural disturbance, or a purely custodial long-term need.
4. **Discharge prerequisites:** goal attainment or documented plateau; clinical stability; safe modified home environment with assistive devices; a trained willing caregiver or funded home package; medication reconciliation with teach-back; named follow-up clinician, booked appointment and written escalation plan; discharge summary to the community team.
5. **Staffing:** geriatrician/medical director, attending physician or nurse practitioner, nurses at a higher ratio, physiotherapist, occupational therapist, speech therapist, pharmacist, dietician, medical social worker/case manager, psychologist, chaplain, trained aides and volunteers.
6. **Process:** interdisciplinary team conference after individual assessments; care plan within 1–2 weeks and reviewed 3-monthly with goals, target dates and assigned responsibility; patient and carer involved.
7. **Environment:** barrier-free access, grab rails, good lighting, orientation aids, restraint-reduction policy, rehabilitation gym and ADL training area, acute-hospital transfer agreement.
8. **Quality indicators:** functional gain, discharge-home rate, 30-day readmission, falls, pressure injuries, restraint use, infection, satisfaction.

3-point current update: (i) **Hospital-at-home / virtual wards** are displacing facility-based transitional care for selected patients, with comparable outcomes and less delirium. (ii) Only **bundled models** (Naylor's Transitional Care Model, Coleman's Care Transitions Intervention) and **pharmacist-led medication reconciliation** reduce readmission; single-component interventions do not. (iii) **India**: no statutory regulation of step-down facilities; **NITI Aayog (2024)** recommends a formal continuum of senior care, and the Maintenance and Welfare Amendment Bill proposes mandatory registration of care homes.

5. SLEEP HYGIENE / PATHOPHYSIOLOGY OF INSOMNIA AND ITS MANAGEMENT — 15 MARKS ★ RECURRING

NORMAL SLEEP AND CHANGES WITH AGE

1. **Sleep** is a reversible state of reduced responsiveness to the environment, comprising **non-rapid eye movement (NREM) sleep — stages N1, N2 and N3 (slow-wave sleep)** — and **rapid eye movement (REM) sleep**. NREM and REM alternate in cycles of about **90–110 minutes**, with **4–6 cycles** a night; slow-wave sleep dominates the first third of the night and REM periods lengthen toward morning.
2. **Changes in old age:** increased **sleep-onset latency**; reduced **total sleep time and sleep efficiency**; marked reduction in the absolute and relative amount of the **deeper stages (N3)** with a smaller reduction in REM; increase in light N1/N2 sleep; increased number and duration of **awakenings (increased wake after sleep onset)**; **circadian phase advance** (earlier bedtime, earlier morning awakening); and increased daytime napping.
3. **These changes are physiological, not pathological.** The older person's **ability** to sleep declines while the **need** for sleep is largely unchanged. Therefore, insomnia in later life is usually explained by **comorbidity, drugs, primary sleep disorders or circadian disruption**, and must never be dismissed as "just ageing".

DEFINITION

4. **Insomnia disorder** (ICSD-3 / DSM-5): dissatisfaction with sleep quantity or quality, with **difficulty initiating sleep, difficulty maintaining sleep, or early-morning awakening with inability to return to sleep**, occurring **despite adequate opportunity and circumstances for sleep**, and accompanied by **daytime impairment** (fatigue, mood disturbance, impaired attention or memory, social or occupational dysfunction). **Chronic insomnia disorder = symptoms at least 3 nights a week for at least 3 months.**
5. Characterise the pattern precisely, because it points to the cause: **delayed sleep onset** (anxiety, RLS, delayed phase, caffeine), **frequent awakenings** (pain, nocturia, sleep apnoea, PLMD, reflux), **early-morning awakening** (depression, phase advance, alcohol).

PATHOPHYSIOLOGY

6. **Hyperarousal is the core mechanism** — chronic insomnia is a disorder of 24-hour hyperarousal, not merely of night-time sleep loss. Evidence: raised whole-body metabolic rate, elevated cortisol and ACTH, sympathetic overactivity with reduced heart-rate variability, increased high-frequency **beta and gamma EEG activity** at sleep onset and during NREM, and increased regional brain glucose metabolism during NREM sleep.
7. **Neurochemistry of the sleep–wake switch:** wakefulness is driven by the ascending arousal system — **orexin/hypocretin** (lateral hypothalamus), histamine (tuberomammillary nucleus), noradrenaline (locus coeruleus), serotonin (raphe), acetylcholine and dopamine. Sleep is driven by the **ventrolateral preoptic nucleus (VLPO)** through **GABA and galanin**, with **adenosine** as the principal endogenous somnogen. The mutually inhibitory VLPO–arousal "flip-flop switch" is **stabilised by orexin**; loss of stabilisation produces state instability and fragmented sleep.
8. **Two-process model: Process S** — homeostatic sleep pressure accumulating with wakefulness (adenosine-mediated); **Process C** — the circadian drive generated by the **suprachiasmatic nucleus**, entrained by light through the retinohypothalamic tract and expressed via pineal **melatonin**. Ageing reduces the amplitude of both processes.
9. **Age-related circadian dysfunction: SCN neuronal loss and reduced SCN output**, decreased and phase-advanced melatonin secretion, reduced daytime light exposure (cataract, reduced pupil size, indoor living, institutionalisation), and reduced physical and social activity (weak *zeitgebers*) → **phase advance and reduced rhythm amplitude**, expressed clinically as early-evening sleepiness, early-morning awakening, daytime napping and, in dementia, **sundowning and irregular sleep–wake rhythm**.
10. **Spielman's 3-P behavioural model** explains the transition from acute to chronic insomnia:
    Predisposing — age, female sex, family history, trait hyperarousal, anxious or ruminative personality.
    Precipitating — acute illness, hospitalisation, pain, bereavement, retirement, relocation, a new drug.
    Perpetuating — maladaptive behaviours (excessive time in bed, prolonged daytime napping, clock-watching, early bedtime) and dysfunctional beliefs ("I must sleep 8 unbroken hours or I will be ill"). **Perpetuating factors are what CBT-I targets, and they are what keep insomnia going long after the trigger has resolved.**
11. **Conditioned (psychophysiological) insomnia** — through repeated pairing, the bed and bedtime routine become learned cues for wakefulness and anxiety rather than for sleep. This is the rationale for **stimulus-control therapy**.

CAUSES AND EVALUATION

12. **Physical symptoms underlying insomnia** (elicit from the patient **and the bed partner**): pain from musculoskeletal disorders; **orthopnoea, paroxysmal nocturnal dyspnoea or cough**; **nocturia**; gastro-oesophageal reflux — all worsened by recumbency. Also breathlessness, anxiety and restlessness.
13. **Symptoms of primary sleep disorders** — **periodic limb movements/restless legs** (uncomfortable leg sensations with an urge to move; RLS prevalence rises to about **20% in those aged 80 and over**); **sleep apnoea** (loud or irregular snoring, waking sweating, anxious and tachycardic, excessive movement, morning drowsiness); and **REM sleep behaviour disorder** (dream enactment, a prodrome of synucleinopathy).
14. **Behavioural factors** — daytime naps **longer than 30 minutes**, earlier bedtime, and increased time spent in bed not sleeping.

15. **Drugs** — **hypnotic withdrawal**, caffeine, **alcohol (causes sleep fragmentation and rebound awakening in the second half of the night)**, certain antidepressants (SSRIs, SNRIs, bupropion), diuretics (nocturia) and corticosteroids; also theophylline, beta-blockers, levodopa, donepezil, decongestants and thyroxine excess.
16. **Psychiatric** — insomnia is a cardinal symptom of depression and anxiety and may be the presenting complaint. But it is also a good example of a symptom that must be evaluated for a treatable underlying cause: **do not attribute insomnia to age or to depression before a comprehensive medical work-up is complete**. Older adults may also hold unrealistic sleep expectations.
17. **Assessment tools** — a **sleep diary for 1–2 weeks** (the single most useful instrument), collateral history from the bed partner or carer, actigraphy where available, and validated scales: **Insomnia Severity Index**, Pittsburgh Sleep Quality Index, Epworth Sleepiness Scale.
18. **Polysomnography is NOT routinely indicated for insomnia.** Reserve it for suspected sleep apnoea, periodic limb movement disorder, REM sleep behaviour disorder or other parasomnia with injury, suspected nocturnal seizures, or insomnia refractory to adequate treatment.

SLEEP HYGIENE

19. **Components of sleep hygiene:** a **fixed wake-up time seven days a week**; get out of bed when unable to sleep; **no clock-watching**; the bedroom dark, quiet and cool and used only for sleep and sex; avoid caffeine after midday; avoid alcohol and nicotine in the evening; avoid heavy meals and large fluid volumes before bed; **limit naps to under 30 minutes and before 3 pm**; regular daytime exercise but not within 2–3 hours of bedtime; **bright light exposure in the morning**; a consistent wind-down routine; and no screens in bed.
20. **In hospitals and care homes** — reduce night-time noise and light, cluster nursing care and avoid routine nocturnal observations and medication rounds, keep the room cooler at night, increase daytime activity and daylight exposure, and treat nocturia. Sleep hygiene is listed among the environmental modifications of the **Acute Care for Elders (ACE) unit**.
21. **Sleep hygiene alone is inadequate as monotherapy for chronic insomnia.** It is one component of CBT-I, not a treatment in its own right — a frequent and heavily penalised error in examinations.

MANAGEMENT

22. **Treat the underlying cause first** — analgesia and joint care; optimise heart failure and COPD; treat nocturia, prostatism and overactive bladder; treat reflux; treat depression and anxiety; correct **iron deficiency in RLS (target ferritin above 75 ng/mL)**; CPAP for sleep apnoea; move diuretics and corticosteroids to the morning; and review every drug on the list.
23. **Cognitive behavioural therapy for insomnia (CBT-I) is first-line for chronic insomnia in all ages**, including older adults and those with comorbidity, delivered face-to-face, in groups, or digitally. Five components:
    • **Stimulus control** — bed only for sleep; go to bed only when sleepy; leave the bed after about 20 minutes of wakefulness; fixed rising time; no daytime naps.
    • **Sleep restriction** — restrict time in bed to actual sleep time (minimum 5–5.5 hours), then titrate upward as efficiency exceeds 85–90%. **Use with caution in fall-prone patients, epilepsy, bipolar disorder and untreated OSA** because of transient daytime sleepiness.
    • **Relaxation training** — progressive muscular relaxation, diaphragmatic breathing, mindfulness.
    • **Cognitive restructuring** — correcting catastrophic beliefs about sleep loss and unrealistic expectations.
    • **Sleep hygiene education.**
    CBT-I benefits are **sustained after treatment ends**, unlike hypnotics.
24. **Circadian rhythm disorders and sundowning — there is currently no evidence for drug treatment**. Non-pharmacological measures, principally **good sleep hygiene practice and especially bright light therapy**, are first-line, supported by structured daytime activity, exercise and social engagement.
25. **Principles of drug therapy** — hypnotics are an adjunct, for the shortest possible period (generally **up to 4 weeks**); **start low and go slow**; monitor target symptoms and side effects closely and adjust; **never use long-acting benzodiazepines in geriatric patients**; and anticipate **rebound insomnia** on stopping benzodiazepines and melatonin used regularly.
26. **Agents** — benzodiazepines: lorazepam 0.5–2.5 mg and temazepam 7.5–30 mg (intermediate acting, half-life 8–20 hours). Benzodiazepine receptor agonists: **zolpidem 5–10 mg** (short acting), **zaleplon 5–20 mg** (ultra-short), **eszopiclone 1–3 mg** (intermediate). Melatonin receptor agonist: **ramelteon 8 mg**. The **effectiveness of short-term hypnotic use in older people has been questioned**, and the long-term effects of chronic hypnotic use in this population are unknown.
27. **AGS Beers Criteria** — benzodiazepines, "Z-drugs" and first-generation antihistamines (diphenhydramine, hydroxyzine, promethazine) are **potentially inappropriate in adults aged 65 and over** because of cognitive impairment, delirium, falls, fractures and motor-vehicle crashes. **Antipsychotics must not be used for insomnia**; all second-generation antipsychotics carry increased mortality in dementia.
28. **Insomnia in dementia** — benzodiazepines are the most frequently used drugs but cause significant adverse effects. **Low-dose trazodone** is indicated in sleeping disorders, especially with night-time agitation, and long-term use may benefit both sleep parameters and cognitive decline in Alzheimer's disease. **Mirtazapine 15 mg showed no improvement in nocturnal sleep duration or efficiency and increased daytime sleepiness. Suvorexant**, an orexin receptor antagonist, improves total sleep time and cognitive performance in probable Alzheimer's dementia with insomnia. Trials of **lemborexant** in Alzheimer's disease and **nelotanserin** for REM sleep behaviour disorder in dementia with Lewy bodies are under way.
29. **Deprescribing hypnotics** — gradual dose tapering (about 25% every 1–2 weeks) **combined with CBT-I** is the most

effective approach; direct-to-patient educational deprescribing (EMPOWER-type interventions) works and should be offered to every long-term hypnotic user.

LATEST UPDATES (2023–PRESENT)

- **European Insomnia Guideline update (European Sleep Research Society, Riemann et al., November 2023)** — **CBT-I, face-to-face or digital, is unequivocally first-line** for adults. Where CBT-I is unavailable or ineffective: benzodiazepines, benzodiazepine receptor agonists and low-dose sedating antidepressants for **short-term use only (up to 4 weeks)**; **daridorexant** may be used for longer, generally up to **3 months** and in individual cases up to **1 year**; **prolonged-release melatonin 2 mg** may be used for up to 3 months in those **older than 55 years**. **Antihistamines, antipsychotics and phytotherapeutic agents are not recommended**, and melatonin generally is not recommended except where circadian factors are involved.
- **Dual orexin receptor antagonists (DORAs)** are the most significant recent advance — **suvorexant, lemborexant and daridorexant**. They block orexin-1 and orexin-2 receptors, reducing the wake drive rather than sedating through GABA. **Daridorexant has the shortest half-life (~8 hours, versus 12 for suvorexant and 17–19 for lemborexant)**, which in theory reduces next-day impairment.
- **Efficacy in older adults** — a secondary analysis of the phase 3 daridorexant trial (n = 364 aged 65 and over) showed **similar reductions in wake after sleep onset and latency to persistent sleep, similar increases in self-reported total sleep time, and similar improvement in daytime functioning (IDSIQ), with a comparable safety profile, in those aged 65 and over and under 65**. A 2026 systematic review and meta-analysis of DORAs in older adults confirmed reduced WASO and latency and increased total sleep time at 1 and 3 months, with a **favourable safety profile for daridorexant**; lemborexant and suvorexant had higher rates of treatment-emergent adverse events without an increase in serious events.
- **Cautions with DORAs** — contraindicated in narcolepsy; avoid with potent CYP3A4 inhibitors or inducers; caution in severe hepatic impairment and (for somnolence) severe renal impairment; adverse effects include somnolence, headache, nightmares, sleep paralysis and hypnagogic hallucinations. Post-marketing data suggest low abuse and overdose potential and no evidence of physical dependence.
- A **2025 European expert consensus on switching and deprescribing hypnotics** notes that **daridorexant and prolonged-release melatonin 2 mg require no special deprescribing protocol**, unlike benzodiazepines and Z-drugs.
- **Sleep and dementia risk** — suvorexant reduced cerebrospinal fluid p-tau181/tau181 ratio by 10–15% and Aβ42 by 10–20% in cognitively unimpaired adults, supporting the **glymphatic clearance hypothesis** that improving sleep may modify amyloid and tau burden. This remains proof-of-concept, not clinical indication.
- **ICSD-3-TR (2023)** retains the merged category of **chronic insomnia disorder**, abolishing the old primary/secondary distinction — **insomnia is treated in its own right even when comorbid**, because treating the comorbidity alone rarely resolves it.
- **Vornorexant** and other next-generation orexin antagonists are in late-stage development; digital CBT-I applications (including validated free tools such as CBT-I Coach) are recommended to widen access.

CURRENT PRACTICE UPDATE

Issue	Textbook position	Current evidence / practice
First-line treatment	Non-pharmacological measures tried before drugs; several drug alternatives listed	**CBT-I is definitively first-line** (ESRS 2023), face-to-face or **digital**; sleep hygiene alone is insufficient as monotherapy
Drug of choice	Benzodiazepines and BzRAs (zolpidem, zaleplon, eszopiclone), ramelteon	**Dual orexin receptor antagonists** preferred where a drug is needed; **daridorexant** has the shortest half-life and best-studied efficacy and safety in those aged 65 and over
Duration of therapy	Short-term use; long-term effects unknown; rebound insomnia expected	BZD/BzRA still **≤4 weeks**; **daridorexant up to 3 months**, extendable to 1 year in individual cases; PR melatonin 2 mg up to 3 months if aged over 55
Classification	Insomnia framed as a symptom of an underlying cause	**ICSD-3-TR** merged category — **chronic insomnia disorder is treated in its own right** even when comorbid
Insomnia in dementia	Benzodiazepines commonly used; low-dose trazodone for night-time agitation	**Suvorexant** improves total sleep time and cognition in probable AD; **mirtazapine 15 mg is ineffective and increases daytime sleepiness**; benzodiazepines to be avoided
Circadian disorders	No evidence for drug treatment	Unchanged — **bright light therapy plus sleep hygiene remain first-line**; structured daytime activity added

EXAM ONE-LINERS

- Ageing reduces the **ability** to sleep, not the **need** for sleep.
- Chronic insomnia = **3 nights a week for 3 months**, despite adequate opportunity, with daytime impairment.
- Core mechanism is **24-hour hyperarousal**, not simple sleep loss.
- **Orexin stabilises the VLPO–arousal flip-flop switch** — hence orexin antagonists as hypnotics.
- **3-P model**: predisposing, precipitating, perpetuating — CBT-I targets the perpetuating factors.
- Alcohol fragments sleep; it is a sedative, not a hypnotic.
- Naps over **30 minutes** and time spent in bed awake are the two commonest perpetuating behaviours.
- **CBT-I is first-line**; sleep hygiene alone is not a treatment.
- Sleep restriction: caution in fallers, epilepsy, bipolar disorder and untreated OSA.
- Beers: **avoid benzodiazepines, Z-drugs and first-generation antihistamines** in those aged 65 and over.
- **Daridorexant** — shortest half-life DORA (~8 h); efficacy and safety equal in those over and under 65.
- Bright light therapy is first-line for circadian rhythm disorder and sundowning; no drug is established.
- PSG is not indicated for uncomplicated insomnia.

★ EXTRA RECALL — RECURRING TOPIC

Sleep hygiene mnemonic — "BEDROOM":
Bedtime and rising time fixed · **E**xercise daily but not late · **D**ark, quiet and cool room · **R**estrict naps to under 30 minutes · **O**nly sleep and sex in bed · **O**ut of bed if awake beyond 20 minutes · **M**eals, caffeine, alcohol and nicotine avoided in the evening

CBT-I five pillars — "S-S-R-C-S": Stimulus control · **S**leep restriction · **R**elaxation · **C**ognitive restructuring · **S**leep hygiene education

Numerical anchors: sleep cycle 90–110 min · chronic insomnia = 3 nights/week × 3 months · naps <30 min, before 3 pm · out of bed after 20 min · sleep efficiency target 85–90% · hypnotics ≤4 weeks · PR melatonin 2 mg, age >55, up to 3 months · daridorexant half-life ~8 h (suvorexant 12 h, lemborexant 17–19 h) · RLS ferritin target >75 ng/mL · RLS prevalence ~20% at age 80+.

Examiner traps:

- Writing **sleep hygiene as the treatment** — it is one of five CBT-I components; CBT-I is first-line.
- Prescribing a benzodiazepine without mentioning **Beers**, falls and fracture risk.
- Attributing insomnia to normal ageing and stopping the work-up there.
- Failing to screen for **OSA, RLS/PLMD, RBD, nocturia, pain and depression** before treating.
- Ordering **polysomnography for uncomplicated insomnia**.
- Omitting the caution about sleep restriction in fall-prone patients.
- Not mentioning **deprescribing** for the patient already on long-term hypnotics — this is often the real question.
- Forgetting **bright light therapy** for sundowning and circadian disorder.

> **15-second answer skeleton:** Normal sleep architecture and cycles → age changes (latency up, N3 down, WASO up, phase advance) — ability not need → ICSD-3 definition (3×3 rule) → pathophysiology: **hyperarousal + orexin flip-flop instability + two-process/SCN decline + Spielman 3-P + conditioning** → causes: physical, primary sleep disorders, behavioural, drugs, psychiatric → evaluation: sleep diary, bed-partner history, ISI, PSG only if indicated → treat the cause → **CBT-I first-line (5 pillars)** + BEDROOM sleep hygiene + bright light for circadian → drugs: short-term, Beers cautions, DORAs preferred, trazodone in dementia → deprescribing.

CONDENSED 7-MARK VERSION — "Short note on insomnia in the elderly / sleep hygiene"

1. **Age changes in sleep:** increased sleep latency and awakenings, reduced total sleep time, sleep efficiency and slow-wave sleep, circadian phase advance, more naps. Ability to sleep declines; need does not.
2. **Definition:** difficulty initiating or maintaining sleep, or early awakening, despite adequate opportunity, with daytime impairment; chronic if 3 nights a week for 3 months.
3. **Pathophysiology:** 24-hour hyperarousal (raised cortisol, sympathetic tone, beta EEG); orexin-stabilised VLPO–arousal flip-flop instability; declining Process S and Process C with SCN degeneration and reduced melatonin and light exposure; Spielman's predisposing–precipitating–perpetuating model; conditioned insomnia.
4. **Causes:** pain, orthopnoea/PND, nocturia, reflux; RLS/PLMD, sleep apnoea, RBD; naps over 30 minutes and excess time in bed; caffeine, alcohol, diuretics, steroids, SSRIs, hypnotic withdrawal; depression and anxiety.
5. **Evaluation:** sleep diary 1–2 weeks, bed-partner history, Insomnia Severity Index, Epworth; polysomnography only for suspected apnoea, PLMD, parasomnia or refractory insomnia.
6. **Sleep hygiene (BEDROOM):** fixed rising time, daytime exercise not late, dark/quiet/cool room, naps under 30 minutes, bed only for sleep and sex, out of bed if awake beyond 20 minutes, no evening caffeine, alcohol or heavy meals; morning bright light.
7. **Management:** treat the underlying cause; **CBT-I first-line** (stimulus control, sleep restriction, relaxation, cognitive restructuring, sleep hygiene); bright light therapy for circadian disorder and sundowning; drugs short-term only, avoiding long-acting benzodiazepines, Z-drugs and antihistamines per Beers; low-dose trazodone in dementia; taper and deprescribe long-term hypnotics alongside CBT-I.

3-point current update: (i) **European Sleep Research Society guideline 2023** — CBT-I (including digital) first-line; BZD/BzRA and sedating antidepressants only up to 4 weeks; antihistamines, antipsychotics and phytotherapy not recommended; PR melatonin 2 mg up to 3 months if aged over 55. (ii) **Dual orexin receptor antagonists** (daridorexant, lemborexant, suvorexant) are the major advance; **daridorexant** has the shortest half-life (~8 h) with equal efficacy and safety in those aged 65 and over, and needs no special deprescribing protocol. (iii) **ICSD-3-TR** treats chronic insomnia as a disorder in its own right even when comorbid, and suvorexant's reduction in CSF tau and amyloid links sleep treatment to dementia-risk modification.

6. SLEEP APNOEA / POLYSOMNOGRAPHY — 15 MARKS

DEFINITIONS

1. **Sleep apnoea–hypopnoea syndrome (SAHS)** — repetitive episodes of complete or partial upper-airway obstruction during sleep producing intermittent hypoxaemia, hypercapnia, sympathetic surges and cortical arousals, with consequent daytime symptoms.
2. **AASM event definitions: Apnoea** = fall in airflow of 90% or more for **10 seconds or longer**. **Hypopnoea** = fall in airflow of 30% or more for 10 seconds or longer with either a **3% or greater desaturation** or an arousal. **RERA** = respiratory-effort-related arousal not meeting apnoea or hypopnoea criteria.
3. **Indices: AHI** = (apnoeas + hypopnoeas) per hour of sleep; **RDI** = AHI plus RERAs per hour; **ODI** = oxygen desaturations per hour; **T90** = percentage of sleep time with saturation below 90%; **hypoxic burden** = area under the desaturation curve.
4. **Types** — **obstructive** (respiratory effort present, airflow absent), **central** (both effort and airflow absent — includes Cheyne–Stokes respiration in heart failure and stroke), **mixed**, and **treatment-emergent central apnoea** appearing on PAP therapy.
5. **Severity:** mild AHI 5–15, moderate 15–30, **severe above 30** per hour. Diagnosis of OSA requires **AHI 15 or more**, or **AHI 5 or more with symptoms or associated comorbidity**.

EPIDEMIOLOGY

6. OSA is **the most common sleep-related problem in older adults**. Development is **age-dependent and male-dominant**, and incidence is higher in the obese and in those with **increased neck circumference**. Prevalence of AHI 15 or more approaches **20–30% in those over 65**, and the male-to-female ratio narrows sharply after the menopause. Sleep-disordered breathing is very common in heart failure.
7. **India** — urban adult prevalence is approximately **9–13% in men and 5–7% in women**. Indians develop OSA at a **lower BMI** than Caucasians because of craniofacial morphology (shorter mandible, narrower airway) and central adiposity; therefore use **Asian-Indian cut-offs — overweight BMI 23 kg/m², obesity 25 kg/m², waist circumference 90 cm in men and 80 cm in women**.

PATHOPHYSIOLOGY

8. Four contributing traits ("**PALM**"): **P**crit — anatomical collapsibility of the pharynx; **A**rousal threshold — a low threshold causes premature arousal and ventilatory instability; **L**oop gain — an unstable, over-responsive ventilatory control system; **M**uscle responsiveness — poor reflex recruitment of genioglossus and other dilators.
9. **Age-related contributions** — reduced upper-airway dilator tone and reflex responsiveness, increased pharyngeal fat deposition and airway length, loss of lung-volume tethering of the airway, increased loop gain, blunted arousal and chemoreflex responses, and reduced protective effect of oestrogen in women after the menopause.
10. **Consequences of untreated OSA** — nocturnal hypoxia with associated risk of **cardiac arrhythmias and myocardial and cerebral infarction**; resistant **hypertension** (a recognised secondary cause); heart failure, atrial fibrillation, pulmonary hypertension, type 2 diabetes and metabolic syndrome; **excessive daytime sleepiness with driving and occupational risk**; cognitive impairment and increased dementia risk; depression; and reduced **olfactory identification and discrimination** in proportion to severity (higher AHI and lower mean arterial oxygen saturation), thought to reflect central cholinergic dysfunction.

CLINICAL FEATURES AND SCREENING

11. **Nocturnal** — loud, irregular snoring; witnessed apnoeas; choking or gasping arousals; **waking sweating, anxious and tachycardic**; excessive movement during sleep; nocturia; dry mouth.
    Daytime — morning headache, morning drowsiness, excessive daytime sleepiness, poor concentration, irritability, depressed mood, reduced libido.
12. **Atypical presentation in older adults** — sleepiness may be absent or attributed to age. Instead consider OSA in **nocturia, nocturnal confusion or sundowning, falls, new or worsening cognitive impairment, refractory hypertension, recurrent atrial fibrillation and unexplained pulmonary hypertension**. Obesity is less prominent than in younger patients. **The bed-partner history is essential and is frequently unavailable in widowed or institutionalised elders** — ask nursing staff instead.
13. **Screening instruments — STOP-BANG** (Snoring, Tiredness, Observed apnoea, blood Pressure, BMI over 35, Age over 50, Neck over 40 cm, Gender male; 3–4 intermediate risk, 5 or more high risk). **Note that the age and gender items make almost every older man score positive, so specificity in geriatric practice is poor.** Also the **Epworth Sleepiness Scale** (above 10 abnormal), the Berlin Questionnaire and NoSAS.

POLYSOMNOGRAPHY

14. **Attended, in-laboratory polysomnography is the gold standard.** AASM levels of testing:
    Level 1 — attended, in-laboratory, full-channel PSG.
    Level 2 — unattended full-channel portable study.
    Level 3 — **home sleep apnoea test (HSAT)**, limited channels: airflow, respiratory effort, oximetry, with or without ECG, heart rate and body position.
    Level 4 — one or two channels, usually oximetry alone.

15. **Level-1 PSG channels:**
    • **Sleep staging** — EEG (frontal, central and occipital derivations referenced to the contralateral mastoid: F4-M1, C4-M1, O2-M1), **electro-oculogram** (two channels) and **submental EMG**.
    • **Airflow** — nasal pressure transducer (for hypopnoea) and oronasal thermistor (for apnoea).
    • **Respiratory effort** — thoracic and abdominal respiratory inductance plethysmography belts.
    • **Gas exchange** — pulse oximetry, with transcutaneous or end-tidal carbon dioxide where hypoventilation is suspected.
    • **Others** — ECG, **bilateral anterior tibialis EMG** (limb movements), body-position sensor, snore microphone, and **synchronised audio-video** (essential for parasomnias and REM sleep behaviour disorder).
16. **Scoring and report** — scored in **30-second epochs** as stages **W, N1, N2, N3 and R**. The report should include total sleep time, sleep efficiency, sleep-onset and REM latency, wake after sleep onset, percentage of each stage, **arousal index**, **AHI overall and in supine and REM sleep separately**, RDI, ODI, minimum and mean oxygen saturation, T90, periodic limb movement index, and cardiac rhythm.
17. **Indications for PSG** — suspected sleep apnoea; suspected **periodic limb movement disorder**; suspected **REM sleep behaviour disorder** or injurious parasomnia; suspected nocturnal seizures; narcolepsy (followed next day by the multiple sleep latency test); **PAP titration**; assessment of treatment failure or persistent symptoms despite therapy; and insomnia refractory to adequate treatment. **PSG is not indicated for uncomplicated insomnia, uncomplicated circadian rhythm disorders, or restless legs syndrome, which is a clinical diagnosis.**
18. **Home sleep apnoea testing** is acceptable in **uncomplicated adults with a high pre-test probability of moderate-to-severe OSA**. It is **not appropriate** where there is significant cardiorespiratory disease, suspected hypoventilation, neuromuscular disease, chronic opioid use, or suspicion of a non-OSA sleep disorder — all of which are common in geriatric practice. HSAT **underestimates the AHI** because total **recording** time rather than total **sleep** time is the denominator; therefore a negative or technically inadequate HSAT in a symptomatic patient **must be followed by in-laboratory PSG**.
19. **Practical problems in older adults** — the first-night effect, difficulty tolerating electrodes, mobility and transfer problems, nocturia interrupting the study, risk of delirium in an unfamiliar environment, and intrinsically fragmented sleep, all of which reduce the reliability of a single night's study.

MANAGEMENT

20. **General and behavioural** — weight reduction; **avoid evening alcohol, benzodiazepines and opioids**, which worsen upper-airway collapse and blunt arousal; smoking cessation; treat nasal obstruction and hypothyroidism; **positional therapy** for supine-predominant disease; optimise heart failure; and give explicit **driving advice** — untreated OSA with sleepiness is a driving hazard and, in many jurisdictions, a reportable one.
21. **Continuous positive airway pressure (CPAP) is first-line** and the standard of care for moderate-to-severe OSA. Auto-titrating PAP is acceptable in uncomplicated disease; **bilevel PAP** for hypoventilation or pressure intolerance. CPAP improves sleepiness, quality of life, blood pressure and mood; **adherence is the limiting factor** (conventional target: at least 4 hours a night on at least 70% of nights). Older patients adhere at least as well as younger ones when adequately supported.
22. **Oral appliances** — **mandibular advancement devices** for mild-to-moderate OSA, CPAP intolerance or supine-predominant disease. They require **adequate dentition**, a major limitation in older adults, and are contraindicated in significant temporomandibular joint disease.
23. **Surgery** — uvulopalatopharyngoplasty, nasal surgery, tonsillectomy and maxillomandibular advancement; results are poorer and risks higher in the elderly, so surgery is reserved for carefully selected patients with a correctable obstruction. **Hypoglossal nerve stimulation** is an option for CPAP-intolerant patients within defined AHI and BMI limits and in the absence of complete concentric palatal collapse.
24. **Central sleep apnoea and Cheyne–Stokes respiration** — optimise heart-failure therapy first; consider nocturnal oxygen; **adaptive servo-ventilation is contraindicated in symptomatic heart failure with an ejection fraction of 45% or less** (SERVE-HF trial showed increased mortality); phrenic nerve stimulation is an option in selected patients.

LATEST UPDATES (2023–PRESENT)

- **SURMOUNT-OSA (NEJM 2024)** — two 52-week phase 3 trials of **tirzepatide** (a dual GIP/GLP-1 receptor agonist, 10 or 15 mg weekly) in adults with moderate-to-severe OSA and obesity (mean baseline AHI about 50). **Trial 1 (not on PAP): AHI fell 25.3 per hour versus 5.3 with placebo. Trial 2 (on PAP): AHI fell 29.3 per hour versus 5.5.** Mean reduction in AHI up to **62.8%**; **43.0% and 51.5% respectively met criteria for disease resolution**. Tirzepatide also reduced body weight (about 18–20%), **hypoxic burden, hs-CRP and systolic blood pressure**, and improved sleep-related patient-reported outcomes. Adverse events were predominantly gastrointestinal.
- **FDA approval, 20 December 2024** — tirzepatide (Zepbound) became the **first ever prescription medicine approved for moderate-to-severe obstructive sleep apnoea in adults with obesity**, used with a reduced-calorie diet and increased physical activity. **CPAP remains first-line**; tirzepatide is an option for patients unable or unwilling to use PAP, or as an adjunct in those with obesity who remain symptomatic on PAP.
- **Other pharmacological agents remain investigational** — **AD109** (aroxybutynin plus atomoxetine, targeting upper-airway muscle tone; phase 3 SynAIRgy and LunAIRo, 2025) and **sulthiame** (carbonic anhydrase inhibitor, targeting loop gain). Neither is approved.
- **AASM Scoring Manual version 3 (2023)** — updated hypopnoea, arousal and paediatric rules, and formal recognition of alternative metrics.
- **Beyond the AHI** — the AHI correlates poorly with symptoms and cardiovascular risk. **Hypoxic burden and sleep-**

apnoea-specific hypoxic burden (SASHB), arousal burden and heart-rate response are prognostically superior, and the field is moving to **phenotype- and endotype-driven therapy based on the PALM traits** rather than AHI-only decision-making.

- **CPAP and cardiovascular outcomes** — SAVE and RICCADSA did not show reduction in major cardiovascular events in intention-to-treat analyses, but on-treatment analyses show benefit in adherent users; the pragmatic message is that CPAP is prescribed for **symptoms, quality of life and blood pressure**, with cardiovascular benefit dependent on adherence.
- **Consumer technology** — FDA-cleared **sleep apnoea notification features on smartwatches and rings** (2024 onwards) are increasing case detection; a positive alert requires confirmatory HSAT or PSG and must not be treated as a diagnosis.

CURRENT PRACTICE UPDATE

Issue	Textbook position	Current evidence / practice
Treatment options	CPAP, dental appliances and uvulopalatopharyngoplasty	CPAP still first-line; add **tirzepatide (FDA-approved December 2024)**, **hypoglossal nerve stimulation**, positional therapy and maxillomandibular advancement
Diagnostic testing	Refer to a sleep centre for further work-up	Tiered testing — **HSAT** for uncomplicated high-probability cases; **attended PSG** mandatory where comorbidity, hypoventilation, opioid use or another sleep disorder is suspected
Severity metric	Apnoea–hypopnoea index	AHI retained for diagnosis, but **hypoxic burden and arousal burden** better predict cardiovascular outcome; **endotype-driven therapy** emerging
Pharmacotherapy	No effective drug therapy	**Tirzepatide** reduces AHI by 25–29 events per hour with disease resolution in 43–51%; AD109 and sulthiame investigational
Central apnoea in heart failure	Not specifically addressed	**Adaptive servo-ventilation contraindicated when LVEF is 45% or less** (SERVE-HF mortality signal)
Case detection	Bed-partner report of loud snoring prompts referral	Bed-partner history often unavailable in older adults; use atypical presentations, and confirm **wearable-generated alerts** with formal testing

EXAM ONE-LINERS

- Apnoea = 90% airflow reduction for 10 seconds; hypopnoea = 30% reduction for 10 seconds with 3% desaturation or arousal.
- Severity: mild 5–15, moderate 15–30, **severe above 30**.
- OSA is **age-dependent and male-dominant**; prevalence approaches 20–30% over 65.
- Indians develop OSA at a **lower BMI** — use Asian cut-offs (BMI 23/25, waist 90/80 cm).
- PALM traits: **P**crit, **A**rousal threshold, **L**oop gain, **M**uscle responsiveness.
- In older adults, suspect OSA from **nocturia, nocturnal confusion, falls, refractory hypertension and cognitive decline**, not necessarily sleepiness.
- STOP-BANG loses specificity in the elderly because age and male sex already score.
- Level 1 PSG needs **EEG + EOG + submental EMG** to stage sleep — that is what an HSAT lacks.
- HSAT **underestimates AHI** (recording time, not sleep time, is the denominator).
- PSG is not indicated for uncomplicated insomnia or for restless legs syndrome.
- CPAP adherence target: **4 hours a night on 70% of nights**.
- Mandibular advancement devices need **teeth** — a real limitation in old age.
- **ASV is contraindicated in heart failure with LVEF 45% or less**.
- **Tirzepatide** (SURMOUNT-OSA; FDA approved December 2024) is the first drug approved for OSA — AHI down 25–29 per hour, resolution in 43–51%.

CONDENSED 7-MARK VERSION — "Short note on obstructive sleep apnoea / polysomnography"

1. **Definition:** repetitive complete (apnoea) or partial (hypopnoea) upper-airway obstruction in sleep causing intermittent hypoxaemia and arousals. Apnoea = 90% airflow fall for 10 s; hypopnoea = 30% fall for 10 s with 3% desaturation or arousal.
2. **Severity:** AHI mild 5–15, moderate 15–30, severe above 30. Diagnosis: AHI 15 or more, or 5 or more with symptoms.
3. **Pathophysiology:** PALM traits — airway collapsibility, low arousal threshold, high loop gain, poor dilator muscle responsiveness; ageing adds reduced muscle tone, pharyngeal fat, reduced lung-volume tethering and blunted chemoreflexes.

4. **Features:** loud irregular snoring, witnessed apnoeas, waking sweating and tachycardic, morning headache and drowsiness, daytime sleepiness. In older adults, presentation may instead be nocturia, nocturnal confusion, falls, refractory hypertension or cognitive decline.
5. **Complications:** arrhythmia, myocardial and cerebral infarction, resistant hypertension, heart failure, atrial fibrillation, pulmonary hypertension, diabetes, cognitive impairment, road-traffic risk.
6. **Polysomnography:** gold standard, Level 1 attended in-laboratory. Channels — EEG, EOG and submental EMG for staging; nasal pressure and thermistor for airflow; thoracoabdominal belts for effort; oximetry; ECG; tibialis EMG; position, snore and video. Scored in 30-second epochs; report AHI (total, supine, REM), RDI, ODI, arousal index, minimum and mean saturation, T90, PLM index. Level 3 home testing for uncomplicated high-probability cases only; it underestimates AHI.
7. **Management:** weight loss, avoid evening alcohol and sedatives, positional therapy, treat nasal obstruction, driving advice. **CPAP first-line** (adherence 4 h/night on 70% of nights); mandibular advancement device if mild-moderate or CPAP-intolerant and dentition adequate; selected surgery or hypoglossal nerve stimulation. ASV contraindicated if LVEF 45% or less.

3-point current update: (i) **SURMOUNT-OSA (NEJM 2024)** — tirzepatide reduced AHI by 25–29 events per hour with disease resolution in 43–51%, and on **20 December 2024 the FDA approved it as the first drug for moderate-to-severe OSA with obesity**; CPAP remains first-line. (ii) The field is moving **beyond the AHI** to hypoxic burden and endotype-driven (PALM) therapy, with AASM Scoring Manual v3 (2023) updating event rules. (iii) **FDA-cleared wearable sleep-apnoea notifications** are increasing detection but require confirmatory HSAT or PSG; in India, use **Asian BMI and waist cut-offs** because OSA occurs at lower BMI.

End of module — Elder abuse · Advance care planning · End-of-life care · Transitional care · Insomnia · Sleep apnoea
Next in series: cardiovascular, respiratory and neurology (Parkinson's disease)

THE TAMIL NADU DR. M.G.R. MEDICAL UNIVERSITY · BRANCH XVI

MD Geriatrics Paper IV

Social and Preventive Geriatrics including Rehabilitation and Advances in Geriatrics

Sub. Code 2060 / 4044

20	240	174	18
EXAM SESSIONS	QUESTIONS ASKED	UNIQUE AFTER DEDUP	THEMED SECTIONS

INSIDE THIS DOCUMENT

A How this paper is examined
Question counts across all four papers, the mark scheme, where the marks actually are, and the most-repeated topics.

B Master question list
All 240 questions ever set, de-duplicated to 174 unique entries and grouped into 18 themed sections with repeat and mark-format badges.

C Ebenezer bank — not yet asked
58 Paper IV topics that appear in the Ebenezer question bank but have never been set by the university.

D Predicted questions
Most likely essays and short notes for the next session, with the reasoning behind each call.

Complete de-duplicated question bank, organised by system and theme

March 2009 – October 2025 university papers · Ebenezer's Geriatric Question Bank · Frequency analysis and predicted questions

Contents

Part A · How this paper is examined . . . 3

Question counts across all four papers . . . 3

The Paper IV mark scheme . . . 4

Where the marks actually are . . . 4

The most-repeated topics . . . 5

What has changed in the examiner's style . . . 5

Part B · Master question list . . . 7

1. Biology of Ageing, Healthy Ageing & Demography . . . 7

2. Comprehensive Geriatric & Functional Assessment . . . 7

3. Health Systems, National Programmes, Policy & Social Geriatrics . . . 8

4. Ethics, Law, Palliative & End-of-Life Care, Pain and Communication . . . 9

5. Rehabilitation, Assistive Technology & Exercise . . . 10

6. Falls, Immobility & Pressure Ulcers . . . 11

7. Bone, Joint & Rheumatology . . . 11

8. Nutrition, Sarcopenia & Frailty . . . 12

9. Cognition, Dementia, Delirium, Sleep & Psychogeriatrics . . . 12

10. Neurology . . . 13

11. Cardiovascular Medicine . . . 14

12. Respiratory Medicine . . . 16

13. Renal, Genitourinary, Continence & Men's Health . . . 16

14. Endocrine & Metabolic . . . 17

15. Infection, Sepsis & Immunisation . . . 17

16. Oncology, Haematology & Cancer Screening . . . 18

17. Gastroenterology & Hepatology . . . 18

18. Perioperative Care, Procedures & Miscellaneous . . . 19

Part C · Ebenezer's bank — Paper IV topics not yet asked . . . 20

Part D · Predicted questions . . . 23

Paper IV · most likely 15-mark essays . . . 23

Paper IV · most likely 7-mark notes . . . 24

A model predicted Paper IV . . . 24

Cross-paper watchlist — Papers I, II and III . . . 26

Part A · How this paper is examined

HOW TO READ THIS DOCUMENT

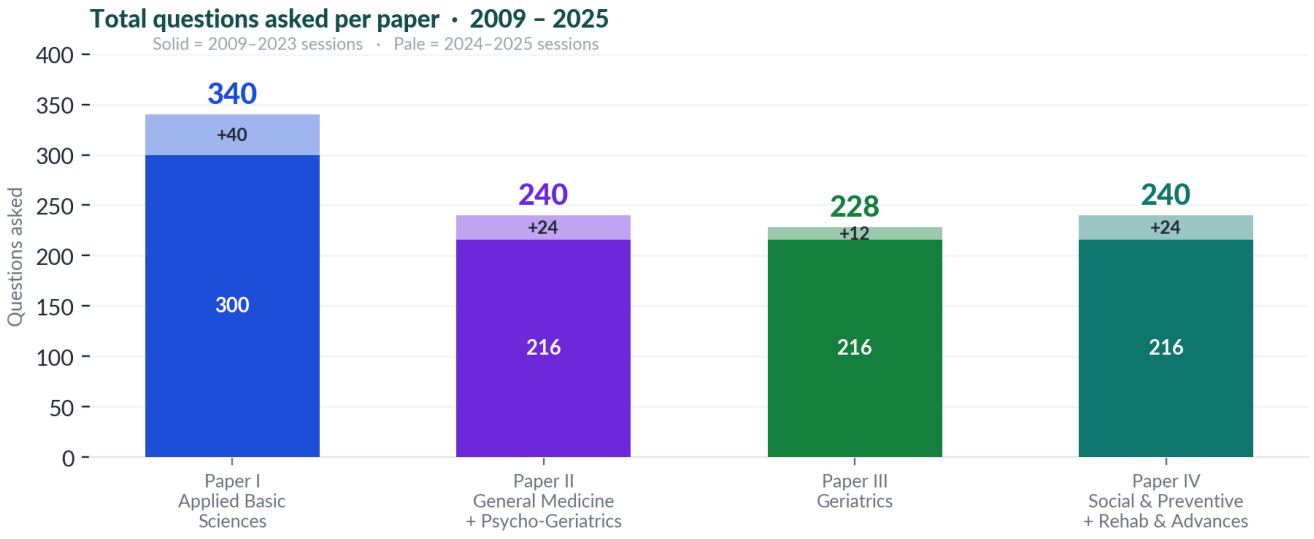
Every question ever set in Paper IV between March 2009 and October 2025 has been extracted, merged with the Ebenezer question bank, stripped of year tags and repeats, and re-grouped by system and theme. Nothing has been dropped: the 240 original question slots have each been traced into one of the 174 unique entries in Part B, and the mapping was verified programmatically before this document was generated.

Question counts across all four papers

The four papers do not carry equal question loads. Paper I uses a six-subject short-note format with twenty questions per session, while Papers II, III and IV each use two long essays plus ten short notes — twelve questions per session. Counts below are taken from the question papers available in the source archive.

Paper	Sub. Code	Sessions available	Questions per session	Total questions
Paper I — Applied Basic Sciences	2059 / 4041	17	20 (4+4+3+3+3+3)	**340**
Paper II — General Medicine incl. Psycho-Geriatrics	2056 / 4042	20	12 (2 essays + 10 notes)	**240**
Paper III — Geriatrics	2057 / 4043	19	12 (2 essays + 10 notes)	**228**
Paper IV — Social & Preventive Geriatrics	2060 / 4044	20	12 (2 essays + 10 notes)	**240**
All four papers combined	—	76	—	**1,048**

Paper I is missing the October 2013, 2014, 2015 and December 2024 Paper III sessions from the archive; the October 2024 Paper III paper is also absent. Counts reflect what is actually in the source files rather than every session the university has ever conducted.



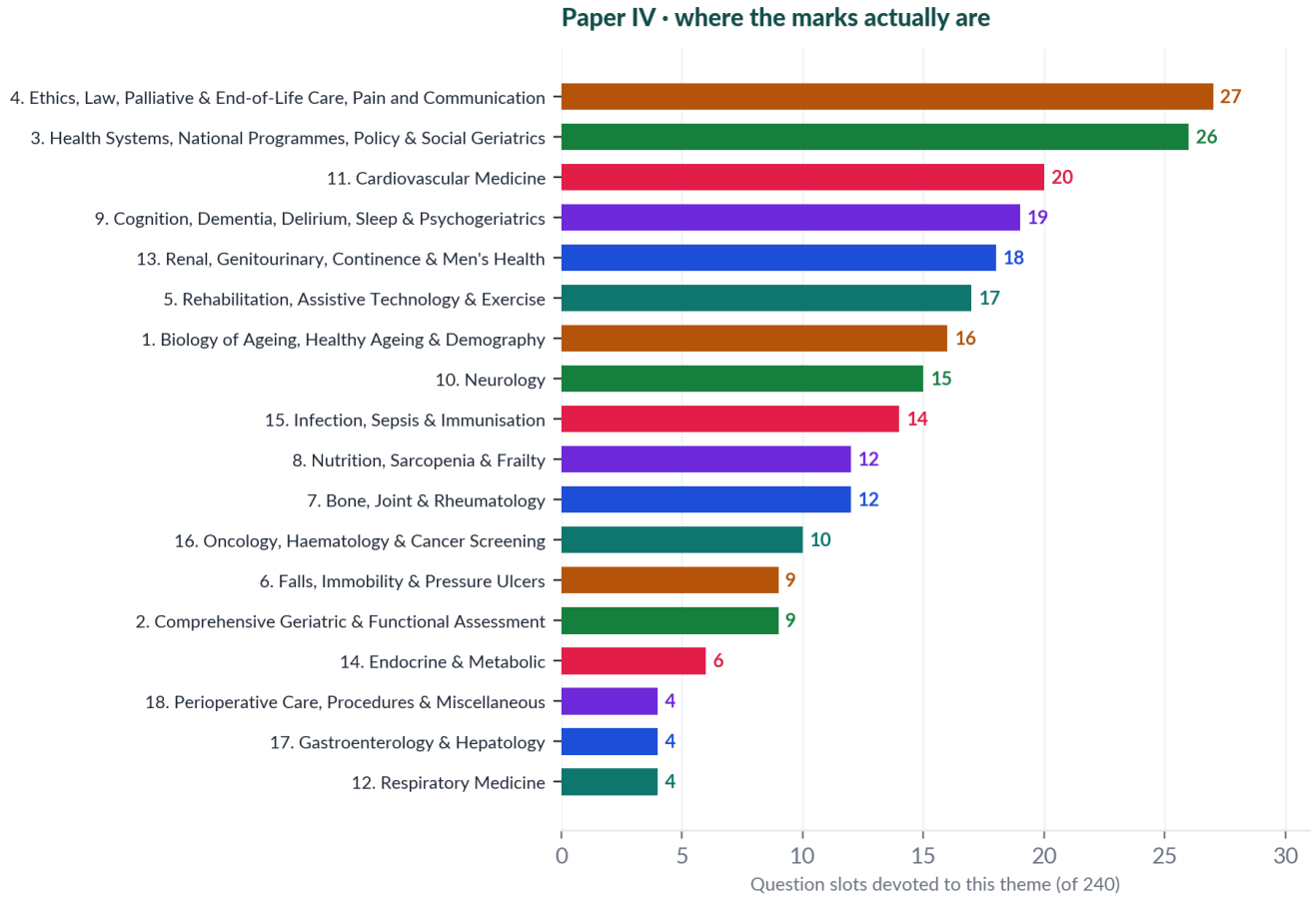
The Paper IV mark scheme

Two formats have been used. Sessions up to March 2010 set two 20-mark essays and ten 6-mark notes. From May 2011 onward the paper has settled into two 15-mark essays and ten 7-mark notes, and this is the format you should calibrate every answer to. Each question in Part B carries its mark-format descriptor so you can size your answers correctly.

Period	Section I	Section II	Answer calibration
2009 – 2010	2 × 20 marks	10 × 6 marks	Historic format — treat 20-mark stems as 15-mark essays
2011 – 2025	2 × 15 marks	10 × 7 marks	Essay: ~11 pages / 35 min. Note: ~4 pages / 10 min

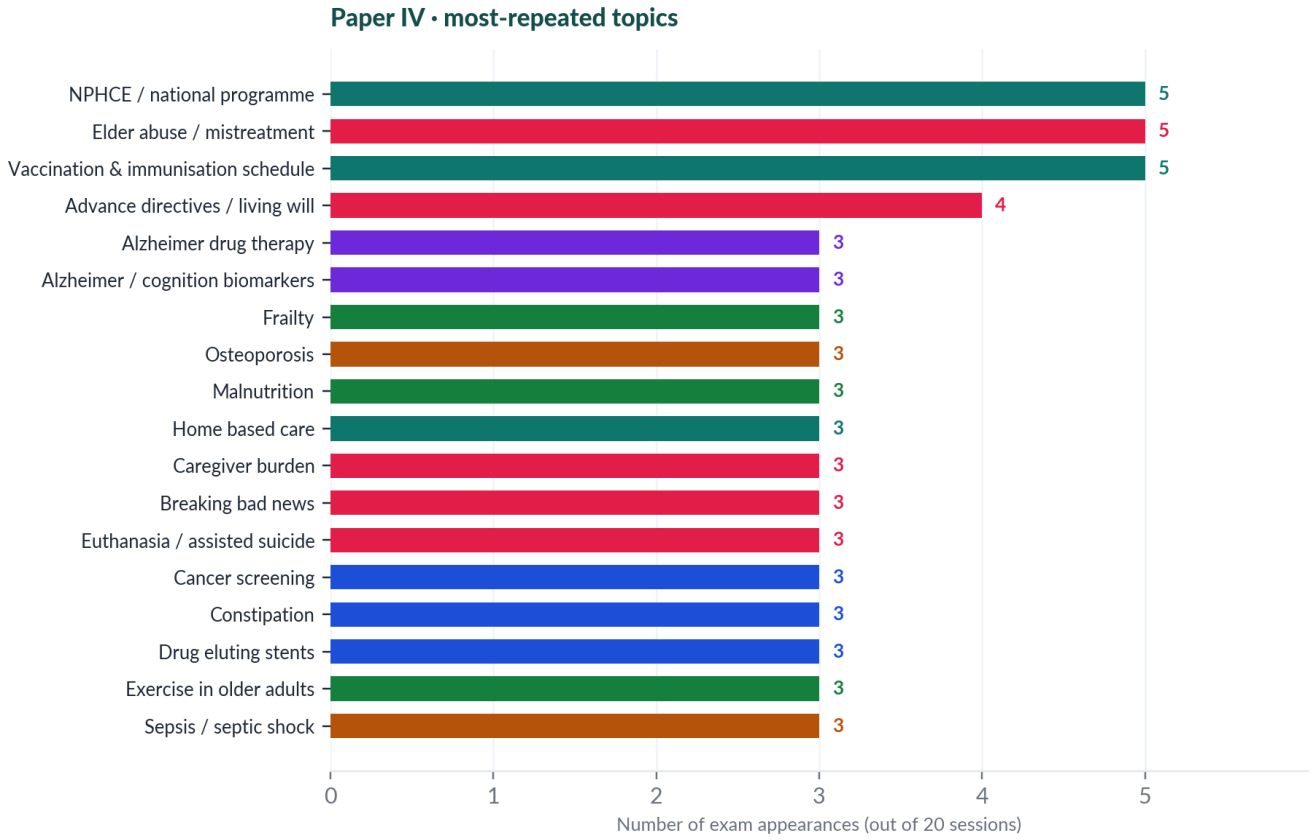
Where the marks actually are

Grouping all 240 question slots by theme shows how lopsided this paper is. Roughly a third of the paper is drawn from just four themes — health systems and national programmes, ethics and end-of-life care, rehabilitation, and cardiovascular medicine. The clinical-medicine sections exist because "Recent Advances" in the paper title is interpreted very broadly by examiners.



The most-repeated topics

Three topics have each appeared five times in twenty sessions: the National Programme for Health Care of the Elderly, elder abuse, and adult immunisation. If you prepare nothing else thoroughly, prepare these. The chart counts distinct exam appearances, not question slots.



What has changed in the examiner's style

SHIFT 1 · FROM TOPIC LABELS TO CLINICAL VIGNETTES

Up to about 2019 most short notes were bare topic labels — "Euthanasia", "Telomere", "Home based care". From 2022 onward a majority of notes are scenarios with a named patient and a specific decision to make: Mr. Atul with a saturation of 60%, Mr. Shivanand who is pale and in failure, the 65-year-old man with three urinary infections in six months. Prepare to write staged answers — differentials, then investigations, then ward management, then discharge planning.

SHIFT 2 · THE EXAMINER NOW ASKS YOU TO BUILD SOMETHING

Recent papers ask candidates to draw up a protocol, prepare a checklist, advise a director on a new service, or head a team. These are not knowledge questions; they are structure questions. Answer them with headings, numbered steps and an explicit evidence base.

SHIFT 3 · "RECENT ADVANCES" MEANS NEWER DRUG CLASSES

A steady stream of questions asks about the newest member of a drug family: newer anticoagulants, newer oral antidiabetics, newer insulins, newer antidepressants, newer Alzheimer drugs, antisclerostin antibodies. Keep a running list of what is new in each class and its geriatric caveats.

SHIFT 4 · INDIAN CONTEXT IS EXPECTED, NOT OPTIONAL

Questions increasingly specify India explicitly — advance directives in India, the social security system in India, how palliative care has evolved in India. Weave in NPHCE, NPOP, the Maintenance and Welfare of Parents and Senior Citizens Act 2007, the Mental Healthcare Act 2017, Elderline 14567, and ICMR and ARDSI data wherever they fit.

Part B · Master question list

All 240 questions ever asked in Paper IV, de-duplicated into 174 unique entries and grouped by system and theme rather than by year. Where a topic has been asked more than once, the wording of the fullest version has been kept and the repeat count is shown as a badge. Mark-format badges tell you what length of answer to prepare.

Badge key

ASKED ×3+ · ASKED ×2 · 15-mark essay · 7-mark note

No badge on repeat count means the question has been asked once. High-yield items — asked three or more times — carry a coral badge.

1 Biology of Ageing, Healthy Ageing & Demography

Theories, biomarkers, anti-ageing science, active ageing, demography

1. Enumerate and explain in detail the theories of ageing.
   ASKED ×2 · 15-mark essay + 7-mark note
2. Biomarkers of ageing.
   7-mark note
3. Anti-ageing interventions.
   7-mark note
4. Calorie restriction — role of caloric restriction in ageing.
   ASKED ×2 · 7-mark note
5. Telomere.
   7-mark note
6. Sirtuins in ageing and disease.
   7-mark note
7. Interleukin-6.
   7-mark note
8. Immunosenescence / immune senescence and its relevance to vaccination in the elderly.
   ASKED ×2 · 7-mark note
9. Interventions for healthy ageing.
   7-mark note
10. Describe active ageing and how will you achieve it?
    ASKED ×2 · 15-mark essay + 7-mark note
11. Geriatric demography and its implications.
    7-mark note
12. Geriatric syndromes.
    7-mark note

2 Comprehensive Geriatric & Functional Assessment

CGA, functional scales, disability assessment, decisional capacity

13. Write in detail on Comprehensive Geriatric Assessment and its usefulness in geriatric practice.
ASKED ×2 15-mark essay

14. Discuss the functional assessment in an 85-year-old lady.
15-mark essay

15. Barthel's Index of Activities of Daily Living.
7-mark note

16. Physical Self-Maintenance Scale.
6-mark note

17. Integrated care of older people (ICOPE).
15-mark essay

18. Discuss the causes of chronic disability in the elderly. Describe the methods of its prevention and management.
15-mark essay

19. What is decisional capacity? What are the factors affecting it and how would one assess decisional capacity in an older person?
ASKED ×2 7-mark note

3 Health Systems, National Programmes, Policy & Social Geriatrics

NPHCE, NPOP, long-term care, home care, day care, social work, service models

20. National Programme for Health Care of the Elderly (NPHCE) — background on which the programme was based, vision, objectives and expected outcomes; packages of services at different levels of care and the activities that centres must perform. As doctor in charge of a District Hospital, which components would you implement and how would you put theory into practice?
ASKED ×5 15-mark essay + 7-mark note

21. National Policy on Older Persons (NPOP) and the various existing beneficiary schemes for the elderly.
ASKED ×2 7-mark note

22. Role of Government in providing health care and economic security for older people.
6-mark note

23. Describe the social security system for older people in India.
ASKED ×2 15-mark essay + 7-mark note

24. Long term care.
ASKED ×2 7-mark note + 6-mark note

25. Community based long term care services.
7-mark note

26. Home based care.
ASKED ×3 7-mark note

27. House call programme.
6-mark note

28. Need for day hospitals in geriatric practice.
7-mark note

29. The director of your hospital is keen on starting a day care centre for older adults and seeks your opinion. Discuss the role, benefits and logistics of running a day care centre facility for older adults.
7-mark note

30. Hospice and respite care.
ASKED ×2 7-mark note

31. Ethno-geriatrics.
7-mark note

32. Role of geriatric social workers.
7-mark note

33. Geriatric Nurse Practitioner — discuss their role in the health care team.
7-mark note

34. Enumerate and briefly explain the components of the Hospital Elder Life Program (HELP).
7-mark note

35. Write a short note on the history of the "Stroke Clinics". Why were they set up and what issues do they address?
7-mark note

4 Ethics, Law, Palliative & End-of-Life Care, Pain and Communication

Elder abuse, advance directives, euthanasia, breaking bad news, palliative care, caregiver burden

36. Elder abuse / mistreatment of the elderly — epidemiology, types and different forms, risk factors, identification, diagnosis and management. Discuss the role of the social worker in identifying elder mistreatment.
ASKED ×5 15-mark essay + 7-mark note

37. Advance directives — current status in India.
ASKED ×4 7-mark note

38. What is a living will? What is the procedure to write a living will in India and how is it executed?
7-mark note

39. Euthanasia and physician-assisted suicide.
ASKED ×3 7-mark note

40. Informed consent.
7-mark note

41. How will you communicate bad news? / Breaking bad news.
ASKED ×3 7-mark note

42. Recognition and management of the terminally ill patient — e.g. a patient diagnosed with end-stage lung cancer with brain and liver metastases. What are all the issues that need to be addressed in the care of the terminally ill?
ASKED ×2 15-mark essay

43. What is palliative care? What are its principles? Name interventions in palliative care which would decrease the suffering of a person with painful bone metastasis, nausea, vomiting, constipation and breathlessness. How has this aspect of medical science evolved in India?
15-mark essay

44. Classify the types of pain. Discuss the pathophysiology, clinical evaluation and management of chronic or persistent pain in the elderly, and the role of palliative care in geriatric practice.
ASKED ×2 15-mark essay

45. End of life care issues.
7-mark note

46. Bereavement.
7-mark note

47. Caregiver burden / caregiver stress / apathy — what is it and how will you manage it?
ASKED ×3 7-mark note

5 Rehabilitation, Assistive Technology & Exercise

Rehab principles, the interdisciplinary team, aids and appliances, exercise, swallow

48. Definition, evaluation and components of rehabilitation in the elderly.
15-mark essay

49. Assessment of disability in older people and rehabilitation.
15-mark essay

50. Community based rehabilitation.
7-mark note

51. You are chosen to head the interdisciplinary team on a rehabilitation ward with neurology rehabilitation patients, post-orthopaedic procedure patients and patients deconditioned by comorbidities. Whom would you choose to be part of your team? Enumerate their job responsibilities. How would you address conflict management and care planning?
15-mark essay

52. Inputs from the occupational therapist on the team caring for the older person are not clearly understood. Highlight the role of the occupational therapist and how his or her presence can affect the care of the patient on the ward and at discharge.
7-mark note

53. Stroke rehabilitation.
7-mark note

54. Prior to initiating long term rehabilitation in a patient with stroke, what are the factors you would take into consideration before admitting such a patient into this facility?
7-mark note

55. Rehabilitation of a patient with osteoarthritis of the knee joints.
7-mark note

56. How will you rehabilitate an older adult with low vision?
7-mark note

57. Mobility aids / assistive devices for the elderly.
ASKED ×2 7-mark note

58. Artificial limb.
6-mark note

59. Gadgets which will improve the quality of life in the older person.
7-mark note

60. Exercise in the elderly — exercises in older adults: beneficial or harmful?
ASKED ×3 7-mark note + 6-mark note

61. Assessment of swallow — when, how, by whom and with what.
7-mark note

6 Falls, Immobility & Pressure Ulcers

Fall risk, prevention, sensor technology, immobility, pressure sores

62. Discuss the etiopathogenesis, risk factors, assessment, adverse consequences, prevention and management of falls in older adults.
15-mark essay

63. Assessment of fall risk and prevention of falls in the elderly.
15-mark essay

64. Fall risk assessment — tools for falls risk assessment.
ASKED ×2 7-mark note

65. How will you prevent falls in the elderly?
7-mark note

66. Role of sensor devices in the prevention of falls.
7-mark note

67. What are the causes of immobility in an elderly person? Write about the management and complications.
15-mark essay

68. Pressure sores — enumerate the prevention of pressure sores in an elderly bedbound patient. What are the stages of a pressure sore and the common sites involved? How is it preventable and what is the appropriate treatment for each stage?
ASKED ×2 7-mark note

7 Bone, Joint & Rheumatology

Osteoporosis, fracture prevention, Paget's, arthritis, vasculitis, myositis

69. Osteoporosis — definition, predisposing and risk factors, pathogenesis, diagnosis, prevention and management including newer interventions. Also elaborate on male osteoporosis and premenopausal osteoporosis.
ASKED ×3 15-mark essay + 7-mark note

70. Antisclerostin antibodies in the treatment of osteoporosis.
7-mark note

71. Prevention of fractures.
7-mark note

72. DEXA scan.
7-mark note

73. Paget's disease.
7-mark note

74. Osteoarthritis — pathogenesis, diagnosis and treatment.
7-mark note

75. Temporal arteritis — diagnosis, clinical features, treatment and prognosis.
7-mark note

76. Diffuse idiopathic skeletal hyperostosis (DISH) — presentation, investigations and treatment options.
7-mark note

77. Dermatomyositis in the elderly — presentation, associated comorbidities, laboratory investigations, treatment options and prognosis.
7-mark note

78. Role of bisphosphonates in osseous metastases.
7-mark note

8 Nutrition, Sarcopenia & Frailty

Malnutrition, nutritional assessment, anorexia, obesity, sarcopenia, frailty

79. Discuss the various nutritional disorders / malnutrition in older adults — causes, approach to diagnosis, assessment and management.
ASKED ×3 20-mark essay + 15-mark essay + 7-mark note

80. Nutritional assessment in older persons.
ASKED ×2 7-mark note

81. Anorexia of ageing.
7-mark note

82. Discuss obesity in the elderly and its management.
20-mark essay

83. Discuss sarcopenia in ageing. Briefly write about the diagnostic criteria, investigations and management.
15-mark essay

84. Frailty is a common syndrome in older adults — discuss the etiopathogenesis, assessment, management and prevention of frailty.
ASKED ×3 15-mark essay + 7-mark note

85. Compare and contrast the tests used to identify / screen for frailty.
7-mark note

9 Cognition, Dementia, Delirium, Sleep & Psychogeriatrics

Alzheimer's, biomarkers, delirium, depression, sleep, addiction, behaviour

86. Alzheimer's dementia — pathogenesis, latest advances in pathogenesis at the molecular level, drugs used in dementia (mechanism of action, efficacy, indications for use and side effects) and newer treatment modalities being tried.
15-mark essay

87. Advances in the diagnosis of early stages of Alzheimer's dementia and mild cognitive impairment.
15-mark essay

88. Biomarkers for Alzheimer's dementia / emerging biomarkers in cognition.
ASKED ×3 7-mark note

89. Current drug therapy, newer therapy and newer drugs for Alzheimer's disease.
ASKED ×3 7-mark note

90. Discuss confusional states in the elderly. How will you do the investigations and plan the management?
20-mark essay

91. Interventions to prevent delirium in post-operative older patients.
7-mark note

92. Discuss any three cognitive screening tests useful in the outpatient department.
7-mark note

93. Geriatric depression scales.
7-mark note

94. What are the newer antidepressants? What is the safety profile of these drugs in the older person?
7-mark note

95. Sleep protocol.
7-mark note

96. What are the causes, risk factors, clinical features, complications and treatment of REM (rapid eye movement) sleep behaviour disorder?
7-mark note

97. Impulse control disorders.
7-mark note

98. Nicotine dependence.
7-mark note

99. What are the effects and ill effects of alcohol in the geriatric population? How would you address the issue of alcoholism in an elderly patient? Write a note on alcohol de-addiction.
ASKED ×2 7-mark note

10 Neurology

Motor neuron disease, coma, stroke, parkinsonism, tremor, neuropathy

100. What is motor neuron disease? Discuss the etiopathogenesis, clinical presentation, symptoms, course, prognosis and the treatment modalities available.

ASKED ×2 | 15-mark essay + 6-mark note

101. Discuss the causes of coma in elderly people. (Asked together with: how will you investigate and manage diabetic ketoacidosis?)

20-mark essay

102. Dizziness in the elderly.

6-mark note

103. Thrombolysis in stroke.

7-mark note

104. Discuss the recent advances in the management of parkinsonism.

7-mark note

105. Surgical management of parkinsonism.

7-mark note

106. Management of motor fluctuations in Parkinson's disease.

7-mark note

107. Parkinsonism-plus syndrome.

6-mark note

108. Pathognomonic MRI findings in progressive supranuclear palsy and multiple system atrophy.

7-mark note

109. Differential diagnosis of tremor in the older person. Discuss the treatment modalities for each of these conditions.

7-mark note

110. What are the risk factors and criteria to diagnose Restless Leg Syndrome? How will you manage a case of restless leg syndrome?

7-mark note

111. Causes, evaluation and management of a person presenting with features of peripheral neuropathy.

7-mark note

112. Myasthenia gravis — presentations, laboratory evaluation and treatment.

7-mark note

113. Ring-enhancing shadow on CT brain.

6-mark note

11 Cardiovascular Medicine

ACS, stents, heart failure, arrhythmia, anticoagulation, risk reduction, lipids

114. What are the types of acute coronary syndromes? How are they diagnosed, investigated and treated based on the latest recommendations published in literature — both acutely and on outpatient follow-up?

15-mark essay

115. Discuss the management of ST elevation myocardial infarction. What are the recent advances in the approach to this entity?

15-mark essay

116. Newer cardiac biomarkers in myocardial injury and their interpretation.

7-mark note

117. Drug eluting stents — pros and cons; advantages and disadvantages over bare metal stents.

ASKED ×3 7-mark note

118. Etiopathogenesis and management of in-stent stenosis.

7-mark note

119. Heart failure is commonly encountered on the geriatric wards. Describe the etiopathology, clinical presentation, stages and management of heart failure.

7-mark note

120. Management of systolic heart failure in an elderly patient with chronic kidney disease with a GFR of 40 ml/min.

7-mark note

121. Complete heart block.

6-mark note

122. AHA guidelines for stroke prevention in atrial fibrillation.

7-mark note

123. CHADS2-VASc score — what is it used in and what is its clinical significance?

7-mark note

124. Role of newer anticoagulants.

7-mark note

125. Low molecular weight heparins — types, indications for use, benefits and side effect profile.

7-mark note

126. The orthopaedic team wants you to draw up an evidence based protocol on venous thromboembolic prophylaxis following elective total hip and knee replacements. Draw up this protocol based on evidence in literature.

7-mark note

127. Enumerate the causes for secondary hypertension — how would you evaluate and treat this entity?

7-mark note

128. Lifestyle modification to reduce cardiovascular risk.

7-mark note

129. Primary prevention of cardiovascular disease.

7-mark note

130. Statins in the elderly.
7-mark note

131. Ezetimibe — indications, mode of action, metabolism, excretion, side effects and dosage.
7-mark note

12 Respiratory Medicine
COPD, oxygen therapy, CPAP, airway pathology

132. Chronic obstructive pulmonary disease — management based on recent recommendations.
7-mark note

133. A known COPD patient on inhalers presents to the Emergency Department with severe breathlessness and an oxygen saturation of 60% after three days of cough with purulent sputum. How would you tailor oxygen requirements and oxygen delivery systems throughout his hospital stay and at discharge?
7-mark note

134. Role of CPAP in diseases of the elderly.
7-mark note

135. Describe the changes seen in the airways in emphysema and asthma.
7-mark note

13 Renal, Genitourinary, Continence & Men's Health
RRT, electrolytes, UTI, incontinence, catheters, LUTS, prostate, testosterone

136. Role of renal replacement therapy in the elderly.
7-mark note

137. Peritoneal dialysis.
6-mark note

138. Hyponatremia.
6-mark note

139. Hyperkalemia — how should you investigate hyperkalemia (including in a diabetic) and what is the line of management?
ASKED ×2 7-mark note + 6-mark note

140. Urinary tract infection in the elderly.
6-mark note

141. Management of recurrent urinary tract infection in the elderly. A 65-year-old man has had three confirmed urinary tract infections, all needing hospitalisation and intravenous therapy in the last six months, and solicits your advice for this recurrent problem. What advice and therapies will you give him?
ASKED ×2 7-mark note

142. Asymptomatic bacteriuria — definition, treatment and prognosis.
7-mark note

143. Behavioural intervention in urinary incontinence.
7-mark note

144. Stress urinary incontinence in women. Add a note on Kegel's exercises.
7-mark note

145. Long term catheterization / continuous bladder drainage.
ASKED ×2 7-mark note

146. What is the preliminary assessment done in an older male with lower urinary tract symptoms? When would you refer to a urologist?
7-mark note

147. Testosterone replacement therapy in the elderly male / testosterone therapy in older men.
ASKED ×2 7-mark note

148. Prostate specific antigen — to screen or not to screen: what does the recent evidence show and why?
7-mark note

149. Alpha receptor antagonists.
6-mark note

14 Endocrine & Metabolic

Diabetes emergencies, oral agents, insulins, diabetic foot, thyroid

150. How will you investigate and manage diabetic ketoacidosis? (Asked paired with: causes of coma in elderly people.)
20-mark essay

151. Newer oral antidiabetic agents.
7-mark note

152. Newer insulin analogues — onset and duration of action, and their advantage over the older insulins.
ASKED ×2 7-mark note

153. Diabetic foot syndrome.
7-mark note

154. What are the clinical features of hyperthyroidism in the older person? What are the investigations to be sent and what should be the line of management?
7-mark note

15 Infection, Sepsis & Immunisation

Adult immunisation, sepsis and shock, tuberculosis, hepatitis, malaria

155. Draw up an immunisation schedule for a fit elderly patient and give reasons for your choice of immunisation protocol based on evidence found in literature. Discuss the current guidelines for vaccination of older persons and the status of vaccination in the elderly with recent evidence.
ASKED ×5 7-mark note

156. Pneumococcal vaccination.
7-mark note

157. Travel recommendations for vaccinations in the elderly — CDC guidelines.
7-mark note

158. Define sepsis, septic shock, systemic inflammatory response syndrome and multiorgan dysfunction syndrome. Describe the epidemiology, etiopathogenesis, clinical presentation, diagnosis, investigations, management and prognosis of an older person with sepsis, and the recent advances in the management of sepsis.
ASKED ×3 15-mark essay + 7-mark note

159. How would you diagnose and manage multidrug-resistant tuberculosis based on the DOTS-Plus regime?
7-mark note

160. Malaria — treatment drugs: classes, uses, contraindications, side effect profile and efficacy. Also mention the drugs used in prophylaxis for malaria.
7-mark note

161. Recent trends in the treatment of Hepatitis C infection.
7-mark note

162. Treatment — older and newer modalities — of chronic Hepatitis B infection.
7-mark note

16 Oncology, Haematology & Cancer Screening

Screening strategy, tumour markers, MDS, plasma cell disorders, anaemia

163. Cancer screening in the elderly.
ASKED ×3 7-mark note

164. Screening for colon cancer.
7-mark note

165. Tumour markers — role in detection and management of cancer.
7-mark note

166. Myelodysplastic syndrome — WHO classification and distinguishing features of each class; diagnostic criteria and the differentials one has to consider.
ASKED ×2 7-mark note

167. POEMS syndrome — clinical features, laboratory investigations and treatment.
7-mark note

168. Bone marrow transplant.
7-mark note

169. A 68-year-old man presents to the outpatient department with shortness of breath. On examination he was found to be pale and in failure. Elaborate on how one should investigate and manage him.
7-mark note

17 Gastroenterology & Hepatology

Constipation, transplantation

170. How do you prevent and manage chronic constipation in the elderly? Discuss the complications and management of constipation.
ASKED ×3 7-mark note + 6-mark note

171. Liver transplantation.

6-mark note

18 Perioperative Care, Procedures & Miscellaneous

Pre-operative assessment, periprocedural planning, organ donation

172. Peri-operative care of the elderly / pre-operative assessment.

ASKED ×2 7-mark note + 6-mark note

173. You are tasked with preparing a checklist for older adults undergoing diagnostic and therapeutic procedures in the ward. Discuss the common medical issues that need planning prior to these procedures and how they need to be addressed in the periprocedural period to avoid adverse patient outcomes — e.g. management of antiplatelet agents in the periprocedural period.

7-mark note

174. Eye donation.

6-mark note

Part C · Ebenezer's bank — Paper IV topics not yet asked

Every Paper IV-relevant heading in Ebenezer's Geriatric Question Bank was cross-checked against the 174 questions in Part B. The topics below appear in the bank but have **never** been set in a Paper IV university paper. They are the highest-value gap list you have: a compiler thought them worth including, and the examiner has not yet used them.

Rehabilitation & Service Models

1. Transitional home care — admission and discharge criteria, and the role of management.
2. Advance care planning (as distinct from the advance directive document).
3. Role of physiotherapy in the elderly.
4. Role of the occupational therapist in an older person with mobility issues.
5. Phases of human walking — describe in detail with a diagram.
6. Deconditioning and its prevention.

Falls & Mobility

7. Post-fall syndrome.
8. Fear of falling — evaluation and prevention.
9. Drop attacks.
10. Gait and balance assessment.
11. The long lie — consequences and management.
12. Vestibular rehabilitation.

Continence & Genitourinary

13. Faecal incontinence — evaluation and management.
14. Overactive bladder.
15. Bladder retraining protocol.
16. Uroflowmetry.
17. Erectile dysfunction and the treatments available.
18. Hormone replacement therapy in older women.
19. Gynaecomastia in the older male.
20. Hirsutism and baldness in the elderly woman.

Psychogeriatrics

21. Non-pharmacological management of agitation in the elderly.
22. Suicide in elders and suicide risk assessment.
23. Anxiety and its management in the elderly.
24. Psychotic disorders in late life; late-onset schizophrenia.

25. Atypical antipsychotics — side effects and safety profile in the elderly.
26. Mood disorders in late life.
27. Age-associated personality changes.
28. Diogenes syndrome.
29. Age-associated changes in sexual activity.
30. Sleep hygiene; approach to insomnia and its treatment.
31. Obstructive sleep apnoea — clinical features, associated comorbidities, laboratory tests, management and prognosis.

Nutrition

32. Mini Nutritional Assessment (MNA) — components, interpretation and interventions to improve it.
33. Vitamin deficiencies in the elderly — recognition and correction.
34. Parenteral nutrition in the older adult.
35. Recommended micronutrients in the elderly, their sources and deficiency states.

Prevention, Pharmacotherapy & Safety

36. Polypharmacy and the polypill — prevention and deprescribing.
37. Anticholinergic burden.
38. The prescribing cascade.
39. Drug interactions in the elderly.
40. Pharmacokinetic and pharmacodynamic changes in the elderly and how they affect drug choice.
41. Evaluation of newer drug therapies.
42. Herd immunity.
43. COVID-19 — prevention, management and vaccination in older adults.
44. Infection control practice in hospitals; device-associated infections — prevention and management.
45. Atypical presentation of infections in the elderly.

Chronic Disease & Recent Advances

46. SGLT2 inhibitors in diabetes and heart failure.
47. SIADH — causes, evaluation and management.
48. Solitary thyroid nodule.
49. Subclinical hypothyroidism.
50. FRAX score and the fracture fragility index.
51. Macular degeneration — clinical presentation, aetiology, diagnosis, treatment and prevention.
52. Visual impairment — approach and age-related causes.
53. Tinnitus.
54. Chronic fatigue syndrome — diagnostic criteria, clinical manifestations, predisposing factors, course, prognosis and treatment.
55. Inanition.

56. Constant itching in the elderly.

57. Polymyalgia rheumatica.

58. Sjogren syndrome.

WHY THIS LIST MATTERS

58 topics sit in the Ebenezer bank without ever having been examined in Paper IV. Several of them — polypharmacy and deprescribing, the Mini Nutritional Assessment, fear of falling, faecal incontinence, obstructive sleep apnoea, and SGLT2 inhibitors — are so central to modern geriatric practice that their continued absence is itself a prediction signal.

Part D · Predicted questions

Predictions are built from three signals: how often a topic has been asked, how long it has been since it last appeared, and whether the recent literature has given the examiner a fresh angle. Probability labels are relative, not absolute — treat "very high" as a topic you cannot afford to walk in without, and "moderate" as one worth a prepared skeleton answer.

Paper IV · most likely 15-mark essays

Predicted essay	Odds	Reasoning
Comprehensive Geriatric Assessment — components, domains, tools, evidence base and application in Indian practice.	Very high	Twice as an essay, but not since 2019. The single most reliable Paper IV essay stem and overdue by six sessions.
NPHCE — objectives, service package at each level of care, and implementation; plus NPOP and current welfare schemes.	Very high	The most-asked Paper IV topic overall (5 appearances). Last set as a 7-mark note in 2024, so an essay slot is due.
Elder abuse — forms, epidemiology in India, risk factors, screening tools, legal framework (Maintenance and Welfare of Parents and Senior Citizens Act 2007, Elderline 14567) and management.	High	Five appearances, three of them essays; absent from both 2024 and 2025 papers.
Rehabilitation in the elderly — definition, evaluation, components, the interdisciplinary team and outcome measurement.	High	Rehabilitation is the single densest theme in Paper IV. Essay versions appeared in 2015, 2020 and 2021 but not since.
Frailty and sarcopenia — overlapping constructs: etiopathogenesis, Fried phenotype vs frailty index, EWGSOP2/AWGS criteria, assessment and management.	High	Frailty has four appearances and sarcopenia one; the examiner has begun pairing constructs in a single essay.
Palliative and end-of-life care — principles, symptom control in advanced malignancy, communication, advance directives and the Indian legal position after Common Cause (2018).	High	Essay in 2014, 2017, 2022 and 2024 — on a roughly three-year cycle.
Malnutrition in older adults — screening, MNA, diagnosis by GLIM criteria, and management including oral nutritional supplementation.	Moderate	Essay in 2009 and 2023; nutritional assessment recurs as a note.
Falls in older adults — multifactorial risk assessment, evidence-based intervention bundles and post-fall management.	Moderate	Essay in 2019 and 2024, so slightly less likely immediately, but never absent for more than three sessions.

Paper IV · most likely 7-mark notes

Predicted note	Odds	Reasoning
Vaccination and immunisation schedule in older adults, with the evidence base (pneumococcal, influenza, herpes zoster, COVID-19, Td).	Very high	Five appearances — the most frequent short-note topic in Paper IV.
Advance directives and the living will — current status and procedure in India.	Very high	Four appearances; a near-fixture of the note section.
Newer drugs for Alzheimer's disease — anti-amyloid monoclonal antibodies, indications, evidence and limitations.	Very high	Alzheimer's therapy has three note appearances; the anti-amyloid era makes a fresh 'recent advances' framing very likely.
Caregiver burden and caregiver stress — assessment tools and management.	High	Three appearances; last asked in 2019.
Home based care and community based long term care services.	High	Home based care has three appearances; the service-model family of questions is asked almost every year.
Cancer screening in older adults — principles, life expectancy thresholds and deprescribing screening.	High	Three appearances plus a colon-cancer-specific note in 2023.
Constipation in the elderly — prevention, pharmacological management and complications.	High	Three appearances across 2009, 2013 and 2019.
Breaking bad news — a structured protocol (SPIKES) applied to a geriatric scenario.	High	Three appearances; last asked in 2021.
Polypharmacy, deprescribing and the prescribing cascade — Beers, STOPP/START and anticholinergic burden.	High	Prominent in the Ebenezer bank but never yet asked in Paper IV. A classic overdue 'preventive geriatrics' note.
Sarcopenia — AWGS 2019 criteria, measurement of muscle mass and resistance exercise prescription.	Moderate	Asked once as an essay in 2012 and never since, despite dominating the recent literature.
SGLT2 inhibitors in the older adult — heart failure, CKD and diabetes indications with geriatric cautions.	Moderate	Newer agents recur in Paper IV ('newer insulins', 'newer anticoagulants', 'newer antidiabetics'); SGLT2 is the obvious next member of that family.
Restless legs syndrome and REM sleep behaviour disorder — diagnosis and management.	Moderate	RLS last appeared in 2013; RBD appeared for the first time in 2025, signalling examiner interest in sleep.
Fear of falling, post-fall syndrome and the long lie.	Moderate	Never asked directly, but falls appear in almost every paper and this is the untouched facet.
Faecal incontinence and overactive bladder — evaluation and management.	Moderate	Urinary incontinence recurs; the faecal and OAB facets remain unexamined.
Wearable and sensor technology in fall prevention and remote monitoring of older adults.	Moderate	Asked once in 2012, when the technology barely existed; 'gadgets to improve quality of life' in 2025 shows renewed interest.

A model predicted Paper IV

If you want a single paper to mock-write against, this is the twelve-question set the frequency and recency analysis produces.

	Question	Marks
I.1	Comprehensive Geriatric Assessment — domains, tools, evidence for benefit, and how you would run it in an Indian district hospital.	15
I.2	Elder abuse — forms, epidemiology, risk factors, screening, the Indian legal framework, and management.	15
II.1	Immunisation schedule for an older adult, with the evidence for each vaccine.	7
II.2	Advance directives and the living will — status and procedure in India.	7
II.3	Newer drugs for Alzheimer's disease, including anti-amyloid antibodies.	7
II.4	Polypharmacy and deprescribing — Beers, STOPP/START, anticholinergic burden.	7
II.5	Caregiver burden — assessment and management.	7
II.6	Sarcopenia — criteria, assessment and management.	7
II.7	Cancer screening in older adults — when to start and when to stop.	7
II.8	Breaking bad news using a structured protocol.	7
II.9	SGLT2 inhibitors in the older adult.	7
II.10	Fear of falling and post-fall syndrome.	7

Cross-paper watchlist — Papers I, II and III

Paper IV does not sit in isolation: the same themes recirculate across papers with a different emphasis. These are the highest-probability areas for the other three papers based on the same frequency and recency analysis.

Paper I — Applied Basic Sciences

1. Anatomy: blood supply and conducting system of the heart; bronchopulmonary segments; dermatomes; innervation of the urinary bladder; portocaval anastomosis.
2. Physiology: ageing changes in the heart; homeostasis and homeostenosis; Frank-Starling law; oxygen–haemoglobin dissociation curve; nephron and sites of electrolyte exchange.
3. Biochemistry: vitamin D; NT-proBNP; purine metabolism and gout; lipid metabolism; bone turnover markers.
4. Pharmacology: pharmacokinetic and pharmacodynamic changes with ageing; oral anticoagulants; drugs in benign prostatic enlargement; MAO-B inhibitors; anticholinergic burden.
5. Pathology: Lewy bodies and neurofibrillary tangles; iron deficiency anaemia smear; fatty liver types; inflammaging.
6. Microbiology: immunosenescence and inflammatory markers in ageing; pneumococcal vaccines; blood cultures in infective endocarditis; ELISA; antibiotic choice in lower UTI.

Paper II — General Medicine including Psycho-Geriatrics

1. Behavioural and psychological symptoms of dementia — types and stepped management.
2. Delirium — assessment tools, prevention bundles and management.
3. Depression in older adults and antidepressant choice; suicide risk assessment in late life.
4. Heart failure with preserved ejection fraction; GDMT and the four-pillar approach.
5. COPD by current GOLD criteria; asthma by GINA; late-onset asthma–COPD overlap.
6. Hyponatraemia and SIADH; hypercalcaemia work-up.
7. Late-onset rheumatoid arthritis versus early-onset disease.
8. Approach to vertigo, syncope and falls presenting to the Emergency Department.

Paper III — Geriatrics

1. The 5 Ms of geriatrics as an organising framework.
2. Pre-operative assessment and perioperative management of hip fracture in a comorbid older adult.
3. Reversible dementias — causes and investigation; Lewy body dementia.
4. Bone mineral density interpretation and the FRAX tool.
5. Screening tools for malnutrition; BMI cut-offs for Asians.
6. Acute kidney injury — differentials and investigation in the older adult.
7. Carotid stenosis — angioplasty versus endarterectomy.
8. Urinary incontinence — types, evaluation and management.

FINAL NOTE ON USING PREDICTIONS

Prediction is a way of ordering revision, not of restricting it. The three topics that have been asked five times each are near-certainties over any two-session horizon, but the paper always carries three or four genuinely novel questions — REM sleep behaviour disorder and Geriatric Nurse Practitioners were both new in the last two sessions. Prepare the high-frequency core to essay depth, keep a one-page skeleton for everything in Part C, and you will have something structured to write for any question on the paper.

MD GERIATRIC MEDICINE

High-Yield Examination Answers

Volume: Biology of Ageing, Health Promotion & Bioethics

Twelve compiled questions | 15-mark answer format

Primary sources: Pathy's Principles and Practice of Geriatric Medicine, 6th edition
Essentials of Clinical Geriatrics, 7th edition
Evidence updates: 2023 – 2026
Indian context: Supreme Court, MoHFW, ICMR, ICOPE
Compiled: August 2026

Textbook content and current evidence are presented separately throughout.

Contents

#	Question	Format
SECTION A — BIOLOGY OF AGEING		
1	Theories of ageing	15 marks
2	Biomarkers of ageing	15 marks
3	Telomeres and ageing	15 marks
4	Sirtuins in ageing and disease	15 marks
5	Interleukin-6 in ageing and disease	15 marks
6	Immunosenescence and its relevance to vaccination	15 marks
SECTION B — INTERVENTIONS & HEALTH PROMOTION		
7	Caloric / dietary restriction in ageing	15 marks
8	Anti-ageing interventions	15 marks
9	Interventions for healthy ageing	15 marks
10	Active ageing and how to achieve it	15 marks
11	Exercise in the elderly — beneficial or harmful?	15 marks
SECTION C — BIOETHICS & MEDICO-LEGAL		
12	Advance directives — current status in India	15 marks
APPENDIX		
—	Rapid Revision Grid — key numbers, trials, mnemonics	Revision

Q1. Theories of Ageing

> *Primary source: Pathy's Principles and Practice of Geriatric Medicine, 6th ed. — Ch. 1 "A Biological Perspective of Ageing" and Ch. 2 "Physiology of Ageing".*

High-Yield Points (Textbook Content)

Definition & Concept

1. **Ageing is a failure to maintain homeostasis under physiological stress.** It is a progressive process associated with dysfunction.
2. **No single theory explains ageing.** Each older person is unique, and the elderly population is **heterogeneous** — a 65-year-old may be immobile while a 90-year-old is fully independent.
3. Evolutionary theories explain **WHY** we age; biological/molecular theories explain **HOW** we age. Only the latter is directly relevant to clinical geriatrics.

Population Perspective

4. The **Gompertz curve** shows the incidence of death rises **exponentially** from reproductive maturity to maximum lifespan. Ageing is therefore the **primary intrinsic factor of death**.
5. The **naked mole-rat is "non-Gompertzian"** — its death rate does not rise with age, proving the curve reflects intrinsic ageing rather than external hazards alone.
6. After age 80 the death rate rises more slowly — the **"late-life mortality plateau."** Medical intervention produces **rectangularization (compression) of mortality** (Fries).

A. Error / Stochastic Theories (damage accumulates → cells harmed → organ function lost)

7. **Wear and tear theory** — the body simply wears out with repeated use.
8. **Free oxygen radical theory (Harman, 1956)** — **reactive oxygen species (ROS)** damage macromolecules. Pathy's notes this theory has been **reshaped**, because increased ROS may *prolong* lifespan in model organisms, and mitochondrial dysfunction can accelerate ageing *without* ROS.
9. **Mitochondrial DNA theory** — mtDNA is damaged with age; reduced biogenesis and poor autophagic quality control.
0. **Somatic DNA damage theory** — genomic instability from imbalance between DNA damage and repair. **Werner syndrome** (DNA helicase mutation) is the classical **progeroid** example.
1. **Cross-linking theory** and **caloric restriction theory** are also grouped under error theories.

B. Programmed Theories (ageing is genetically driven)

2. **Neuroendocrine theory** — the **hypothalamic-pituitary-adrenal (HPA) axis** is the most important regulator of ageing.
3. **Immunological theory** — **thymic atrophy** reduces mature T-lymphocyte production; humoral immunity falls, antibody production drops, and **infection risk rises**.
4. **Cellular senescence / Hayflick limit** — human fibroblasts stop dividing after **~50 divisions**. **Telomeres shorten** with each division until division stops. This protects against uncontrolled growth **at the expense of ageing**.
5. **Cell death theory** — **apoptosis** (programmed cell death) is responsible for tissue remodelling and homeostasis.

C. Evolutionary Theories

6. **Medawar's mutation accumulation theory** — natural selection cannot remove deleterious genes acting only in late life (the **"selection shadow"**), so harmful effects accumulate towards the end of life.
7. **Williams' antagonistic pleiotropy theory** — a gene with a **small early benefit but a large late harm** is still selected for, because early fitness matters more.
8. **Kirkwood's disposable soma theory** — limited energy must be divided between **reproduction/function** and **repair/maintenance**. The soma is maintained only until the germline is passed on.

D. Hallmarks of Ageing (Lopez-Otin 2013, as quoted in Pathy's)

9. **Nine hallmarks in three groups:**
   - **Primary (causes of damage):** genomic instability, telomere attrition, epigenetic alterations, loss of proteostasis
   - **Antagonistic (responses to damage):** deregulated nutrient sensing, mitochondrial dysfunction, cellular senescence
   - **Integrative (culprits of the phenotype):** stem cell exhaustion, altered intercellular communication
0. Pathy's Table 1.1 lists **eight components of biological ageing**: genomic instability, epigenetic changes, mitochondrial dysfunction, loss of proteostasis, metabolic dysfunction, cell senescence, stem cell exhaustion, and **immunosenescence**.

Clinical Application

1. Key mechanisms: **SASP** (senescence-associated secretory phenotype), **inflammageing**, **insulin/IGF-1 and mTOR downregulation**, and **epigenetic (DNA methylation) clocks** which predict age with error <5 years.
2. **Telomere length is NOT currently accepted as a biological age marker** despite its association with mortality — longitudinal changes are erratic.
3. **Geroscience hypothesis** — multimorbidity and functional loss in old age are driven by **common biological mechanisms**. **Frailty** and **intrinsic capacity** serve as clinical metrics of biological ageing.
4. **Therapeutic opportunities** listed in Pathy's: **senolytics, inflammasome inhibitors, mesenchymal stem cells, calorie-restriction mimetics (rapamycin, metformin), autophagy inducers**, plus **exercise and diet**.

Current Practice Update

Aspect	Pathy's 6th ed. (2022)	Current Evidence (2023–2026)
Hallmarks of ageing	**9 hallmarks** (Lopez-Otin 2013)	**12 hallmarks** (Lopez-Otin 2023) — three added
New hallmarks	Not mentioned	**Disabled macroautophagy, chronic inflammation, dysbiosis**
Explanatory paradigm	Damage/maintenance model dominant	**Hyperfunction and programmatic theories** now serious rivals
Biological age markers	Epigenetic (methylation) clocks only	**Organ-specific proteomic and MRI-based clocks**
Pace-of-ageing measures	Not covered	**DunedinPACE, DunedinPACNI** (third-generation clocks)
Senolytic drugs	Listed as "promising research"	**Phase 1 human trials completed; Phase 2 underway**
Metformin as geroprotector	Listed as potential anti-ageing drug	**21-year DPP follow-up shows no multimorbidity benefit**

Latest Updates (2023–Present)

1. Twelve Hallmarks of Ageing. Lopez-Otin and colleagues expanded the framework to twelve hallmarks: the original nine plus **disabled macroautophagy, chronic inflammation and dysbiosis**. These are interconnected and link to the proposed "hallmarks of health" (spatial compartmentalisation, maintenance of homeostasis, adequate stress responses). A process qualifies as a hallmark only if it **appears with age, accelerates ageing when accentuated, and slows ageing when therapeutically targeted**. *(Cell 2023)*

2. Challenge to the damage paradigm — hyperfunction theory. Blagosklonny's hyperfunction theory reframes ageing as the late-life continuation of growth-promoting pathways that become pathological once selective pressure declines. De Magalhaes' developmental theory describes ageing as a mismatch between developmental programmes optimised for early life and their persistence into later life. Continued mTOR signalling after growth is complete is itself considered hyperfunctional. These are **"quasi-programs"** — run-on of developmental programmes. *(Ageing Res Rev 2022; Aging 2022)*

3. Organ-specific and multi-omic ageing clocks. Plasma proteomic signatures of organ ageing track health and disease; brain and immune system ageing predict healthspan and longevity; organ-specific signatures predict 20-year risk of age-related disease in Whitehall II. Brain-imaging measures such as DunedinPACNI estimate pace of ageing from a single MRI. **First-generation clocks (Horvath, Hannum) predict chronological age; second-generation (PhenoAge, GrimAge) predict mortality; third-generation (DunedinPACE) measure rate of ageing.** *(Nature 2023; Lancet Digit Health 2025; Nat Aging 2025)*

4. Senolytics move into human trials. Two open-label Phase 1 trials — SToMP-AD and STAMINA — tested 12 weeks of intermittent oral **dasatinib plus quercetin** in older adults with or at risk of Alzheimer's disease. In STAMINA, reductions in plasma TNF-alpha correlated with improvement in MoCA ($r = -0.65$, $p = 0.02$). Safety, feasibility, CNS penetrance and biological activity were confirmed; a **Phase 2 RCT is now underway**. *(eBioMedicine 2025)*

5. Metformin as a geroprotector — a setback. In a 21-year follow-up of the Diabetes Prevention Program, metformin did not significantly reduce multimorbidity risk; 85% of participants developed two or more chronic conditions (82% lifestyle, 85% metformin, 87% placebo). Intensive lifestyle intervention was associated with lower long-term multimorbidity burden. **Lifestyle currently outperforms any pharmacological geroprotector.** TAME remains the pending definitive trial. *(JAMA 2026)*

Examiner's Tip — Minimum Skeleton

Definition → Gompertz curve → Error (stochastic) vs Programmed classification → Evolutionary theories (Medawar / Williams / Kirkwood) → Hallmarks of ageing (9 in Pathy's, 12 in 2023) → Geroscience hypothesis and therapeutic implications.

Q2. Biomarkers of Ageing

Primary source: Pathy's 6th ed. — Ch. 1, Ch. 2, Ch. 7 "Physical Fitness and Exercise".

High-Yield Points (Textbook Content)

Concept & Rationale

1. A **biomarker of ageing** is a measurable parameter reflecting **biological age** rather than **chronological age**, which predicts adverse outcomes in late life.
2. The rationale is the **geroscience hypothesis** — accumulation of diseases and loss of function is driven by **common biological mechanisms**. Measuring these allows prediction of outcomes and testing of interventions that extend **healthspan**.
3. **Chronological age is only a surrogate marker** — it stands in for the specific impairments that actually increase vulnerability.
4. **The core problem: there is no agreed definition of what a biomarker of ageing should measure or predict.** This is the biggest obstacle in the field.
5. **Death alone is an insufficient outcome**, because death is often preceded by years of multimorbidity and disability.
6. Three outcomes currently anchor biomarkers: **age-related multimorbidity**, **frailty**, and **intrinsic capacity**.

A. Molecular & Cellular Biomarkers (Pathy's Table 1.1)

7. **Epigenetic clocks (DNA methylation age)** — methylation status of **CpG islets** predicts donor age with average error **<5 years** (Hannum, Horvath).
8. The gap between **methylation age and chronological age** is associated with **cognitive and physical function, frailty and age-related diseases**, and is an **independent predictor of all-cause mortality**. These are the **best-validated molecular markers**.
9. **Telomere length is NOT currently accepted as a biological age marker.** A meta-analysis of 25 studies showed telomere attrition predicts all-cause mortality, but longitudinal changes are **erratic**, probably due to **measurement error**.
0. **Genomic instability** — somatic mutations, translocations, aneuploidy. Robust evidence linking mutation burden to human ageing phenotypes is still lacking.
1. **Cellular senescence — p16INK4a** is the key marker. It correlates with age in human skin, and **p16INK4a-positive cell burden in thigh adipose tissue correlates with muscle strength and walking performance**.
2. In a meta-analysis of **372 GWAS**, the **ink4/ark locus was linked to the greatest number of age-associated diseases** — Alzheimer's, cardiovascular disease, cancer and type 2 diabetes.
3. **SASP** — matrix metalloproteases and pro-inflammatory cytokines released by senescent cells; a measurable output of senescence.
4. **Mitochondrial function** can now be measured **in vivo** and is associated with **muscle strength and mobility**. Powerful but **needs standardisation**.
5. **Proteostasis and autophagy** — autophagy is better maintained in offspring of exceptionally long-lived individuals.
6. **Metabolic markers** — insulin resistance, **increased visceral fat with reduced skeletal muscle mass**, and decline in **sex steroids, GH and IGF-1**. Reduced insulin/IGF-1/mTOR signalling is associated with longevity.
7. **Inflammageing and immunosenescence** — **raised IL-6, raised CRP, reduced IL-2 and IL-2 responsiveness, thymic atrophy, fewer naive T cells with inflation of memory T cells**, and shrinkage of the antigenic repertoire (Table 2.3). Plasma IL-6 rises with normal ageing and **correlates inversely with functional ability**.
8. **Stem cell exhaustion** is conceptually important but **difficult to measure before its clinical consequences appear**.

B. Clinical & Functional Biomarkers

9. **Physical function is the proposed clinical biomarker of healthy ageing** — aerobic capacity, **gait speed, balance, mobility, muscle strength**. It predicts adverse events, disability and mortality.
0. **Gait speed predicts mortality in older adults better than chronic diseases such as hypertension**, and takes no longer to measure than a routine vital sign.
1. **Grip strength** — EWGSOP2 cut-offs for low strength are **<27 kg in men and <16 kg in women**; low appendicular muscle mass is **<20 kg (men) and <15 kg (women)**.
2. **Short Physical Performance Battery (SPPB)** — 4-metre walk + repeated chair stands + balance; predicts **future disability, hospitalisation and mortality**.
3. **Frailty is the clinical metric of biological ageing.** Frail patients show higher **factor VIII, D-dimer, CRP,**

leukocytes, fibrinogen and glucose, with **low vitamin D and low haemoglobin**.

4. **Intrinsic capacity (WHO)** — the composite of all physical and mental capacities (cognition, psychological, sensory, locomotion, vitality) — is the key determinant of functional ability.

Current Practice Update

Aspect	Pathy's 6th ed. (2022)	Current Evidence (2023–2026)
Validation standards	No agreed framework; the main obstacle	**Biomarkers of Aging Consortium validation framework**
Type of clock	DNA methylation clocks only	**Proteomic, metabolomic, imaging and organ-specific clocks**
Scope of measurement	Single whole-body "biological age"	**Organ-by-organ ageing** — brain, heart, kidney, immune
What is measured	Age *gap* (biological minus chronological)	**Pace of ageing** — DunedinPACE, DunedinPACNI
Blood biomarker for frailty	IL-6, CRP, D-dimer, fibrinogen	**GDF-15** emerging as the strongest single candidate
Regulatory role	Research tools only	**Surrogate endpoints for gerotherapeutic trials**

Latest Updates (2023–Present)

1. A formal validation framework now exists. The Biomarkers of Aging Consortium published recommendations on how biomarkers of ageing must be validated before clinical translation, noting that systematic validation is what will accelerate their use in gerotherapeutic trials. The stated problem was that although omics-based biomarkers could serve as surrogate endpoints, no consensus existed on validation. A joint NIA–Consortium symposium in 2024 emphasised understanding the longitudinal, complex and heterogeneous nature of ageing. *(Nature Medicine 2024)*

2. Organ-specific ageing clocks. Ageing is no longer one number for the whole body. Plasma proteomic signatures of organ ageing track health and disease; brain and immune ageing predict healthspan and longevity; organ-specific signatures predict 20-year risk of age-related diseases. **Clinical meaning: a patient may have a "young heart" but an "old brain", allowing organ-targeted prevention.** *(Nature 2023; Nat Med 2025; Lancet Digit Health 2025)*

3. Third-generation clocks measure pace, not level. DunedinPACE measures the pace of ageing and is sensitive to short-term change and intervention; DunedinPACNI estimates pace from a single brain MRI. In the Framingham Offspring cohort, faster DunedinPACE was associated with worse global cognition and steeper cognitive decline after adjusting for education. *(Trends Mol Med 2025; Nat Aging 2025)*

4. GDF-15 — the most promising single blood biomarker in geriatrics. In the longitudinal FRASNET study, higher baseline GDF-15 prospectively identified individuals at high risk of developing **frailty (AUC 0.85–0.96), malnutrition (AUC 0.94) and falls (AUC 0.98)** over six years. In LIFE, GDF-15 showed one of the strongest associations with slower walking speed. BIOFRAIL evaluated plasma GDF-15 in 429 mobility-limited older adults (mean age 79.6) against CFS ≥5 and EWGSOP2 sarcopenia. **Mechanism: GDF-15 reflects mitochondrial dysfunction and inflammageing via AMPK signalling.** *(GeroScience 2025; Ageing Res Rev 2026)*

5. Organ-specific consensus frameworks. The Aging Biomarker Consortium now defines biomarker panels organ by organ — **cardiac ageing, vascular ageing, visual system ageing and adipose tissue ageing** — each providing a multi-dimensional framework covering functional, morphological and molecular biomarkers. *(Life Medicine 2023–2025)*

Examiner's Tip

Define biological vs chronological age → geroscience hypothesis → the validation problem → classify into **MOLECULAR** (epigenetic clocks, p16INK4a, SASP, mitochondrial, IL-6/inflammageing) and **CLINICAL/FUNCTIONAL** (gait speed, grip strength, SPPB, frailty, intrinsic capacity) → state that gait speed and epigenetic clocks are best validated → finish with organ-specific clocks and GDF-15.

Ideal biomarker criteria: must **change with age, predict outcomes better than chronological age, be measurable repeatedly without harm, and respond to intervention.**

Q3. Telomeres and Ageing

Primary source: Pathy's 6th ed. — Ch. 1, Ch. 2, Ch. 42 (respiratory ageing); supplemented by Essentials of Clinical Geriatrics.

High-Yield Points (Textbook Content)

Structure & Basic Biology

1. **Telomeres are repetitive DNA sequences capping the ends of chromosomes**, protecting coding DNA from degradation and end-to-end fusion.
2. With **each cell division the terminal end of the chromosome is not fully replicated**, so the telomere progressively **shortens** (the end-replication problem).
3. Telomere shortening is a **specific type of genomic damage**, and **telomere attrition is one of the primary hallmarks of ageing**.
4. **Telomerase** adds DNA bases back onto telomeres. Transfecting its catalytic component into senescent cells **lengthens telomeres, extends replicative lifespan, and restores a "young" pattern of gene expression**.

Hayflick Limit & Replicative Senescence

5. **Hayflick limit** — human fibroblasts in culture stop dividing after about **50 doublings**. The classic example of **programmed ageing**.
6. When telomeres are exhausted the cell can no longer proliferate — this is **replicative senescence**.
7. **Replicative lifespan in vitro correlates inversely with donor age**.
8. **Key exam concept:** telomere shortening **protects the cell against uncontrolled division and abnormal growth — at the expense of ageing.** It is fundamentally an **anti-cancer (tumour-suppressor) mechanism**.

Which Tissues Are Affected

9. Cells fall into three groups by replicative capacity:
    - **Continuously replicating** — epidermal, gastrointestinal, haematopoietic (telomere biology matters most here)
    - **Replicating on challenge** — liver
    - **Non-replicating** — neurons, cardiac and skeletal muscle
0. In non-replicating tissues, **telomere shortening is not the mechanism** — cell loss causes a permanent deficit instead (e.g. dopaminergic neuron loss affecting gait and balance).
1. **Lung ageing:** decreased **telomerase activity** plus replicative senescence plus rising pro-inflammatory cytokines produce **low-grade chronic pulmonary inflammation — "lung inflammageing"** (Table 42.1).

Downstream Consequences

2. Telomere-triggered senescence produces the **SASP** — matrix metalloproteases and pro-inflammatory cytokines that spread senescence and drive **inflammageing**.
3. Senescence is **not only telomeric** — it is also triggered by **non-telomeric DNA damage** and **excessive mitogenic signalling** via **p16INK4a**.
4. **Stem cell exhaustion** is an integrative consequence of telomere shortening together with DNA damage, epigenetic alterations, senescence and mitochondrial dysfunction.

Evidence in Humans

5. **Telomere shortening occurs during normal ageing in mice and humans**, and experimentally altering telomere loss changes lifespan in mice.
6. A **meta-analysis of 25 studies showed telomere attrition predicts all-cause mortality**.
7. **Critical exam point — telomere length is NOT currently accepted as a biomarker of biological age.** Longitudinal studies show **erratic changes over time**, probably due to **measurement error**.
8. In the **oldest old, no relationship was found between telomere length and mortality**. However, **healthy centenarians had significantly longer telomeres than unhealthy centenarians** — telomere length reflects **health and function rather than exceptional longevity**.

Clinical & Therapeutic Relevance

9. **Telomerase inhibitors are potent potential anticancer agents**, because most cancers reactivate telomerase to achieve immortality.
0. **Werner syndrome** (DNA helicase mutation) is the classical **progeroid** illustration of genomic instability; the **telomere syndromes** are a separate recognised group of premature-ageing disorders.
1. **How far telomere shortening explains in-vivo human ageing remains unknown** — it explains finite cell life in

culture better than whole-organism ageing.

Current Practice Update

Aspect	Pathy's 6th ed. (2022)	Current Evidence (2023–2026)
Telomere length as biological age marker	Rejected — erratic longitudinal change	Position unchanged; **epigenetic clocks have replaced it**
Direction of cancer risk	Short telomeres → genomic instability → cancer	**Mendelian randomisation shows LONGER telomeres causally increase cancer risk**
Clinical entity	Telomere syndromes in passing	**Telomere Biology Disorders (TBD)** now a defined adult diagnosis
Measurement	Not specified	**Flow-FISH and qPCR**; age-adjusted TL **≤1st percentile** is diagnostic
Treatment	None described	**Danazol 800 mg/day** — stabilises telomere attrition
Experimental therapy	Telomerase transfection (lab only)	**ZSCAN4-based ex vivo telomere elongation** of haematopoietic stem cells

Latest Updates (2023–Present)

1. Telomere attrition retained as a core hallmark. Telomere attrition remains one of the **twelve hallmarks of ageing** in the updated framework. *(Cell 2023)*

2. The telomere–cancer paradox confirmed by Mendelian randomisation. In a two-sample MR study using East Asian cohorts, longer telomere length per 1-SD increase due to germline variants was associated with higher risk of site-specific cancer, strongest for **thyroid cancer (OR 2.49)**. Separate MR analysis found significant associations with **primary lymphoid malignancy (OR 1.52), Hodgkin lymphoma (OR 1.64), non-Hodgkin lymphoma (OR 1.70) and B-cell lymphoma (OR 1.57)**. MR in breast disease found longer telomeres increase risk of breast cancer, benign neoplasms and breast inflammation, with no reverse causation. **Take-home: short telomeres cause degenerative disease; long telomeres permit malignancy. Telomere length is a double-edged sword.** *(Cancer Epidemiol Biomarkers Prev 2025; Aging 2024)*

3. Telomere Biology Disorders — a real adult diagnosis. TBDs are inherited bone marrow failure syndromes caused by pathogenic germline variants in telomere-related genes, with a heterogeneous phenotype including **bone marrow failure, pulmonary and liver disease, and predisposition to haematologic and solid malignancies**. **Genes to remember: TERT, TERC, DKC1, RTEL1.** Trial inclusion requires age-adjusted telomere length ≤1st percentile and/or identified mutations plus one cytopenia and/or pulmonary fibrosis.

4. Danazol — first drug shown to preserve telomeres in humans. Long-term results of a prospective phase 1/2 study showed danazol **improved haematologic parameters and stabilised telomere attrition** in TBD. Patients received **danazol 800 mg daily for two years**, with telomere attrition as the primary biologic endpoint and flow-FISH for serial measurement. Androgens are thought to work by **enhancing telomerase activity and reducing the rate of telomere attrition**. *(Blood Advances 2026)*

5. Ex vivo telomere elongation of stem cells. Clinical use of **ZSCAN4** for telomere elongation in haematopoietic stem cells has been reported, forming the basis of the EXG34217 trial in TBD with bone marrow failure — the first attempt at directly lengthening human telomeres therapeutically. *(NEJM Evidence 2025)*

Examiner's Tip

Structure → end-replication problem → Hayflick limit → replicative senescence → SASP and inflammageing → anti-cancer role → telomerase and its inhibitors → why telomere length FAILS as a biomarker → telomere biology disorders and danazol.

Two lines that earn marks:
• "Telomere shortening protects against uncontrolled cell division **at the expense of ageing**."
• "Telomere length is **not currently accepted as a marker of biological age** because longitudinal change is erratic and measurement-error prone."

Q4. Sirtuins in Ageing and Disease

Primary source: Pathy's 6th ed. — Ch. 1; supplemented by Essentials of Clinical Geriatrics (Ch. 1, Theories of Aging).

High-Yield Points

Definition & Basic Biology

1. **Sirtuins are a family of NAD+ dependent enzymes** with **histone deacetylase** activity — the mammalian homologues of the yeast **Sir2 (Silent Information Regulator 2)** gene.
2. Because they **require NAD+**, sirtuins act as **cellular energy sensors** — activated when the NAD+/NADH ratio is high, i.e. in **caloric restriction, fasting and exercise**.
3. **Seven human isoforms (SIRT1–SIRT7)**: **SIRT1, SIRT6 and SIRT7 are mainly nuclear; SIRT2 is cytoplasmic; SIRT3, SIRT4 and SIRT5 are mitochondrial.**
4. **SIRT1 and SIRT6 are the two most relevant to ageing.** Sirtuins regulate genomic stability, lifespan, energy metabolism and tumorigenesis.

Mechanism — Why They Matter in Ageing

5. **NAD+ levels fall progressively with age**, reducing sirtuin activity — the central link between metabolism and ageing.
6. **Sirtuins are the molecular mediators of caloric restriction.** Dietary restriction extends lifespan by exerting **multiple effects on chromatin through sirtuins, TOR and other factors**.
7. This makes ageing partly an **epigenetic** phenomenon — sirtuins act by **deacetylating histones and non-histone proteins**, altering gene expression **without changing the DNA sequence**.
8. **SIRT1 targets** include **p53, FOXO transcription factors, NF-kappaB and PGC-1alpha** — linking sirtuins to apoptosis, stress resistance, inflammation and mitochondrial biogenesis.
9. **SIRT6 maintains chromatin structure at telomeres** by deacetylating **histone H3 lysine 9**, preventing telomeric DNA damage and cellular senescence. **SIRT6-deficient mice show genomic instability and premature ageing**, and SIRT6 is upregulated during caloric restriction.
0. **SIRT1 and SIRT6 act synergistically** — SIRT1 deacetylates SIRT6 at residue K33, and deacetylated SIRT6 then remodels chromatin.
1. **Anti-inflammatory action:** SIRT1, SIRT2 and SIRT6 **inhibit NF-kappaB signalling**, reducing the cytokine output that drives **inflammageing**.
2. **Mitochondrial sirtuins (SIRT3–5)** regulate oxidative metabolism and reduce ROS — relevant to **sarcopenia and frailty**.

Evidence — The Rise and Fall of the Sirtuin Hypothesis (key discrimination point)

3. **Initial excitement:** activation of a sirtuin was found to **extend lifespan in yeast**, generating intense interest in sirtuins as "longevity genes".
4. **Subsequent reversal:** the sirtuin story then **unravelled — those experiments were not replicated by other laboratories**. *(Science 2011)*
5. **Current textbook position:** in mice, **modulation of sirtuins does NOT extend lifespan, but it does expand healthspan** — less diabetes mellitus, healthier metabolic profiles and healthier livers.
6. **Resveratrol**, a molecule from red wine acting through a sirtuin, extended lifespan in **worms and flies**, but **these findings were also not confirmed elsewhere**. It may extend **healthspan rather than lifespan**.

Position Within the Wider Biology of Ageing

7. Sirtuins sit within **deregulated nutrient sensing**, one of the **antagonistic hallmarks of ageing**. The other arms are the **insulin/IGF-1 axis, mTOR and AMPK**.
8. **Reduced insulin/IGF-1 and mTOR signalling is associated with longevity**. Intense trophic and anabolic activity accelerates ageing; downregulation is a **defensive response**.
9. **Rapamycin (mTOR inhibitor)** extended lifespan in genetically heterogeneous mice **even when started in late life**; the practical barrier to human use is **immunosuppression**.
0. **Metformin** is a potential anti-ageing drug because of its effects on **deregulated nutrient sensing, mitochondrial dysfunction, DNA damage and inflammation**. It has since been shown to be a **direct SIRT1-activating compound**.
1. **Clinical bottom line:** caloric restriction is not practical in humans, so research has shifted to **caloric-restriction mimetics** — sirtuin activators, rapalogs, metformin and NAD+ precursors.
2. **Cautionary note:** resetting the epigenome carries a **risk of malignancy** in cells that have already accumulated mutations and then acquire increased proliferative potential.

Current Practice Update

Aspect	Textbook Position (2012–2022)	Current Evidence (2023–2026)
Isoform focus	"A sirtuin" (SIRT1 implied)	**SIRT1 and SIRT6** are the recognised ageing sirtuins
Lifespan vs healthspan	Lifespan claims discredited; healthspan retained	**Position unchanged and confirmed**
SIRT7	Not addressed	**SIRT7 knockout extends lifespan** — opposite direction
Resveratrol	Unconfirmed lifespan effect	Largely abandoned; **direct SIRT6 activators** developed
Pharmacology	Resveratrol only	**MDL-800 and pyrroloquinoxaline SIRT6 activators**
NAD+ boosters	Not covered	**NR and NMN reliably raise NAD+ but clinical outcomes remain negative**
Metformin	Anti-ageing candidate via nutrient sensing	Now shown to be a **direct SIRT1 activator**

Latest Updates (2023–Present)

1. SIRT1 and SIRT6 consolidated as the ageing sirtuins. Sirtuins are framed as NAD+ dependent enzymes regulating genomic stability, lifespan, energy metabolism and tumorigenesis, with SIRT1 and SIRT6 specifically regulating ageing-related signalling at molecular, cellular, tissue and whole-organism levels. **Name SIRT6 if asked for the single most important anti-ageing sirtuin** — it protects telomeric chromatin and its loss causes progeria in mice. *(Ageing Res Rev / Cellular Signalling 2023)*

2. The SIRT7 paradox — not all sirtuins are pro-longevity. While SIRT1 and SIRT6 are predominantly anti-inflammatory, SIRT7 can be **pro-inflammatory** in a tissue-specific manner under physiological stress; recent in vivo evidence shows **SIRT7 knockout mice have a longer lifespan and delayed onset of age-related conditions**. This overturns the simple "sirtuins = longevity genes" model. *(Narrative review 2026)*

3. Direct sirtuin activators have replaced resveratrol. Resveratrol's supposed SIRT1 activation was an assay artefact. Attention has moved to cellularly active SIRT6 allosteric activators: **MDL-800** improves genomic stability and pluripotency of aged murine iPS cells, and pyrrolo[1,2-a]quinoxaline derivatives have been developed as potent, selective SIRT6 activators. *(Exploration of Drug Science 2025)*

4. NAD+ precursors (NR and NMN) — biochemically effective, clinically disappointing. In a 12-week double-blind RCT of **nicotinamide riboside 500 mg twice daily** in 52 older adults with amnestic MCI, blood NAD+ approximately **doubled (23.4 to 48.5 micromol/L)** and left hippocampal perfusion increased significantly — but **cognitive function did not significantly improve**. A systematic review of NMN trials found a significant effect on raising NAD+ yet most clinically relevant metabolic outcomes did not differ from control. A further meta-analysis concluded evidence for NMN and NR effects on skeletal muscle mass and function in older adults **remains inconclusive**. *(Crit Rev Food Sci Nutr 2025; J Cachexia Sarcopenia Muscle 2025)*

5. Practical message. **No sirtuin-targeted agent is licensed for any ageing indication.** The interventions with proven benefit on the same nutrient-sensing pathways remain **exercise, dietary modification and weight management** — and metformin where indicated for diabetes.

Examiner's Tip

Define sirtuins as NAD+ dependent deacetylases (Sir2 homologues) → seven isoforms and compartments → NAD+ decline with age → mediators of caloric restriction → SIRT1 (p53/FOXO/NF-kappaB/PGC-1alpha) and SIRT6 (telomeric chromatin, H3K9) → the yeast lifespan claim and its failure to replicate → healthspan not lifespan → position within deregulated nutrient sensing alongside mTOR, IGF-1, AMPK → current drugs.

The line that earns the mark: "Sirtuin modulation does not extend lifespan in mammals — it expands healthspan."

Q5. Interleukin-6 in Ageing and Disease

Primary source: Pathy's 6th ed. — Ch. 1, Ch. 2, Ch. 22 (anaemia), Ch. 28 & 36 (cardiovascular), Ch. 43 (respiratory infection).

High-Yield Points (Textbook Content)

Basic Biology

1. **IL-6 is a pro-inflammatory cytokine** produced by macrophages, T cells, endothelium, adipocytes and skeletal muscle — a central component of the innate immune cascade along with **IL-1, TNF-alpha, complement proteins and immunoglobulins**.
2. **Increased IL-6 is one of the defining age-related changes of the immune system** (Table 2.3), alongside **decreased IL-2 and IL-2 responsiveness, thymic atrophy, decreased cell-mediated immunity, accumulation of memory T cells, increased autoantibodies, impaired macrophage function**, and increased NK cell number with decreased cytotoxicity.
3. **Plasma IL-6 rises as a function of normal ageing** — not merely a marker of disease. **It is the best-characterised molecular marker of "inflammageing".**
4. **KEY EXAM POINT: plasma IL-6 correlates INVERSELY with functional ability in older people.** The higher the IL-6, the poorer the function.
5. **Higher circulating CRP and cytokine receptor levels are independently associated with physical dysfunction and disability.**

Why IL-6 Rises With Age

6. **Ageing behaves like a chronic stress state** — with **increased cortisol and catecholamines** and **decreased sex hormones and growth hormone**.
7. **Cortisol and catecholamines stimulate IL-6 and TNF-alpha release, whereas sex hormones inhibit IL-6.** The fall in oestrogen and testosterone with age therefore **disinhibits IL-6** — the core mechanism to quote.
8. Other drivers feeding into IL-6: **mitochondrial dysfunction, cellular senescence (SASP), loss of proteostasis, epigenetic alterations**, plus extrinsic factors such as **chronic infection (especially CMV) and altered microbiota**.
9. **Senescent cells secrete IL-6 as part of the SASP**, spreading senescence to neighbouring cells.

The Inflammageing Paradox

0. The ageing innate immune system shows **basal immune stimulation but immune paralysis when a specific response is required** — this paradox *is* inflammageing.
1. Clinically: **baseline alveolar IL-6 and TNF-alpha are higher in aged lungs**, yet **toll-like receptor activation is blunted**, so the acute response to a real pathogen is dampened. High baseline levels indicate a **dysregulated, not a better, immune response**.

Systemic Consequences — Where IL-6 Shows Up Clinically

2. **Anorexia of ageing** — **IL-1, IL-6 and TNF-alpha are satiating cytokines** that reduce food intake and body weight centrally and peripherally. **IL-1 and IL-6 are elevated in older people with cachexia.**
3. **Sarcopenia and frailty** — inflammation drives age-related loss of muscle mass and strength. Frail patients have higher **CRP, fibrinogen, factor VIII, D-dimer, leukocytes and glucose**, with **low vitamin D and low haemoglobin**.
4. The overlap of frailty and cardiovascular biomarkers suggests a **common biological pathway** linking frailty and CVD.
5. **Anaemia of chronic disease** — the classic IL-6 story. **IL-6 mediates the hypoferraemia of inflammation by inducing hepatic synthesis of hepcidin.**
6. **Hepcidin** blocks the **ferroportin** transporter, blocking duodenal iron absorption and iron release from macrophages, so iron is **locked in ferritin stores** — a **functional iron deficiency**.
7. **Laboratory picture of ACD: normocytic anaemia**; haemoglobin low; **serum iron low-to-normal, transferrin low-to-normal, transferrin saturation low, ferritin normal-to-high, soluble transferrin receptor NORMAL** (this last distinguishes it from iron deficiency, where sTfR is high).
8. Pro-inflammatory cytokines also **lower erythropoietin production**, damage EPO-producing renal cells, downregulate EPO receptors, and **directly damage erythroid precursors** by cytokine-mediated apoptosis.
9. **Atherosclerosis and endothelial dysfunction** — **TNF-alpha, IL-1beta and IL-6** released from ageing endothelium activate **NF-kappaB and AP-1**, inducing **COX-2 and iNOS**, generating superoxide and peroxynitrite (Figure 36.1).
0. **Depression in older adults** is associated with **raised IL-6**, along with vitamin D deficiency and white-matter hyperintensities.
1. **COVID-19** — the "cytokine storm" of dysregulated host immune response underlies complications, and **IL-6 is one such**

target. **Tocilizumab and sarilumab** were studied; a systematic review of observational data suggested tocilizumab reduced complication rate and illness duration.

2. **Proof of principle for anti-cytokine therapy: IL-1beta inhibition with canakinumab (CANTOS)** reduced the incidence of age-related diseases in humans.

Current Practice Update

Aspect	Pathy's 6th ed. (2022)	Current Evidence (2023–2026)
IL-6 as target in CVD	Promising; canakinumab proof-of-concept	**ZEUS trial NEUTRAL — no MACE benefit despite target engagement**
IL-6 blockade in PMR	Not addressed	**Sarilumab FDA-approved (2023) — first biologic for PMR**
IL-6 blockade in GCA	Not addressed	**Tocilizumab approved; sarilumab GCA trial terminated early**
IL-6 in COVID-19	Observational data only	Established; in severe COVID guidelines
IL-6 as frailty biomarker	Correlates inversely with function	Used in **multi-marker panels with GDF-15, FGF-21, sRAGE, NfL**
Key conceptual lesson	Inflammation is a modifiable target	**Biomarker–outcome dissociation: lowering IL-6 does not equal better outcomes**

Latest Updates (2023–Present)

1. ZEUS trial — IL-6 inhibition fails in cardiovascular disease (July 2026). ZEUS was a multinational double-blind placebo-controlled trial of **6,376 participants with ASCVD, CKD and hsCRP ≥2 mg/L**, randomised to **ziltivekimab 15 mg subcutaneously monthly** or placebo. Mean age 69.5 years, 65.7% diabetes, 41.3% heart failure, mean eGFR 44.5 mL/min/1.73 m^2, median hsCRP 4.5 mg/L, median IL-6 4.9 pg/mL. Although ziltivekimab demonstrated **target engagement with expected reductions in free IL-6 and hsCRP**, this did **not translate into any MACE reduction (HR 0.99; 95% CI 0.88–1.11)**. Overall adverse event rates were similar, but a higher proportion of ziltivekimab-treated participants had a **serious infection**. HERMES (heart failure) and ARTEMIS (post-MI) continue. **Exam message: this demonstrates biomarker–outcome dissociation.** *(JAMA Cardiology 2026)*

2. Sarilumab — first biologic approved for polymyalgia rheumatica (2023). On **28 February 2023** the FDA approved **sarilumab**, an IL-6 receptor antagonist, for adults with PMR in whom glucocorticoids proved inadequate or who cannot tolerate a taper. Approval was based on the phase 3 **SAPHYR** trial in steroid-resistant active PMR: sarilumab 200 mg every two weeks with a 14-week steroid taper versus placebo with a 52-week taper, achieving **sustained remission in 28% versus 10% at 52 weeks (p = 0.0193)**. Tocilizumab, though beneficial in PMR studies, is **not FDA-approved for PMR**; it is approved for **giant cell arteritis**, which overlaps with PMR in about half of cases. **This is the first steroid-sparing biologic option for an almost exclusively geriatric disease.**

3. IL-6 blockade in giant cell arteritis. Tocilizumab showed beneficial effects in GCA leading to its approval; a phase 3 trial of sarilumab in GCA was conducted but recruitment difficulties exacerbated by the COVID-19 pandemic led to **premature discontinuation by the sponsor**. *(Arthritis Research & Therapy 2023)*

4. IL-6 in contemporary frailty biomarker panels. IL-6 is no longer used alone. Current work combines **IL-6 with GDF-15, FGF-21, sRAGE and neurofilament light** to identify frailty in community-dwelling older adults, with **GDF-15 emerging as the strongest single predictor** of incident frailty, malnutrition and falls.

Examiner's Tip

Define IL-6 → rises with normal ageing → mechanism (raised cortisol/catecholamines stimulate, falling sex hormones disinhibit, SASP contributes) → the inflammageing paradox → clinical consequences: anorexia, sarcopenia, frailty, anaemia of chronic disease via hepcidin, atherosclerosis, depression → therapeutics: tocilizumab, sarilumab, ziltivekimab → ZEUS negative result.

Two lines that earn marks:
• "Plasma IL-6 rises with normal ageing and correlates inversely with functional ability."
• "IL-6 induces hepcidin, which blocks ferroportin, producing the functional iron deficiency of anaemia of chronic disease."

Q6. Immunosenescence and its Relevance to Vaccination

Primary source: Pathy's 6th ed. — Ch. 1, Ch. 2, Ch. 9 (preventive geriatrics), Ch. 39 (pneumonia), Ch. 43 (respiratory infection in the elderly).

High-Yield Points (Textbook Content)

Definition

1. **Immunosenescence covers all age-related changes that result in decreased immune function in the elderly** — not restricted to senescence of immune cells alone but **encompassing every immune system modification that characterises ageing**.
2. **Innate immunity is relatively little affected; adaptive (acquired) immunity is greatly affected.** This is the most important organising principle.
3. **Two components must be described together: immunosenescence (loss of specific responses) and inflammageing (excess non-specific inflammation).** The paradox is **basal immune stimulation with immune paralysis when a specific response is required**.

Changes in the Immune System With Age (Pathy's Table 2.3 — learn this list)

4. **Thymic atrophy with loss of thymic hormones**; decreased bone marrow production of B cells.
5. **Decreased cell-mediated immunity** and **decreased delayed-type hypersensitivity**.
6. Production of **lower-affinity antibodies**; **increased autoantibodies**.
7. **Increased IL-6**; **decreased IL-2 and IL-2 responsiveness**.
8. **Accumulation of memory T cells (CD45)**; decreased proliferative response to mitogens.
9. **Impaired macrophage function**; **NK cell number increases but cytotoxicity falls**.

Innate Immune Changes in Detail

0. **Toll-like receptor activation is diminished**, producing **dampened downstream cytokine responses** when a real pathogen arrives.
1. **Neutrophils** show impaired **phagocytosis, reduced ROS generation, reduced chemotaxis and decreased degranulation**.
2. **Dendritic cells** show impaired phagocytosis, cytokine release and **migratory capacity to lymph nodes**. **Impaired antigen-presenting cell activity is the hallmark of impaired innate AND acquired immunity in the elderly.**
3. **Efferocytosis** — clearance of apoptotic cells and debris from airways — is impaired.

Adaptive Immune Changes — The Basis of Vaccine Failure

4. **Thymic involution reduces thymic output**, so there are **few naive T cells relative to antigen-experienced memory T cells**. **This directly hampers vaccine efficacy** and the ability to respond to newly encountered pathogens.
5. **Cytokine production by CD4+ and CD8+ T cells is impaired**, and the **CD4:CD8 ratio is altered**.
6. **B cells: dearth of naive B cells**, and antigen-experienced cells lose function over time.
7. There is **shrinkage of the antigenic repertoire** of lymphocytes — "holes in the repertoire".
8. **Chronic CMV infection drives clonal expansion of CD8 T cells**; the resulting repertoire shrinkage **predicts mortality** in the very elderly (the **immune risk phenotype**).
9. **Haematopoietic stem cell exhaustion is thought to be a major driver of immunosenescence.**
0. Net result: **increased prevalence of infection, increased malignancy, and increased autoimmunity**, plus failure to clear **pre-malignant cells, senescent cells and misfolded proteins**.

> **The Key Vaccine Mechanism — Most Examinable Single Fact**
>
> **Studies showing reduced efficacy of pneumococcal vaccination in older versus younger adults found NO difference in IgG levels.** The explanation may be an **altered IgM response leading to reduced opsonisation and phagocytosis of bacteria**.
>
> **Antibody QUANTITY is preserved; antibody QUALITY and function are lost.**

Vaccination in Older Adults (Textbook Content)

Influenza

1. **Yearly influenza vaccination is indicated for ALL older patients** — robust, frail, demented and end-of-life alike. **More than 90% of deaths from pneumonia and influenza during epidemics occur in those aged 65 and over.**

2. Vaccination **reduces the risk of developing influenza by 50% and influenza mortality by 70%**.
3. **In nursing homes it is only 40% effective at preventing clinical illness, but is more effective at preventing pneumonia, hospitalisation and death. Vaccinating more than 80% of nursing home residents prevents outbreaks.**
4. **Adjuvanted influenza vaccine** contains a substance that **stimulates the immune response and enhances immunogenicity** — particularly valuable in **institutionalised elderly and those weakened by chronic disease**. **This is the direct clinical answer to immunosenescence.**
5. **Cocooning:** vaccinating healthcare personnel in long-term care reduces respiratory infection incidence and overall mortality among residents, **even when resident coverage is already high**. Extend the offer to regular visitors and family.

Pneumococcal

6. Pneumococcal vaccine is **less effective at producing protective antibody levels in older adults**, but still reduces **pneumococcal bacteraemia** in high-risk patients.
7. **CAPITA trial** — PCV13 reduced the incidence of pneumococcal pneumonia by **45%** in community-dwelling adults over 65. A large Japanese nursing home trial found **marked reduction in mortality and pneumonia** with PPSV23.

Herpes Zoster — The Best Illustration of Overcoming Immunosenescence

8. Zoster incidence rises with age, affecting **up to half of individuals aged 85 and over**; **post-herpetic neuralgia occurs in more than 40% of those over 60**.
9. **Zostavax (live attenuated) is only 30% protective. Shingrix (recombinant, adjuvanted) is 90% protective.** A **two-dose series is recommended from age 50**, and **those who previously received Zostavax should receive Shingrix**.

Tetanus, COVID-19 and Non-Vaccine Measures

0. **Over half of tetanus cases occur in people aged 60 and over.** Complete the primary series if history is uncertain, then **booster every 10 years**, with **at least one dose given as Tdap**.
1. **SARS-CoV-2 vaccination** is indicated for adults including those over 65, healthcare professionals, and family members in contact with older individuals.
2. **Physical activity may ameliorate immunosenescence** and thereby reduce age-related multimorbidity; **Mediterranean diet promotes epigenetic rejuvenation** (NU-AGE study).

Current Practice Update

Aspect	Pathy's 6th ed. (2022)	Current Evidence (2023–2026)
Pneumococcal age threshold	Routine from age 65	**Lowered to age 50 (ACIP, Oct 2024)**
Pneumococcal products	PCV13 then PPSV23 one year later	**PCV20 or PCV21 alone, or PCV15 then PPSV23**
New pneumococcal vaccine	Not available	**PCV21 (CAPVAXIVE) — 21 serotypes, adult-targeted**
RSV vaccination	Not available	**Three vaccines licensed; routine at 75+, risk-based 50–74**
High-dose influenza vaccine	Adjuvanted vaccine mentioned	**DANFLU-2 missed its primary endpoint**
Frailty as vaccine modifier	Associated with poor response	Now a formal **RSV eligibility criterion**

Latest Updates (2023–Present)

1. Pneumococcal vaccination — age threshold lowered to 50 years. As of **23 October 2024**, ACIP recommends that all PCV-naive adults aged **50 and over** receive a single dose of a pneumococcal conjugate vaccine — **PCV15 (Vaxneuvance), PCV20 (Prevnar 20) or PCV21 (Capvaxive)**. If PCV15 is used it must be followed by **PPSV23 at least one year later**, or after **8 weeks** in the immunocompromised or those with a cochlear implant or CSF leak. Before October 2024, PCV was recommended only for all adults 65 and over plus those aged 19–64 with risk conditions. PCV21 was FDA-approved in June 2024. *(MMWR 2025;74(1):1–8)*

2. RSV vaccination — an entirely new vaccine for older adults. Three vaccines are FDA-approved: **Abrysvo, Arexvy and mResvia**. ACIP routinely recommends a **single dose for all adults aged 75 and over**, and a single dose for adults **50–74 at increased risk** due to high-risk conditions, **frailty**, or **high-risk living arrangements such as residence in a long-term care facility**. RSV vaccination is currently a **single dose only**; revaccination is not recommended. **Frailty and institutionalisation are now formal eligibility criteria — a direct clinical application of immunosenescence.**

3. DANFLU-2 — high-dose influenza vaccine misses its primary endpoint. A pragmatic registry-based open-label randomised trial in Denmark across 2022/23 to 2024/25 enrolling **332,438 adults aged 65 and over**, randomised 1:1 to high-dose or standard-dose inactivated influenza vaccine. The high-dose vaccine **did not significantly lower hospitalisation for influenza or pneumonia**. However, hospitalisation for **any cardiorespiratory disease was lower** with high dose (relative vaccine effectiveness 5.7%; 95% CI 1.4–9.9%). A prespecified secondary analysis of 43,881 participants with diabetes found high-dose vaccine associated with reduced cardiorespiratory, cardiovascular, heart failure, influenza and laboratory-confirmed hospitalisations irrespective of diabetes status. **Exam message: enhanced-immunogenicity vaccines improve immune response and softer clinical endpoints, but a hard mortality benefit over standard dose has not been demonstrated.** *(NEJM 2025; JAMA Intern Med 2026)*

Examiner's Tip

Define immunosenescence → innate spared, adaptive affected → list Table 2.3 changes → thymic involution → fewer naive T cells, more memory T cells, shrunken repertoire, CMV-driven clonal expansion → inflammageing paradox → **consequence for vaccines: normal IgG titres but defective IgM/functional antibody** → strategies to overcome.

The four strategies to overcome immunosenescence — quote as a block:
(1) Adjuvants **(2)** Higher antigen dose **(3)** Recombinant/conjugate platforms **(4)** Herd protection by vaccinating carers

Q7. Caloric / Dietary Restriction in Ageing

> *Primary source: Pathy's 6th ed. — Ch. 1, Ch. 13 (anorexia of ageing), Ch. 14 (weight loss); supplemented by Essentials of Clinical Geriatrics (Ch. 1).*

High-Yield Points (Textbook Content)

Definition & Concept

1. **Caloric restriction (CR) means reducing caloric intake without depriving essential nutrients** — described in the textbook as **"nutritionally balanced semi-starvation."**
2. **Dietary restriction remains the single major intervention shown to enhance longevity** across species — the reference standard against which all anti-ageing interventions are compared.
3. CR sits within **deregulated nutrient sensing**, one of the **antagonistic hallmarks of ageing**, alongside the **insulin/IGF-1 axis, mTOR, AMPK and sirtuins**.

Evidence Across Species — Learn This Ladder

4. **Yeast, worms (C. elegans), flies (Drosophila) and rodents: CR reliably increases lifespan.** In rodents it **delays maturation** and can extend lifespan **by as much as 50%**.
5. **Primates: the data are conflicting.** The Wisconsin group (2009) reported CR **delayed disease onset and mortality in rhesus monkeys**; a later study did not confirm this.
6. **Humans: body weight below ideal is NOT associated with increased lifespan.** This is the crucial translational failure point.
7. **CR is therefore judged unlikely to be a successful intervention in humans** — hence the shift to **CR mimetics**.

Mechanisms

8. The mechanism is not fully understood but is thought to relate to **reduced IGF-1 levels**.
9. **Reduced GH signalling, and reduced insulin/IGF-1, are associated with increased longevity** in rodents, worms and flies.
0. **CR lowers oxidative stress and oxidative damage** and extends the maximum lifespan of rodents.
1. **CR acts epigenetically** — multiple effects on **chromatin through sirtuins, TOR and other factors**, making these molecules **targets for lifespan extension without actual food restriction**.
2. **mTOR senses cellular nutrient levels** and regulates rates of **protein synthesis and energy utilisation**.
3. **Protein restriction alone works too:** animals fed **isocaloric diets with reduced protein have increased lifespan**.
4. In animal models CR **decreases incidence and delays onset of disease** — including **chronic glomerulonephritis, muscular dystrophy and carcinogenesis**.

Caloric Restriction Mimetics

5. **Rapamycin (mTOR inhibitor)** extended lifespan in genetically heterogeneous mice **even when begun in late life**; the barrier to human use is **immunosuppression**.
6. **Metformin** is a candidate anti-ageing drug for its effects on **nutrient sensing, mitochondrial dysfunction, DNA damage and inflammation**.
7. **Sirtuin activation and resveratrol** produced initial excitement but were **not replicated**; they may **extend healthspan rather than lifespan**.

Caloric Restriction as a Clinical Intervention in Older Adults

8. In overweight older adults, **short-term caloric reduction improves blood pressure, cholesterol and metabolic rate — but these benefits require at least a 20% reduction in caloric intake.**
9. **Critical geriatric finding:** in long-term follow-up of weight-loss RCTs, the **caloric-restriction group lost more fat mass BUT ALSO more lean body mass** than the exercise group, and **in the long term there was no difference in physical performance** between the two.
0. **Look AHEAD:** intensive lifestyle intervention maintained weight loss, fitness and CV risk factor improvement, **but showed no reduction in cardiovascular events**, and there was **marginally greater cognitive decline in obese subjects over five years**.
1. **Improvement in comorbidities is largely independent of whether weight is actually lost** — blood pressure falls with dietary change without weight loss, CAD prevention is independent of weight loss, and **physical function improves without weight loss**.
2. A randomised trial found **no mortality difference** between those who lost a mean of **4.4 kg** and controls.

Why Caloric Restriction Is Dangerous in the Old — The Counter-Argument

23. **The anorexia of ageing means older people are already calorie-restricted involuntarily.** Energy intake falls by about **30% between ages 20 and 80** (NHANES III: **−1321 kcal/day in men, −629 kcal/day in women**).
24. **16–18% of community-dwelling older people already consume fewer than 1000 kcal daily.**
25. **Loss of homeostatic compensation (Roberts et al.):** when young and old men were underfed by about 750 kcal/day for 21 days, the **young men over-ate afterwards and rapidly regained weight; the old men did not compensate and never regained the lost weight. This is the single most important reason not to prescribe CR to older adults.**
26. **Weight loss after age 60 is disproportionately of lean tissue** — up to **3 kg of lean body mass lost per decade after age 50**, i.e. sarcopenia.
27. **Unintentional weight loss of 5% or more over 6–12 months signals adverse outcomes; 10% or more very likely means protein-energy malnutrition.**
28. **Ideal weight ranges are almost certainly higher in older than in younger adults.** A **BMI of 25–29.9 kg/m^2 carries no significant excess mortality** in older adults, and **BMI over 30 only about 10%** excess.
29. **Malnutrition prevalence** (Mini Nutritional Assessment): overall **22.8%** — rehabilitation units **50.5%**, hospitalised **38.7%**, community-dwelling **5.8%**.
30. **Protein-energy malnutrition is a strong independent predictor of mortality** in older people, in every care setting.

Current Practice Update

Aspect	Textbook Position	Current Evidence (2023–2026)
Human evidence for CR	None; considered impractical	**CALERIE RCT — first positive human trial**
Outcome measured	Lifespan / mortality	**Pace of biological ageing (DunedinPACE)**
Size of effect	Not quantified in humans	**2–3% slowing of pace of ageing**
Which clock responds	Not addressed	**DunedinPACE yes; PhenoAge and GrimAge no**
Alternative protocols	Continuous CR only	**Intermittent fasting and time-restricted eating**
Main geriatric risk	Lean mass loss	**Confirmed — TRE causes disproportionate lean mass loss**

Latest Updates (2023–Present)

1. CALERIE — first randomised human evidence that CR slows ageing. In a post hoc analysis of CALERIE, in which **220 adults without obesity were randomised to 25% caloric restriction or ad libitum diet for two years**, the intervention **slowed the pace of ageing measured by DunedinPACE**, but did not significantly change PhenoAge or GrimAge. The change corresponded to roughly a **3% slowing of the pace of ageing**; in independent studies a 3% slower DunedinPACE is associated with a **15% lower risk of death** — described as comparable to a smoking cessation intervention. **This is the first randomised evidence supporting the geroscience hypothesis in humans, and shows pace-of-ageing clocks are more sensitive to intervention than static biological-age clocks.** *(Nature Aging 2023)*

2. Intermittent fasting and time-restricted eating in adults 60 and over. A 2026 systematic review and network meta-analysis of seven RCTs found **moderate-certainty evidence** that **time-restricted eating (16:8)** and intermittent Sunnah fasting produce significant reductions in body weight (**ISF −2.36 kg; TRE 16:8 −1.92 kg**) and BMI versus usual diet, with improvements in HbA1c and blood pressure — while calling for longer trials with longitudinal muscle mass evaluation and sarcopenia biomarkers.

3. The lean mass warning — directly relevant to geriatrics. An RCT in adults with overweight or obesity found a 12-week hypocaloric time-restricted eating regimen caused **greater loss of lean mass** than a conventional hypocaloric diet, with lean mass accounting for approximately **65% of total weight loss** — far higher than the typical 20–30% with standard hypocaloric diets — leading to explicit calls for caution in those at risk of sarcopenia. A cross-sectional study of **3,680 community-dwelling Chinese adults aged 60 and over** found TRE significantly associated with **increased risk of severe sarcopenia**, possibly because compressing the eating window concentrates protein intake and fails to provide sustained substrate for muscle protein synthesis in a population with **anabolic resistance**. *(Frontiers in Nutrition 2025)*

4. The protein counter-prescription. Because exercise and protein intake are the primary anabolic stimuli preserving skeletal muscle with ageing, current recommendations are to distribute protein evenly across waking hours at approximately **0.4 g/kg per meal**, with a daily intake of about **1.2 g/kg/day**. **Any restriction protocol in an older adult must be paired with adequate protein and resistance exercise.**

Examiner's Tip

Define CR → evidence ladder (yeast/worm/fly/rodent positive → primates conflicting → humans no lifespan benefit) → mechanisms (reduced IGF-1, mTOR, sirtuins, reduced oxidative stress, epigenetic) → CR mimetics → CALERIE as first human RCT → **then pivot to the geriatric counter-argument** (anorexia of ageing, loss of homeostatic compensation, lean mass loss, higher ideal BMI in the old).

Two sentences that earn the highest marks:

• "Older adults do not compensate after underfeeding — unlike young adults, they do not regain the lost weight."

• "Caloric restriction is a longevity strategy for the young and middle-aged; in the frail older adult the priority is the opposite — preventing undernutrition and sarcopenia."

Q8. Anti-Ageing Interventions

Primary source: Pathy's 6th ed. — Ch. 1, Ch. 2, Ch. 7, Ch. 9; supplemented by Essentials of Clinical Geriatrics.

High-Yield Points (Textbook Content)

Rationale & Framework

1. **Geroscience hypothesis:** the accumulation of diseases and loss of function with ageing is driven by **common biological mechanisms**. Targeting these should **delay multiple diseases simultaneously** and extend **healthspan**.
2. **Interventions that slow ageing are likely to delay the onset of cancer, diabetes, cardiovascular disease and neurodegenerative disease together** — the fundamental argument for anti-ageing medicine.
3. Pathy's lists the therapeutic opportunities as **senolytics, inflammasome inhibitors, mesenchymal stem cells, calorie-restriction mimetics and autophagy inducers** — plus **lifestyle interventions (physical activity, diet)**.

A. Exercise — The Best-Evidenced Intervention

4. **Habitual physical activity increases average life expectancy by about two years.**
5. **Many changes attributed to biological ageing can be prevented by chronic physical activity**, particularly the body-composition changes: **decreased muscle mass, decreased bone mass and strength, increased adipose tissue and central fat deposition**.
6. Exercise prevents and/or treats **obesity, cardiovascular disease, type 2 diabetes, hypertension, osteoarthritis, osteoporosis, stroke, peripheral vascular disease, renal failure, dementia and depression**.
7. **Strong evidence** that exercise improves **muscle strength, muscle mass, bone density, falls incidence and mobility** across the whole health and frailty spectrum.
8. **High-intensity resistance training is the optimal prescription for sarcopenia**, also improves balance, and **improves aerobic capacity as much as moderate-intensity aerobic training** — one prescription targeting both major deficits.
9. **Aerobic exercise does NOT improve strength or balance and is insufficient as an isolated prescription.** Falls-prevention programmes based on **walking are inferior** to strength-and-balance programmes and have been **associated with increased fracture rates** in those at risk.
0. **Explosive resistance training ("power training") is central**, because muscle **power** declines far more steeply than strength with age (fast-twitch fibre atrophy plus altered neural recruitment).
1. **Multicomponent exercise (resistance + balance + gait retraining) improves all the hallmarks of frailty.** The **VIVIFRAIL** programme provides individualised prescription "passports" based on **SPPB and gait speed**.
2. **Sequence in the very frail follows the physiology of mobility: strength/power to stand up → balance to stay upright → endurance to walk.**
3. **High-intensity strength training works even in nonagenarians** (Fiatarone, 1990) and in **acutely hospitalised and institutionalised** older adults.
4. **Physical activity may ameliorate immunosenescence** and thereby reduce age-related multimorbidity.

B. Nutritional Interventions

5. **Caloric restriction** increases lifespan by up to **50%** in mammals, acting through **reduced IGF-1, sirtuins and TOR** — but is **not practical in humans**, hence the search for **CR mimetics**.
6. **Mediterranean diet promotes epigenetic rejuvenation** (NU-AGE study).
7. **Antioxidant vitamins have failed:** there is **no significant loss of scavenging enzymes with ageing**, and **vitamins C and E do not increase longevity in experimental animals**. Broad-spectrum vitamin supplementation is nonetheless considered reasonable for most older people.
8. **Vitamin D 800–2000 IU daily** reduces fracture risk and prevents falls. **Salt restriction and weight loss (TONE trial)** effectively lower blood pressure in older people.

C. Hormonal Interventions — Mostly Negative

9. **Growth hormone:** GH falls with age causing muscle loss, **but GH replacement did NOT restore muscle strength despite increasing muscle mass, and was associated with increased cardiovascular mortality risk**, glucose intolerance and diabetes. Paradoxically, **reduced GH signalling correlates with increased longevity in rodents**.
0. **DHEA:** levels fall with age, but **replacement had no beneficial effect on memory or muscle strength** — only a slight effect on libido, mainly in women.
1. **Oestrogen:** KEEPS showed control of postmenopausal symptoms without a serious increase in cardiovascular mortality or VTE, but **HRT is not recommended after age 60**. WHI showed observational benefits were exaggerated and risks of

heart disease, stroke and cancer.

2. **Testosterone:** free testosterone falls with age (raised SHBG, raised fat mass) and correlates with **muscle mass, strength, frailty, sexual function and cognition**. Replacement improves symptoms and reverses **lower urinary tract symptoms**. Trials show benefit in **grip strength, Functional Independence Measure, and post-operative ability to stand**, with effects **confined to men aged 75 and over and the frail**. Evidence is **not conclusive but justifies further study**.
3. **Key exam line: growth hormone and testosterone cannot be recommended for older men with normal or low-normal hormone levels.**

D. Cellular & Pharmacological

4. **Senolytics** — selective elimination of senescent cells **attenuates age-related deterioration of several organs and extends lifespan in mice**. **Rapamycin** extends lifespan in mice even when begun late (barrier: immunosuppression). **Metformin** acts on nutrient sensing, mitochondrial dysfunction, DNA damage and inflammation. **Anti-IL-1beta (canakinumab) reduced the incidence of age-related diseases in humans.**
5. **Stem cells and epigenetic reprogramming:** transplanting **muscle-derived stem cells from young mice extended health- and lifespan in progeroid mice even where donor cells were undetectable**, suggesting benefit via **secreted factors**. However, **embryonic and pluripotent stem cells are NOT a reversal of ageing — they form tumours**. **Epigenetic reprogramming carries a malignancy risk**; the goal is to **reset the ageing clock while leaving the differentiated state untouched**.

Current Practice Update

Intervention	Textbook Position	Current Evidence (2023–2026)
Caloric restriction	Impractical in humans; no human data	**CALERIE: 2–3% slowing of DunedinPACE**
Senolytics	Mouse data only	**Phase 1 human trials complete; Phase 2 underway**
Metformin	Promising geroprotector	**21-year DPP follow-up: no multimorbidity benefit**
Epigenetic reprogramming	Conceptual, high malignancy risk	**OSK (c-Myc omitted) + cyclic dosing; first-in-human planned**
Framework	9 hallmarks	**12 hallmarks (Cell 2023)**
Measuring success	Lifespan/mortality	**Validated biomarkers of ageing (Nat Med 2024 framework)**

Latest Updates (2023–Present)

1. CALERIE — first randomised human evidence that an intervention slows ageing. 220 adults randomised to 25% caloric restriction versus ad libitum diet for two years showed slowing of the pace of ageing by DunedinPACE, without significant change in PhenoAge or GrimAge; roughly **3% slowing**, associated with **15% lower risk of death**. *(Nature Aging 2023)*

2. Senolytics reach human trials. STOMP-AD and STAMINA tested 12 weeks of intermittent oral **dasatinib plus quercetin** in older adults with or at risk of Alzheimer's disease; in STAMINA, reductions in plasma TNF-alpha correlated with MoCA improvement ($r = -0.65$, $p = 0.02$). Safety, feasibility, CNS penetrance and biological activity were established, and a **Phase 2 RCT is now underway**.

3. Metformin as a geroprotector — a major setback. In a 21-year DPP follow-up, metformin did not significantly reduce multimorbidity risk; **85% of participants developed two or more chronic conditions** (82% lifestyle, 85% metformin, 87% placebo). Intensive lifestyle intervention was associated with lower long-term multimorbidity burden. TAME remains the pending definitive test. *(JAMA 2026)*

4. Partial epigenetic reprogramming moves toward the clinic. In vivo reprogramming through transient Yamanaka factor expression shows promise for restoring regenerative competence across retina, skeletal muscle, heart, liver, brain and intestine, but carries critical safety concerns — **teratoma formation, organ failure and loss of cell identity** — mitigated by **cyclic induction and targeted delivery**. A protocol of **2 days on and 5 days off per week** has allowed safe long-term reprogramming; in neuron-restricted models cyclic induction improves hippocampal activity, restores youthful heterochromatin and enhances memory, whereas **continuous expression is ineffective or deleterious**. A platform using **Oct4, Sox2, Klf4 (OSK), omitting the c-Myc oncogene**, has its lead programme **ER-100** in development for optic neuropathies including glaucoma and NAION, with plans to enter the clinic in Q1 2026. *(Aging Cell 2025)*

5. The field now has validation standards. The Biomarkers of Aging Consortium published recommendations on validating biomarkers of ageing, noting systematic validation is required before use as surrogate endpoints in gerotherapeutic trials. *(Nature Medicine 2024)*

Examiner's Tip — Classify by Evidence Strength

PROVEN — exercise (high-intensity/power resistance + balance + multicomponent), smoking cessation, BP and lipid control, vaccination, vitamin D, Mediterranean diet.
PROMISING — caloric restriction and its mimetics (rapamycin, metformin), senolytics, anti-inflammatory agents.
DISPROVEN OR HARMFUL — growth hormone, DHEA, antioxidant megadoses, HRT after 60.
EXPERIMENTAL — stem cells, partial epigenetic reprogramming, telomerase activation.

The line that earns the mark: "Exercise remains the only intervention with proven benefit on physical function, cognition, falls, multimorbidity and mortality in older humans — every pharmacological geroprotector is still investigational."

Q9. Interventions for Healthy Ageing

Primary source: Pathy's 6th ed. — Ch. 1, Ch. 7, Ch. 9, Ch. 12–14 (nutrition); supplemented by Essentials of Clinical Geriatrics (Ch. 5, Prevention).

High-Yield Points (Textbook Content)

Concept & Definitions

1. **WHO defines healthy ageing as the process of developing and maintaining the functional ability that enables wellbeing in older age.**
2. **"Functional ability — retaining autonomy and independence as people age — is the cornerstone of healthy ageing."**
3. **Intrinsic capacity** is the **composite of all of an individual's physical and mental capacities** and is the **key determinant of functional ability**. Domains: **cognition, psychological state, sensory function (vision and hearing), vitality/nutrition and locomotion**.
4. **Healthspan = duration of life in good health.** Fries described **compression of morbidity** — medicine **rectangularises the survival curve**, delaying death closer to maximum lifespan without changing maximum lifespan itself.
5. **Key conceptual point:** "healthy ageing" and "successful ageing" are **biologically vague as descriptions**. Their proper function is **prescriptive** — a norm of intervention: **"keep as many people as possible as healthy as possible for as long as possible."**
6. **Frailty is the clinical counterpart** — loss of in-built capacity across multiple systems with reduced physiological reserve and resilience. **The focus in older patients should be on functionality rather than disease diagnosis.**

Measuring Healthy Ageing

7. **Physical function is proposed as the biomarker of healthy ageing** — aerobic capacity, **gait speed, balance, mobility and muscle strength** — predicting adverse events, disability and mortality.
8. **Gait speed predicts mortality in older adults better than chronic diseases such as hypertension.**
9. Older adults with **handgrip strength above the weakness thresholds** had lower odds of adverse events across most intrinsic capacity domains and lower hospitalisation rates.
0. **Multimorbidity is NOT the most important factor modulating intrinsic capacity domains** — function matters more than disease count.

Pillar 1 — Exercise (Strongest Evidence)

1. **Habitual physical activity increases average life expectancy by about two years** and prevents most of the body-composition changes of ageing.
2. Exercise prevents/treats **obesity, cardiovascular disease, type 2 diabetes, hypertension, osteoarthritis, osteoporosis, stroke, peripheral vascular disease, renal failure, dementia and depression**.
3. **Multicomponent exercise (resistance + balance + gait retraining) improves all the hallmarks of frailty. VIVIFRAIL** gives individualised "prescription passports" based on **SPPB and gait speed**.
4. **High-intensity and explosive (power) resistance training** is the optimal prescription for **sarcopenia**, and also improves **balance and cognition** (particularly in MCI).
5. **Sequence in the frail follows the physiology of mobility: strength/power to stand → balance to stay upright → endurance to walk.**
6. **Aerobic exercise alone does not improve strength or balance. Walking-based falls programmes are inferior** to strength-and-balance programmes and have been associated with **increased fracture rates** in those at risk.

Pillar 2 — Nutrition

7. **Screen for the anorexia of ageing and malnutrition** using **SNAQ or MNA** whenever weight falls **more than 5 lb (2.3 kg) per year**; diagnose using **GLIM criteria** (phenotypic + aetiologic).
8. **Oral nutritional supplements produce 1–3 kg weight gain over 1–3 months**, but have **not shown mortality or functional benefit in the general elderly population** — benefit is confined to those who are **frail, malnourished or recently ill**.
9. **Mediterranean diet promotes epigenetic rejuvenation** (NU-AGE); adequate **protein** and **vitamin D 800–2000 IU/day** support muscle and bone.

Pillar 3 — Preventive Geriatrics (Stratify by Prognosis)

0. **The Health Maintenance Clinical Glidepath stratifies every intervention into four categories:**
    - **Robust** — life expectancy over 5 years, functionally independent

- **Frail** — life expectancy under 5 years or significant functional impairment
- **Moderately demented** — life expectancy 2–10 years
- **End of life** — life expectancy under 2 years, functionally dependent

1. **At EVERY visit, for all categories:** blood pressure **including orthostatics**, weight, **pain assessment**, **medication review including OTC and herbal**, **awareness of elder abuse**, and **Rapid Geriatric Assessment**. Lifestyle education at each visit for the robust and frail.
2. **Yearly:** ADLs and IADLs, urinary incontinence, ADAM screen for hypogonadism in men; **consider** annual visual acuity and auditory testing.
3. **Immunisation:** influenza yearly in **all four categories**; pneumococcal; tetanus booster every 10 years with at least one dose as Tdap; **zoster — Shingrix 90% protective versus Zostavax 30%**.
4. **Screening tied to life expectancy:** osteoporosis by **DXA plus FRAX**; **one-time AAA ultrasound** in male ever-smokers aged 65–75; sleep apnoea (Epworth); targeted lipid and TSH screening; **cancer screening only if life expectancy is 10 years or more**; **smoking cessation must be prioritised at every encounter**.

Pillar 4 — Psychosocial & Environment

5. **Plasticity is retained into old age** — cognitive training, physical training and even neurogenesis are demonstrable. Longitudinal cohorts show that **good vision, volunteer or paid work, physical activity and IADL independence independently predict continued functional independence over 7 years**. Address the transitions of ageing — **retirement, bereavement, loss of driving, sensory loss** — and support **social connectedness and home environment modification**.

Current Practice Update

Aspect	Pathy's 6th ed. (2022)	Current Evidence (2023-2026)
Operational framework	Intrinsic capacity as a concept	**WHO ICOPE Handbook 2nd edition (2024) + App**
Who delivers it	Geriatrician-led	**GPs, nurses and community health workers — no specialist needed**
Real-world data	None	**INSPIRE ICOPE-CARE: over 3000 followed, delayed functional loss**
Dementia prevention	Risk factors listed generally	**Lancet Commission 2024: 14 factors, ~45% preventable**
New dementia risk factors	Not included	**High LDL cholesterol and untreated vision loss**
Multidomain trials	FINGER only	**7 of 10 trials in the 2020s show cognitive benefit**
Biological validation	Not possible	**CALERIE: lifestyle intervention slows DunedinPACE 2–3%**

Latest Updates (2023–Present)

1. WHO ICOPE Handbook, 2nd edition (2024) — the operational tool for healthy ageing. It supports health and care workers to put evidence-based recommendations into practice in primary care and community settings, describing pathways to **detect declines in intrinsic capacity, identify social care and support needs, and develop a personalised care plan**; an interactive App generates a printable summary. The pathway is designed for **general practitioners and trained non-experts such as community health workers or nurses, without requiring specialised geriatric expertise** — usable from urban clinics to rural outreach. Pilot programmes in **India, Mexico and Vietnam** demonstrated feasibility in resource-constrained environments with improved functional outcomes and reduced healthcare utilisation. In France, the **INSPIRE ICOPE-CARE** programme had over **3000 individuals actively followed** by October 2024, with high adherence and evidence of delayed functional loss. *(European Geriatric Medicine 2026)*

2. ICOPE Step 1 screening — what it actually finds. In a Singapore feasibility study of **367 community-dwelling older adults (mean age 71.8)**, ICOPE Step 1 screening found **284 (77.4%) with signs of intrinsic capacity decline** — commonest being **visual impairment (42.0%), hearing loss (33.5%) and cognitive deterioration (31.3%)**, followed by limited mobility (24.3%), malnutrition (16.1%) and depressive symptoms (16.1%). **Clinical message: sensory impairment is the commonest reversible threat to intrinsic capacity — screen vision and hearing first.**

3. Lancet Commission 2024 — dementia prevention as healthy ageing. The 2024 report adds **untreated vision loss and high LDL cholesterol**; around **45% of dementia cases are potentially preventable** by addressing **14 modifiable risk factors** across the life course: low education in early life; hearing loss, hypertension, obesity, excessive alcohol, traumatic brain injury, high LDL cholesterol, depression, physical inactivity, diabetes and smoking in mid-life; and social isolation, air pollution and visual impairment in late life.

4. Multidomain interventions have come of age. Five multidomain trials published 2015–2019 including FINGER did not show consistent cognitive improvement — but in the 2020s, **seven of ten trials reported beneficial effects** of multidomain interventions on cognitive outcomes, with additional benefits on physical function, reduced multimorbidity and healthcare resource use. *(Geriatrics & Gerontology International 2025)*

5. Biological proof that lifestyle slows ageing. CALERIE showed a lifestyle intervention slowed the pace of ageing by DunedinPACE — roughly **3% slowing**, associated with **15% lower risk of death**. *(Nature Aging 2023)*

Examiner's Tip

Define healthy ageing (WHO) → intrinsic capacity vs functional ability → compression of morbidity → measure it (gait speed, grip strength, SPPB, frailty) → **FOUR PILLARS**: (1) exercise — multicomponent with power training, (2) nutrition — protein, vitamin D, Mediterranean pattern, malnutrition screening, (3) preventive geriatrics — stratified by life expectancy, vaccination, screening, deprescribing, (4) psychosocial and environmental → finish with WHO ICOPE and Lancet Commission 2024.

The line that earns the mark: "Healthy ageing is defined by function, not by the absence of disease — so the target of intervention is intrinsic capacity, and the best-evidenced intervention remains multicomponent exercise."

Q10. Active Ageing and How to Achieve It

> *Primary source: Pathy's 6th ed. — Ch. 4 (Social and Community Aspects of Ageing), Ch. 7, Ch. 9, Ch. 11; supplemented by Essentials of Clinical Geriatrics.*

High-Yield Points

Concept & Framework

1. **WHO defines active ageing as "the process of optimising opportunities for health, participation and security in order to enhance quality of life as people age."**
2. **CRITICAL DEFINITION POINT: "active" does NOT mean merely physically active or economically productive.** It means **continuing participation in social, economic, cultural, spiritual and civic affairs**.
3. **Three pillars of active ageing: HEALTH, PARTICIPATION and SECURITY.** The slogan is **"add life to years," not just years to life**.
4. **Determinants:** health and social services; behavioural factors; personal (biological and psychological) factors; physical environment; social environment; economic factors — with **culture and gender as cross-cutting determinants**.
5. **Relationship to other terms:** *Successful ageing* (Rowe & Kahn) = absence of disease + high function + engagement. *Healthy ageing* (WHO) = maintaining **functional ability**. *Active ageing* is the **broadest policy framework**, adding participation and security to health.

Pillar 1 — Health

6. **Exercise is the single most effective intervention.** Habitual physical activity adds about **2 years** to life expectancy. **Multicomponent exercise improves all the hallmarks of frailty**; **VIVIFRAIL** provides individualised prescription passports.
7. **Exercise as a mental health intervention:** high-intensity aerobic exercise is **equipotent with antidepressants** in older adults with major depression. High-intensity progressive resistance training achieved a **61% clinical response versus 29% with low-intensity training and 21% with GP care** — showing that **intensity matters**.
8. **Correct sensory impairment — the highest-yield "participation" intervention.** At least **one-third of older adults have visual impairment and up to two-thirds report hearing loss**. **Each line of reduction in presenting visual acuity increases the risk of institutionalisation by 7%.**
9. **Hearing loss is associated with social isolation, depression and cognitive impairment.** Many older adults do not volunteer it. **Personalised sound amplifiers are an inexpensive alternative** to hearing aids in mild-to-moderate loss; **low-vision rehabilitation** should be used alongside standard ophthalmic care.
0. **Preventive geriatrics stratified by prognosis:** influenza vaccine yearly in **all** categories; pneumococcal, zoster and tetanus vaccination; **medication review including OTC and herbal preparations at every visit**; nutrition screening (SNAQ/MNA) if weight loss exceeds 5 lb per year.

Pillar 2 — Participation

1. **Employment:** adults over 55 make up about **22% of the US workforce**, and **workers aged 70–75 are expected to show the largest relative growth**. Many industries are **eliminating mandatory retirement ages**.
2. **But discrimination persists** — an adult over 50 who loses a job takes **much longer to find new work** and is **more likely to accept a pay cut**.
3. **Retirement is a risk period: about one-quarter of adults report that their quality of life WORSENED after retirement** — due to **boredom, financial worries or health changes**.
4. **The retirement prescription:** counsel older adults to **participate in social activities, foster connections with friends and family, exercise regularly, and engage in mentally stimulating activities** — all correlating with **higher quality of life and lower risk of dementia and social isolation**.
5. **Distinguish clearly: social isolation = objective physical separation from others; loneliness = subjective distress at feeling separated or alone.** Correlated but **not synonymous**.
6. **Epidemiology:** about **one-quarter** of community-dwelling older adults are socially isolated; **20–45% report feeling lonely** some or all of the time.
7. **Health consequences of loneliness/isolation:** increased **anxiety, depression and suicidality**; increased risk of **cognitive impairment and delayed dementia diagnosis**; **chronic inflammation leading to cardiovascular disease**; and a **suppressed immune system**.
8. **Treatment of loneliness has no one-size-fits-all approach.** Options: **treat psychological comorbidity, optimise hearing and vision, arrange reliable transport, cognitive-behavioural therapy, peer groups based on shared language/culture/interests, volunteering, senior centres, companionship volunteer organisations, and engagement with a religious community**.

9. **Technology enables participation:** two-thirds of Americans over 65 use the internet, 4 in 10 own a smartphone, 30% use social media. Benefits include **online ordering, on-demand transport, online banking, telehealth and chronic disease self-management**. Barriers are **lack of confidence and physical difficulty with devices**; counsel on **scams and misinformation**.
0. **Measure participation:** the **Saint Louis University Social Activities Assessment** quantifies frequency of going out socially, gardening, worship attendance, telephone contact, eating out, walking, dancing, concerts/theatre, time with grandchildren, and **satisfaction with social activity**.

Pillar 3 — Security

1. **Financial security:** only about 30% of older adults receive pension income and about 21% still earn from employment. **Older women, particularly those who never married, are at the highest risk of poverty.** Globally, poverty rates in over-65s range from **3% (Netherlands, Czech Republic) to about 50% (Republic of Korea) to 80% (Zambia)**.
2. **Elder abuse affects an estimated 5–10% of older adults, and nearly half of those with dementia.** It is grossly under-recognised — one US emergency department study reported an incidence of **0.013%**, orders of magnitude below true prevalence. Refer to **Adult Protective Services** or the local equivalent; report to the **ombudsman** as well if abuse occurs in a nursing home.
3. **Scams and fraud:** older adults are preferentially targeted. Protective measures include **freezing credit, credit monitoring, and granting a trusted family member account access to monitor for suspicious activity**.
4. **Housing — "age in place" first.** Suboptimal reasons to move (structural problems, financial difficulty, mobility limitation, perceived isolation) should first be addressed with **grants and volunteer repair services, social work input, PT/OT assessment with ramps and assistive devices, and community engagement**. Ideal reasons to move include an **unsafe or unimprovable environment or inability to live alone safely**. Innovative models include **senior-friendly communities, co-housing and student-for-services schemes**.

Cross-Cutting — Combating Ageism

Ageism — "one of the last socially acceptable forms of prejudice" — is stereotyping (how we think), prejudice (how we feel) and discrimination (how we act) on the basis of age. In healthcare it causes **delirium and disability to be dismissed as "normal ageing"**, and older people with ischaemic heart disease to be **less thoroughly investigated and less often offered intervention even when clinically indicated**. The geriatrician's unique skill is **striking the balance between therapeutic nihilism and therapeutic heroism**.

Frameworks Institute strategies (Pathy's Table 4.1):

- **Eliminate the "othering"** — say "when we age", not "when they age"
- **Reframe ageing** — avoid "fighting" or "battling" ageing; emphasise wisdom and life satisfaction, but **avoid unrealistic idealism**
- **Increase awareness** — call out ageism when encountered
- **Emphasise that social policies (transport, housing, social security) influence ageing** — not just individual choices
- **Appeal to values of justice, inclusion and equity**

Current Practice Update

Aspect	Pathy's 6th ed. (2022)	Current Evidence (2023–2026)
Global policy vehicle	WHO active ageing framework	**UN Decade of Healthy Ageing (2021–2030)**
Action areas	Health/participation/security	**Ageism, age-friendly environments, integrated care, long-term care**
Implementation tool	None specified	**WHO ICOPE Handbook 2nd edition (2024) + App**
Monitoring	Not addressed	**TAG4MHA indicator framework (2025); mid-term evaluation 2026**
Elder abuse response	Refer to APS	**WHO database of 89 promising interventions (2024)**
Social isolation	Health consequences described	**Now a formal modifiable dementia risk factor (Lancet 2024)**

Latest Updates (2023–Present)

1. The UN Decade of Healthy Ageing (2021–2030). Led by WHO, it focuses on four action areas — **age-friendly environments, combating ageism, integrated care and long-term care** — with the vision to **"add life to years,"** supported by four enablers: meaningful engagement of older people, leadership and capacity building, connecting stakeholders, and strengthening data, research and innovation. **By 2030, 1.4 billion people will be aged over 60, with low- and middle-income countries hosting 80% of older populations.**

2. First progress report — where countries actually stand. Based on a survey of **136 countries** (November 2023), there was an **over-20% increase** in countries reporting legislation against ageism, legislation supporting access to assistive products, national policies on comprehensive assessment of health and social care needs, and national age-friendly cities programmes. However, **fewer than one-third of countries reported having adequate resources** to deliver on the four action areas.

3. Measurement framework and mid-term evaluation. WHO, with its Technical Advisory Group for the Measurement of Healthy Ageing, published a monitoring framework and core indicator set (May 2025), with mandatory reporting due in **2026 and 2029**. As of late 2025, **162 of 194 Member States (83%)** were participating in the mid-term evaluation survey.

4. Elder abuse — from recognition to intervention. For World Elder Abuse Awareness Day 2024, WHO released an interactive database of **89 interventions** that show promise to prevent and respond to abuse of older people — the first product of the Decade's **Intervention Accelerator** initiative.

5. Social isolation formally enters the dementia prevention agenda. The 2024 Lancet Commission includes **social isolation** among the 14 modifiable dementia risk factors, alongside hearing loss and visual impairment, with addressing all 14 potentially preventing or delaying around **45% of dementia cases** worldwide. **This gives the "participation" pillar a hard clinical endpoint.**

Examiner's Tip

Define active ageing (WHO) → stress that "active" is not merely physical activity → **THREE PILLARS: Health, Participation, Security** → determinants → then **how to achieve it** under each: Health (exercise, nutrition, vaccination, sensory correction, deprescribing), Participation (flexible employment, retirement counselling, treating loneliness, technology, volunteering), Security (financial protection, elder abuse detection, safe housing, age-in-place) → cross-cutting: combat ageism → finish with the UN Decade four action areas.

Two lines that earn marks:

• "'Active' refers to continuing participation in social, economic, cultural, spiritual and civic affairs — not merely the ability to be physically active or to work."

• "Correcting vision and hearing is the highest-yield single intervention for participation — each line of lost visual acuity raises institutionalisation risk by 7%."

Q11. Exercise in the Elderly — Beneficial or Harmful?

Primary source: Pathy's 6th ed. — Ch. 7 "Physical Fitness and Exercise" (Tables 7.5, 7.7, 7.9), Ch. 11 (Patient Safety).

THE VERDICT (state this first). Exercise in the elderly is overwhelmingly **BENEFICIAL**. Harm arises not from exercise itself but from the **wrong modality**, the **wrong intensity**, the **wrong sequence**, or inadequate supervision. Pathy's is explicit: **"Considering the current evidence of the benefits of exercise in frail older adults, it is not ethical to NOT prescribe physical exercise to these individuals, as this means doing harm by withholding evidence-based and effective treatment."**

Part A — The Case for Benefit

1. **"There is no age above which physical activity ceases to have benefits across a wide range of diseases and disabilities."** Exercise is feasible in **nursing home residents up to 101 years of age** and in **hip fracture patients within 12 weeks of surgery**.
2. **"Insufficient physical activity and excess sedentary behaviour are lethal conditions; physical activity is the antidote."** Sedentary behaviour is a potent risk factor for **all-cause and cardiovascular mortality, obesity, sarcopenia, hypertension, insulin resistance, diabetes, stroke, colon cancer, depression, dementia, osteoarthritis, osteoporosis, recurrent falls, frailty and disability**.
3. **Ageing and disuse produce nearly identical physiological changes** — much of what is attributed to chronological age is in fact deconditioning.
4. **Habitual physical activity increases average life expectancy by about two years.**
5. **Exercise reduces cardiovascular disease risk by about 50%**, even after controlling for smoking, obesity, hypertension and dyslipidaemia.
6. **It is never too late:** sedentary middle-aged adults with low fitness who became fit at follow-up had **markedly reduced cardiovascular mortality** — "preventive exercise prescriptions instituted in middle age or beyond may be as important as those initiated at younger ages."
7. **Body composition:** exercise prevents the decreases in **muscle mass and bone density** and the increase in **central adiposity**. A **5% fall in total body fat can produce a 25% or greater reduction in visceral fat**.
8. **Sarcopenia:** high-intensity resistance training is the **optimal prescription**, and — less well known — it **improves aerobic capacity as much as moderate-intensity aerobic training**, addressing both major deficits with one prescription.
9. **Frailty:** a **12-week multicomponent programme with explosive resistance training in institutionalised frail nonagenarians** improved **muscle cross-sectional area, muscle fat infiltration, maximal strength, power output, balance, gait and sit-to-stand ability, with a reduction in falls**. **Frailty is not a contraindication to robust exercise — it is one of the strongest indications for it.**
0. **Depression:** high-intensity aerobic exercise is **equipotent with antidepressants** in older adults with major depression. High-intensity progressive resistance training produced a **61% clinical response, versus 29% with low-intensity training and 21% with GP care** (equivalent to placebo).
1. **Cognition:** high-intensity resistance training is the most promising modality, particularly in **mild cognitive impairment**; four months of low-intensity **high-speed** resistance training improved both cognition and physical performance in MCI.
2. **Fracture prevention:** in the **EPIDOS** study of 6901 women over 75, **low physical activity more than doubled the risk of proximal humerus fracture (RR 2.2)** — a **greater** risk than **low bone density (RR 1.4), maternal history of hip fracture (RR 1.8) or impaired balance (RR 1.8)**.
3. **Hospital and institutional settings:** exercise reduces functional decline in **very elderly patients during acute hospitalisation** and improves cognition; **triceps strength is critical to transfer ability, and improving it reduces nursing home admission after hip fracture**. The VIVIFRAIL programme **protected institutionalised older adults from severe functional decline through 14 weeks of COVID confinement**.
4. **Randomised trial evidence exists** for exercise in the prevention of **secondary cardiovascular events, diabetes mellitus and osteoporotic fracture**.

Part B — The Case for Harm (where marks are actually won)

5. **WRONG MODALITY IS HARMFUL.** "Falls-prevention programmes **inclusive of walking are inferior** to those focusing on strength and balance exercises and have also been **associated with increases in osteoporotic fracture rates in those at risk.**" **Walking is not a falls-prevention intervention.**
6. **WRONG SEQUENCE IS HARMFUL.** "Prescribing progressive aerobic training in the absence of sufficient balance or

strength is likely to result in **knee pain, fear of falling, falls, fractures**, and limited ability to progress aerobically — and **is not recommended**."

7. **Musculoskeletal injury with power training:** risk of **injury to tendons and cartilage, particularly the rotator cuff and knee** where degenerative tears are commonplace, and **exacerbation of abdominal or inguinal hernias**. Avoid **heavy repetitive workloads** and **unfavourable positioning** such as overhead or military press in rotator cuff disease.
8. **BUT the measured risk is very small:** a systematic review of resistance training in frail older adults reported **only ONE case of shoulder pain across 20 studies and 2544 subjects**.
9. **HIIT is unproven in the frail:** high-intensity interval training (85–95% peak heart rate for 1–4 minutes) has been studied mainly in **healthy and cardiovascular cohorts**; its **feasibility in frail older adults with multiple comorbidities remains to be established**.
0. **Low-intensity exercise is not "safer" — it is ineffective, which is its own harm.** Low-intensity resistance training is **ineffective in peripheral vascular disease**; low-intensity aerobic training in depression is **no better than social contact**; and there is **no evidence of benefit for low-intensity, minimally progressive multimodal exercise for cognitive outcomes**.
1. **Uncertain benefit in care homes:** a Cochrane meta-analysis concluded the evidence is **uncertain regarding the effect of exercise, medication review or multifactorial interventions on fall rates in care homes**.
2. **Non-responders exist:** there is significant **inter-individual variability in response** to exercise, including **adverse metabolic responses** in a minority.
3. **Acute illness requires caution:** in hospitalised patients, **"it is crucial to pay special attention to clinical vital signs before performing the physical exercise intervention, to prevent adverse events."**

Part C — How to Prescribe Safely (Pathy's Table 7.9)

Modality	Frequency	Volume	Intensity
Resistance	2–3 days/week	1–3 sets × 8–12 reps, 8–10 major muscle groups	**Borg 15–18 (70–80% 1RM)**, 6 s per rep, 1 min rest between sets
Aerobic	3–7 days/week	20–60 min/session	**Borg 12–14 (40–60% HR reserve)**
Balance	1–7 days/week	1–2 sets × 4–10 static and dynamic postures	Progressive difficulty as tolerated

Safety Rules to Quote

- **Titration method:** measure **1 RM**, then use **50%, 60%, 70% and 80% of 1 RM over the first four days**; thereafter progress by perceived exertion.
- **Power training** at **20–80% 1 RM**, concentric phase as fast as possible; **80% gives optimal adaptation**. **Screen for rotator cuff and meniscal tears first.**
- **Start with ONE modality**, then add others — introducing everything at once compromises adherence and adaptation.
- **Correct strength and balance deficits BEFORE starting aerobic training.**
- **Sequence in the frail: strength/power (to stand) → balance (to stay upright) → endurance (to walk).**
- **Flexibility/stretching** has limited independent clinical benefit — best used as a **cool-down component**.
- **Screen for sedentary behaviour at every major healthcare encounter.**

Current Practice Update

Aspect	Pathy's 6th ed. (2022)	Current Evidence (2023–2026)
Falls risk framework	Exercise recommended generically	**World Falls Guidelines: low / intermediate / high risk stratification**
Who gets exercise	All older adults	**All — but tailored by fall-risk category**
Cognitive impairment	Exercise data emerging	**WFG recommends individualised exercise even in cognitive impairment**
Global consensus	ACSM 2009 position stand	**Izquierdo 2024 ICFSR global recommendations**
Framing	Exercise as intervention	**Exercise as precision medicine — individualised prescription**
Drug review alongside	Not linked	**STOPPFall before prescribing fall-risk-increasing drugs**

Latest Updates (2023–Present)

1. World Falls Guidelines — exercise stratified by risk. The 2022 WFG classify an older adult with no fall history, or a single non-severe fall without gait or balance problems, as **low risk** — needing primary prevention and annual reassessment. Those with a single non-severe fall **plus gait or balance problems** are **intermediate risk** and benefit from a **strength and balance exercise intervention**. **High risk** includes those with a fall plus any of: accompanying injury, two or more falls in 12 months, known frailty, inability to get up for at least an hour, or suspected transient loss of consciousness. Developed by **96 multidisciplinary experts from 39 countries**. *(Age and Ageing 2022)*

2. AGS endorsement and additions (2024). The AGS response to the WFG endorsed several additions to the earlier AGS/BGS guideline: assessment and intervention for **hearing impairment** and **concern about falling**, **assessment and individualised exercises for older adults with cognitive impairment**, and performing a standardised assessment such as **STOPPFall** before prescribing a medication that could increase fall risk. *(JAGS 2024)*

3. Global exercise recommendations for longevity (Izquierdo 2024). Comprehensive global recommendations developed through an international consensus process from the ICFSR. Because programmes should be individualised, they form part of the evolving discipline of **precision medicine**; recommendations include **structured exercise supplemented by incidental lifestyle activity**, and strongly support **multicomponent interventions extending beyond aerobic exercise**. *(J Nutr Health Aging 2025)*

4. Resistance training and mobility disability. A meta-analysis of 24 articles from 22 studies (**3,656 participants, mean age 63–87**) found resistance training produced greater improvements than control in **6-minute walk distance (+16.1 m), lower extremity strength, and usual gait speed (+0.05 m/s)** — results held whether resistance training was the sole intervention or part of a multicomponent programme.

5. Falls prevention in aged care — where evidence remains weakest. WHO guidelines recommend all older adults undertake physical activity for falls prevention and other benefits including reduced mortality and improved cardiovascular, mental and cognitive health — but trial endpoint meta-analyses in **long-term residential aged care** continue to show this is the setting where evidence is least certain, consistent with the textbook position. *(Age and Ageing 2023)*

Examiner's Tip

Open with the verdict → benefits by system (mortality, CVD, body composition, sarcopenia, frailty, falls/fracture, depression, cognition, hospitalisation) → **then the harms section** (wrong modality, wrong sequence, power-training injury, HIIT unproven in frail, low-intensity ineffective, non-responders, acute illness) → resolve with the safe prescription table and sequencing rule → close with the ethical statement.

Three lines that earn the highest marks:

• "Frailty is not a contraindication to robust exercise — it is one of the most important reasons to prescribe it."
• "Falls-prevention programmes based on walking are inferior to strength-and-balance programmes and have been associated with increased fracture rates in those at risk."
• "It is not ethical to withhold exercise from frail older adults — that is doing harm by withholding effective treatment."

Q12. Advance Directives — Current Status in India

> *Primary source: Essentials of Clinical Geriatrics, 7th ed. — Ch. 17 "Ethical Issues in the Care of Older Persons" (Table 17-2); Pathy's 6th ed. Indian legal position from Supreme Court judgments and MoHFW/ICMR documents.*

Part A — Concept and Types (Textbook)

1. An **advance directive (AD)** is a **legal document that allows a patient to convey decisions about the care they want if they later become unable to express their wishes**.
2. **Purpose:** it makes the person think about care preferences **before a crisis**, simplifies decision-making, and **lets the family feel confident they did what the patient wanted** regardless of their own beliefs.
3. **Two main types:**
   - **Living will** — specifies care preferences in a **terminal state or vegetative state**, usually covering CPR, intubation, artificial nutrition and hydration, dialysis and IV therapy.
   - **Durable power of attorney for health care (proxy/agent)** — appoints a person to make decisions on the patient's behalf.
4. **Limitation of the living will:** it addresses only a few defined clinical situations. It **cannot decide the nuanced questions**, such as when an antibiotic is life-supporting versus comfort care.
5. **Components of a durable power of attorney** (Table 17-2): statement of intention citing the enabling statute; **designation of the agent with contact details**; general statement of the authority granted; **statement of desires, special provisions and limitations**; dated signature; and **witnesses — who must NOT be the agent, healthcare providers, or employees of the facility giving care**.
6. **Decision standard for the proxy:** use **substituted judgment** (what the patient would have wanted); where uncertain, fall back on **beneficence** — weighing benefits against burdens.
7. **Who should be proxy?** Not automatically the next of kin, but **the person who best knows the patient's preferences** — which may be a close friend or clergyman rather than a rarely seen relative.
8. **Pitfalls:** agents may pursue **their own interests**; surrogate decisions may be **incongruent with the patient's actual wishes**; decisions once made are hard to revoke; and **there is no reliable test of capacity** to decide whether someone can change their mind.
9. **Key clinical caution:** making end-of-life judgments while in good health is difficult. **Do not assume the old-old are "ready to die."** What a patient wants at **85 may differ from what they want at 97** — so **discussions must be ongoing, not one-off**.
0. **POLST/MOLST** (Physician/Medical Orders for Life-Sustaining Treatment) convert an AD into an **actionable standing medical order**, particularly for those who want to avoid life-sustaining treatment, live in long-term care, or **may die within the next year**.
1. **Legislation alone does not work:** the US Patient Self-Determination Act (1991) mandated that all persons entering a hospital or nursing home be offered the opportunity to make an AD, yet it had **minimal impact on the number actually completing one**.
2. **A trial of therapy is permissible** — patients and families must be told that **a treatment once started need not be continued indefinitely**.

Part B — The Indian Legal Position

Evolution

3. **Aruna Shanbaug v. Union of India (2011)** — the Supreme Court **permitted passive euthanasia** for the first time in India.
4. **Common Cause (A Regd. Society) v. Union of India (2018) 5 SCC 1** — a **five-judge Constitution Bench** held that the **right to die with dignity is a fundamental right under Article 21**. It **legalised passive euthanasia** and gave **legal recognition to Advance Medical Directives (AMD)**.
5. The **2018 procedure proved unworkable** — it required **countersignature by a Judicial Magistrate First Class (JMFC)** plus **two layered medical boards**, causing delays that defeated the purpose.
6. **Common Cause (Modification Order), 24 January 2023** — following a Miscellaneous Application by the **Indian Society of Critical Care Medicine**, the Supreme Court **substantially simplified the procedure**. **This 2023 order is the operative law today.**

Requirements of a Valid AMD in India (Post-2023)

7. Must be executed by an **adult with decision-making capacity, voluntarily and without coercion**, and be **in writing**.

8. Must state in **unambiguous terms** the decision to withhold or withdraw treatment, and specify the **circumstances in which it applies**.
9. Must be signed in the presence of **two attesting witnesses** and **attested by a Notary or a Gazetted Officer** — **the JMFC requirement has been removed**.
0. Must **nominate at least two guardians or close relatives** empowered to give consent, and a copy must be given to the **nominated persons, the family physician and the local custodian**.
1. **Revocable at any time** before implementation. An AMD **does not apply if circumstances have arisen that the executor did not anticipate** and which would have altered the decision.
2. **Applies only to terminal illness with no hope of recovery, or persistent vegetative state.**

The Implementation Process

3. **Primary Medical Board** — constituted by the hospital, minimum **three treating doctors**. If it certifies the patient is terminal/irreversible, the case goes to the **Secondary Medical Board**, which must **decide within 48 hours**. The **Chief District Medical Officer nominates one member** of the Secondary Board.
4. **Where there is NO advance directive**, the same medical board procedure applies with the consent of the family/surrogate — **High Court permission is no longer routinely required**.

Critical Distinctions

25. **Passive euthanasia (withholding/withdrawing life-sustaining treatment) is LEGAL. Active euthanasia and physician-assisted suicide remain ILLEGAL** under the Bharatiya Nyaya Sanhita provisions on abetment of suicide and culpable homicide.
26. **A separate and independent framework exists under Section 5 of the Mental Healthcare Act, 2017**, which permits advance directives for **mental health treatment**. The two frameworks are **not interchangeable**.
27. **A patient refusing life-sustaining treatment remains fully entitled to palliative care** — withdrawal of LST is never withdrawal of care.

Current Practice Update

Aspect	Textbook (US model)	India (2023–2026)
Legal basis	State statute + PSDA 1991	**Article 21; Common Cause 2018 + Modification Order 2023**
Attestation	Witnesses, ideally notarised	**Two witnesses + Notary OR Gazetted Officer**
Judicial involvement	None routinely	**JMFC requirement removed in 2023**
Actionable order	POLST/MOLST	**Primary + Secondary Medical Board; 48-hour decision**
Registry	Electronic health record integration	**Local government "custodian"; no national registry yet**
Scope of euthanasia	Varies by state	**Passive only — active euthanasia remains a criminal offence**
Uptake	Low despite legislation	**Very low; awareness minimal outside a few states**

Latest Updates (2023–Present)

1. The 2023 Modification Order — what actually changed. The clarification order **removed the requirement of Magistrate attestation**, replaced the earlier two-board arrangement with a streamlined board of **three doctors**, allowed **hospital ethics committees** to participate, and introduced **digital registration and storage**. The current position is that a valid Indian AMD requires **written form, two witnesses, and attestation by a Notary or Gazetted Officer**, and should be accessible to the nominated person and to the treating physician. *(Common Cause v. Union of India, 2023 SCC OnLine SC 99, decided 24 January 2023)*

2. MoHFW draft guidelines for withdrawal of life support (September 2024). The Ministry issued draft guidelines translating into clinical reality the streamlined procedure set out by the Supreme Court. **Terminal illness is defined as an irreversible or incurable condition from which death is inevitable in the foreseeable future**, and **severe brain injury with no recovery after 72 hours** is included. Doctors are advised to base the decision on factors including **declaration of brainstem death, poor prognosis with no likely benefit from treatment, or documented informed refusal** by the patient or family. The end-of-life care pathway is adapted from the **ISCCM–IAPC Expert Consensus and Position Statements for End of Life and Palliative Care in the ICU**.

3. State-level operationalisation — the real bottleneck. **Karnataka** became the second state after **Kerala** to implement the order, with a Health Department circular of **30 January 2025** mandating primary and secondary medical boards in hospitals to review WLST requests based on living wills, together with a state AMD format; the Indian Association of Palliative Care noted this made Karnataka the **first state government in India to make the WLST process feasible statewide**, with the secondary medical board required to confirm or refuse within **48 hours**. **Goa** became the first state to fully operationalise the AMD facility, with a Bombay High Court judge executing the first living will before two witnesses and a gazetted officer.

4. Persistent implementation problems. The orders require an officer of the local government to be nominated as **"custodian"** of all advance medical directives, but have proved challenging: decisions about withholding or withdrawing life-sustaining treatment often need to be made **within hours**, assembling six different medical experts in that time is difficult **especially in non-urban settings**, and it is impractical to approach the district Chief Medical Officer to nominate a Secondary Medical Board member for every such decision.

5. Supporting ICMR documents. Two ICMR publications — on **definitions of terms used in limitation of treatment**, and on **Do-Not-Attempt-Resuscitation** — clarified terminology and helped standardise and remove ambiguity around terminal illness, withholding/withdrawal, and end-of-life care. *(Indian Journal of Medical Research 2020 and 2026)*

Examiner's Tip

Define AD → two types (living will vs durable power of attorney/proxy) → limitations of each → **INDIAN LAW**: Aruna Shanbaug 2011 → Common Cause 2018 (Article 21, five-judge bench) → why it failed → 2023 Modification Order (notary/gazetted officer, JMFC removed, 48-hour board) → requirements of a valid AMD → Primary and Secondary Medical Board process → passive legal / active illegal → Mental Healthcare Act 2017 Section 5 as a separate track → state implementation (Kerala, Karnataka, Goa) → MoHFW 2024 draft guidelines → barriers.

Three lines that earn marks:

• "The right to die with dignity is a fundamental right under Article 21 — *Common Cause v. Union of India*, 2018."
• "The 2023 Modification Order removed the Judicial Magistrate requirement; an AMD now needs only two witnesses and attestation by a Notary or Gazetted Officer."
• "Withdrawal of life-sustaining treatment is never withdrawal of care — palliative care continues in full."

Appendix — Rapid Revision Grid

> *Last-night revision sheet: the numbers, trials and one-liners most likely to earn marks.*

1. Numbers You Must Not Get Wrong

Item	Value
Hayflick limit	**~50 population doublings**
Hallmarks of ageing	**9 (2013, in Pathy's) → 12 (2023)**
Epigenetic clock accuracy	**Error <5 years**
Human sirtuin isoforms	**7 (SIRT1–7); SIRT1 & SIRT6 = ageing**
Grip strength — low (EWGSOP2)	**<27 kg men, <16 kg women**
Low appendicular muscle mass	**<20 kg men, <15 kg women**
Caloric restriction — rodent lifespan gain	**Up to 50%**
CALERIE caloric restriction dose	**25% for 2 years, n = 220**
CALERIE effect size	**~3% slower DunedinPACE = 15% lower death risk**
Fall in energy intake, age 20→80	**~30% (men −1321 kcal/d, women −629 kcal/d)**
Lean mass lost per decade after 50	**Up to 3 kg**
Significant unintentional weight loss	**≥5% in 6–12 months (≥10% = PEM)**
Malnutrition prevalence (MNA)	**Overall 22.8%; rehab 50.5%; hospital 38.7%; community 5.8%**
Exercise — life expectancy gain	**~2 years**
Exercise — CVD risk reduction	**~50%**
Resistance training dose	**2–3 d/wk, 1–3 sets × 8–12 reps, Borg 15–18 (70–80% 1RM)**
Aerobic training dose	**3–7 d/wk, 20–60 min, Borg 12–14 (40–60% HRR)**
Influenza vaccine efficacy	**50% illness reduction; 70% mortality reduction**
Influenza vaccine in nursing homes	**40% for illness; >80% coverage prevents outbreaks**
Zoster vaccine efficacy	**Zostavax 30% vs Shingrix 90%**
Post-herpetic neuralgia over age 60	**>40%**
Deaths from pneumonia/influenza aged 65+	**>90% during epidemics**
CAPITA trial (PCV13)	**45% reduction in pneumococcal pneumonia**
Pneumococcal vaccine age threshold	**Lowered from 65 to 50 (ACIP, Oct 2024)**
RSV vaccine	**Routine at 75+; risk-based 50–74; single dose**
Vitamin D dose	**800–2000 IU/day**
Protein target in the older adult	**1.2 g/kg/day; ~0.4 g/kg per meal**
Elder abuse prevalence	**5–10% overall; ~50% in dementia**
Loneliness / social isolation	**Lonely 20–45%; isolated ~25%**
Visual acuity and institutionalisation	**Each line lost = +7% risk**
Preventable dementia (Lancet 2024)	**~45% via 14 risk factors**
ICOPE Step 1 — commonest deficits	**Vision 42%, hearing 33.5%, cognition 31.3%**
Secondary Medical Board decision (India)	**Within 48 hours**

2. Named Trials and Studies

Trial	Field	Result to quote
CALERIE	Caloric restriction	25% CR × 2 yr slowed DunedinPACE ~3%; no change in PhenoAge/GrimAge
DPP / DPPOS	Metformin as geroprotector	21-yr follow-up: **no** multimorbidity benefit; lifestyle superior
TAME	Metformin	**Awaited** — definitive geroprotector trial
SToMP-AD / STAMINA	Senolytics	Dasatinib + quercetin Phase 1 safe; TNF-alpha fall correlated with MoCA gain
ZEUS	IL-6 inhibition	Ziltivekimab, n = 6376: **HR 0.99 — no MACE benefit** despite hsCRP fall
CANTOS	IL-1beta inhibition	Canakinumab reduced incidence of age-related diseases
SAPHYR	PMR	Sarilumab sustained remission **28% vs 10%** at 52 weeks
CAPITA	Pneumococcal	PCV13 reduced pneumococcal pneumonia by 45%
DANFLU-2	Influenza	n = 332,438: high-dose **missed primary endpoint**; cardiorespiratory rVE 5.7%
EPIDOS	Fracture	Inactivity RR 2.2 > low BMD RR 1.4 for humerus fracture
Look AHEAD	Weight loss	Risk factors improved but **no CV event reduction**
TONE	Hypertension	Salt restriction + weight loss lowers BP in older adults
FINGER	Multidomain	First multidomain dementia prevention trial; 7/10 trials positive in the 2020s
NU-AGE	Diet	Mediterranean diet promotes epigenetic rejuvenation
KEEPS / WHI	HRT	HRT **not recommended after age 60**
VIVIFRAIL	Exercise	Individualised passports by SPPB + gait speed; protected residents in COVID confinement
Fiatarone 1990	Exercise	High-intensity resistance training effective in nonagenarians
Roberts et al.	Caloric restriction	Old men **fail to compensate** after underfeeding; never regain weight

3. Classifications and Frameworks

Framework	Contents
Theories of ageing	Error/stochastic • Programmed • Evolutionary
Evolutionary theories	Medawar (mutation accumulation) • Williams (antagonistic pleiotropy) • Kirkwood (disposable soma)
9 hallmarks (2013)	**Primary:** genomic instability, telomere attrition, epigenetic alterations, loss of proteostasis. **Antagonistic:** deregulated nutrient sensing, mitochondrial dysfunction, cellular senescence. **Integrative:** stem cell exhaustion, altered intercellular communication
3 added in 2023	Disabled macroautophagy • chronic inflammation • dysbiosis
Epigenetic clock generations	1st Horvath/Hannum (chronological age) • 2nd PhenoAge/GrimAge (mortality) • 3rd DunedinPACE (pace)
Intrinsic capacity domains	Cognition • psychological • sensory (vision, hearing) • vitality/nutrition • locomotion
Active ageing pillars	Health • Participation • Security
UN Decade action areas	Ageism • age-friendly environments • integrated care • long-term care
Health Maintenance Glidepath	Robust • Frail • Moderately demented • End of life
World Falls Guidelines risk	Low • Intermediate (fall + gait/balance problem) • High (injury, ≥2 falls, frailty, long lie, TLoC)
Ageism (3 components)	Stereotyping (think) • prejudice (feel) • discrimination (act)
Indian AMD requirements	Written • adult with capacity • two witnesses • Notary OR Gazetted Officer • two nominees

4. Mnemonics and Memory Hooks

Hook	Use
"MWK"	**M**edawar – **W**illiams – **K**irkwood: the three evolutionary theories, in order
"Stand → Stay → Stride"	Exercise sequence in the frail: strength/power → balance → endurance
"Quantity kept, quality lost"	Vaccine failure in the elderly: IgG normal, IgM/opsonisation defective
4 ways to beat immunosenescence	Adjuvant • higher dose • recombinant/conjugate • vaccinate the carers (cocooning)
"IL-6 → hepcidin → ferroportin block"	The whole pathogenesis of anaemia of chronic disease in one line
"Double-edged telomere"	Short = degenerative disease; long = cancer permitted
"Healthspan, not lifespan"	The correct verdict on sirtuins, resveratrol and most geroprotectors
"Function, not diagnosis"	The organising principle of healthy ageing and of geriatric assessment
"2018 → 2023"	India: right recognised (Common Cause) → procedure made workable (Modification Order)

5. Guidelines and Documents to Cite by Name

- **Cell 2023** — Hallmarks of aging: an expanding universe (12 hallmarks)
- **Nature Medicine 2024** — Biomarkers of Aging Consortium validation framework
- **Nature Aging 2023** — CALERIE / DunedinPACE
- **Lancet Commission 2024** — dementia prevention, 14 risk factors, ~45% preventable
- **WHO ICOPE Handbook, 2nd edition (2024)** + ICOPE App
- **WHO / UN Decade of Healthy Ageing 2021–2030** — four action areas; mid-term evaluation 2026
- **World Falls Guidelines 2022** (Age and Ageing) + **AGS response 2024** (JAGS); **STOPPFall**
- **Izquierdo 2024** — ICFSR global exercise recommendations for longevity
- **ACIP / MMWR 2025** — pneumococcal from age 50; RSV at 75+ and risk-based 50–74
- **EWGSOP2** — sarcopenia cut-offs; **GLIM** — malnutrition diagnosis
- **Common Cause v. Union of India (2018) 5 SCC 1** and **2023 SCC OnLine SC 99** (Modification Order)
- **MoHFW Draft Guidelines for Withdrawal of Life Support in Terminally Ill Patients (Sept 2024)**
- **ISCCM–IAPC Position Statement** on End of Life and Palliative Care in the ICU
- **ICMR** — Definitions in limitation of treatment; Consensus guidelines on DNAR
- **Mental Healthcare Act 2017, Section 5** — advance directive for mental health treatment

The Twelve One-Liners — If You Read Nothing Else

1. Ageing is a failure to maintain homeostasis under physiological stress.
2. Chronological age is only a surrogate; gait speed predicts mortality better than hypertension.
3. Telomere shortening protects against uncontrolled division — at the expense of ageing.
4. Sirtuin modulation does not extend lifespan in mammals; it expands healthspan.
5. Plasma IL-6 rises with normal ageing and correlates inversely with functional ability.
6. In vaccine failure, antibody quantity is preserved but antibody quality is lost.
7. Older adults do not compensate after underfeeding — they never regain the lost weight.
8. Exercise is the only proven anti-ageing intervention; every geroprotector remains investigational.
9. Healthy ageing is defined by function, not by the absence of disease.
10. "Active" means continued participation — not merely being physically active.
11. Frailty is not a contraindication to robust exercise; it is the strongest indication for it.
12. Withdrawal of life-sustaining treatment is never withdrawal of care.

MD GERIATRIC MEDICINE — POSTGRADUATE EXAMINATION PREPARATION

Metabolic Bone Disease & Geriatric Rheumatology

High-yield 15-mark answers with Latest Updates (2023–present), Current Practice Update tables and Exam One-Liners

1. Osteoporosis — definition, pathogenesis, diagnosis and management including newer interventions; male and premenopausal osteoporosis
2. Predisposing factors, prevention and management of osteoporosis
3. DEXA scan
4. Antisclerostin antibodies in the treatment of osteoporosis
5. Paget's disease of bone
6. Osteoarthritis — pathogenesis, diagnosis and treatment
7. Temporal arteritis — diagnosis, clinical features, treatment and prognosis
8. Dermatomyositis in the elderly — presentation, comorbidities, investigations, treatment and prognosis
9. Diffuse idiopathic skeletal hyperostosis — presentation, investigations and treatment

---

Primary source: *Pathy's Principles and Practice of Geriatric Medicine*, 6th edition
Supplementary: *Kane's Essentials of Clinical Geriatrics* and current international & Indian guidelines
Indian conventional units used throughout

Q1. Osteoporosis — Definition, Pathogenesis, Diagnosis and Management including Newer Interventions; Male and Premenopausal Osteoporosis

A. Definition and Burden

1. Osteoporosis is a **systemic skeletal disease** in which bone mass is reduced and the **microarchitecture of bone tissue deteriorates**, so that bone becomes fragile and fractures easily.

2. The **WHO densitometric definition** uses the DXA T-score: **normal ≥ −1.0**, **osteopenia −1.0 to −2.5**, **osteoporosis ≤ −2.5**, and **severe (established) osteoporosis ≤ −2.5 with a fragility fracture**.

3. One in three women and one in five men over 50 will sustain an osteoporotic fracture. About **15% of women at age 80 have had a hip fracture**, and hip fracture incidence rises **exponentially with age**.

4. Typical osteoporotic fractures are **vertebral, hip, distal radius (Colles) and proximal humerus**. Hip fracture carries the greatest burden of mortality and disability-adjusted life years.

5. Indian context: LASI wave-1 data show a very high burden of low bone mass; osteoporosis presents roughly **a decade earlier** than in the West, vitamin D deficiency exceeds 70%, and average dietary calcium intake is often **under 400 mg/day** against an ICMR-NIN requirement of 1000 mg/day.

B. Pathogenesis

6. Bone is continuously remodelled in **bone multicellular units (BMUs)** — **osteoclasts** resorb, **osteoblasts** form, and **osteocytes** couple the two processes. Osteoporosis results when resorption exceeds formation in each BMU.

7. Oestrogen normally increases osteoclast apoptosis, suppresses **RANKL**, and increases **osteoprotegerin (OPG)**, the decoy receptor for RANKL. Menopausal **hypo-oestrogenism prolongs the resorption phase and shortens the formation phase**, giving rapid postmenopausal bone loss.

8. The **RANK–RANKL–OPG axis** is the final common pathway for osteoclast formation and survival — the target of **denosumab**.

9. The **Wnt/β-catenin pathway** drives osteoblastic bone formation. **Sclerostin**, secreted by osteocytes, blocks it at LRP5/6 — the target of **romosozumab**.

10. Age-related contributors: **secondary hyperparathyroidism** from vitamin D and calcium deficiency, decline of the **GH/IGF-1 axis**, **sarcopenia and immobility** (reduced mechanical loading), and **inflammaging** (IL-1, IL-6, TNF-α).

11. Trabecular bone (spine, femoral neck, distal radius) is lost earlier and faster; increasing **cortical porosity** dominates later hip fracture risk.

C. Risk Factors and Secondary Causes

12. Non-modifiable: age, female sex, **prior fragility fracture** (strongest single predictor), parental hip fracture, early or surgical menopause, low BMI, Asian or Caucasian ethnicity.

13. Modifiable: smoking, alcohol above 3 units/day, immobility, low calcium and vitamin D, low protein intake, falls.

14. Secondary osteoporosis: glucocorticoids (commonest drug cause), rheumatoid arthritis, hyperthyroidism, primary hyperparathyroidism, hypogonadism, type 1 and type 2 diabetes, coeliac disease and IBD, aromatase inhibitors, androgen deprivation therapy, anorexia nervosa, transplantation, COPD, HIV, CKD-MBD, myeloma, anticonvulsants and long-term PPIs.

D. Diagnosis

15. DXA of the lumbar spine and hip is the reference standard. Screen **women ≥65 and men ≥70**, and younger patients with risk factors. USPSTF 2024 reaffirmed screening of women ≥65 and younger postmenopausal women at increased risk.

16. FRAX integrates clinical risk factors with or without BMD to give the **10-year probability of a major osteoporotic fracture and of hip fracture**. It can be modulated by **TBS, glucocorticoid dose, falls history, type 2 diabetes and hip axis length**. An **India-specific FRAX** model is available.

17. Vertebral fracture assessment (VFA) or lateral spine X-ray is indicated for **height loss over 4 cm**, new kyphosis, or unexplained back pain — **two-thirds of vertebral fractures are clinically silent**.

18. Baseline laboratory screen to exclude secondary causes: serum **calcium (mg/dL)**, phosphate, **alkaline phosphatase**, **creatinine (mg/dL)**, **25(OH)D (ng/mL)**, PTH, TSH, complete blood count, serum protein electrophoresis, **testosterone (ng/dL) in men**, coeliac serology and 24-hour urinary calcium.

19. Bone turnover markers — **s-CTX** (resorption) and **P1NP** (formation) — are used at **3–6 months** to confirm adherence and response, not to make the diagnosis.

E. Management

20. Universal measures for everyone: calcium **800–1000 mg/day** (diet preferred), protein **≥1 g/kg/day**, vitamin D **800–1000 IU/day**, daily weight-bearing and resistance exercise, balance training, smoking and alcohol reduction, **falls prevention**, and medication review using **Beers/STOPP-START and STOPPFall**.

21. Risk-stratified pathway (Kanis/ESCEO): *low risk* → reassurance, lifestyle, consider MHT or a SERM; *high risk* → **oral or IV bisphosphonate, or denosumab**; *very high or imminent risk* → **anabolic agent first, followed by an antiresorptive**.

22. Bisphosphonates: alendronate 70 mg weekly, risedronate 35 mg weekly (gastro-resistant formulation improves adherence), **zoledronic acid 5 mg IV yearly**. Zoledronate is first-line after hip fracture, reducing both re-fracture and mortality.

23. Denosumab 60 mg SC 6-monthly — anti-RANKL, safe in renal impairment, but **must never be stopped abruptly**: discontinuation causes **rebound multiple vertebral fractures**, so always follow with a bisphosphonate for 1–2 years.

24. Anabolic agents: teriparatide 20 mcg SC daily (up to 24 months), **abaloparatide** 80 mcg SC daily (18 months), **romosozumab** 210 mg SC monthly (12 months). All are self-limited and **must be followed by an antiresorptive** or the gains are lost.

25. Other options: raloxifene (reduces vertebral fracture and breast cancer risk, increases VTE and hot flushes), menopausal hormone therapy in early postmenopause, and **local osteo-enhancement procedure (LOEP)** in selected patients at high hip fracture risk. Calcitonin is now largely obsolete.

26. Drug holiday: consider after **5 years of oral or 3 years of IV bisphosphonate** in low-risk patients, reviewing yearly; **continue treatment in high-risk patients** and after any incident fracture. **No holiday is permissible with denosumab.**

27. Fracture Liaison Services (FLS) and the IOF **"Capture the Fracture"** initiative systematically identify and treat every patient after a fragility fracture; orthogeriatric co-management improves hip fracture outcomes. The **treatment gap remains large worldwide, especially in the very old.**

F. Male Osteoporosis

28. Men account for about **one-third of hip fractures worldwide** and have **higher post-fracture mortality** (approaching 30% at one year) than women, yet are far less often investigated or treated.

29. Secondary causes are present in 40–60% of men — hypogonadism (including androgen deprivation therapy for prostate cancer), alcohol excess, glucocorticoids, smoking, hypercalciuria, myeloma, COPD, hyperthyroidism and malabsorption. A **secondary work-up is mandatory in every man**.

30. Use the **same T-score threshold of ≤ −2.5** for diagnosis; ISCD recommends a **male reference database** for men.

31. Testosterone replacement (target **300–400 ng/dL** in older men, lower than the young adult target) increases BMD in genuinely hypogonadal men but has **no fracture-reduction data** — it is not a substitute for a bisphosphonate. **Prostate carcinoma is an absolute contraindication**; monitor PSA and haematocrit.

32. Alendronate, risedronate, zoledronate, denosumab, teriparatide, abaloparatide and romosozumab are all **approved for men**.

G. Premenopausal Osteoporosis

33. In premenopausal women, use the **Z-score, not the T-score**. A **Z-score ≤ −2.0** is reported as "below the expected range for age". Diagnosis requires a **fragility fracture or an identified secondary cause** together with low bone mass.

34. Causes: anorexia nervosa and the female athlete triad, glucocorticoids, coeliac disease and IBD, hyperthyroidism, primary hyperparathyroidism, idiopathic hypercalciuria, **pregnancy- and lactation-associated osteoporosis**, and idiopathic disease.

35. Treat the underlying cause first — restore nutrition, weight and menses, correct calcium and vitamin D, and reduce glucocorticoid exposure. Reserve drugs for continuing fractures or ongoing bone loss.

36. Bisphosphonates cross the placenta and have a very long skeletal half-life — counsel about contraception. **Teriparatide is preferred** when a drug is needed in a woman who may become pregnant.

Latest Updates (2023–Present)

- **ASBMR/BHOF goal-directed treatment position statement (JBMR 2024)** introduced **treat-to-target** for osteoporosis: aim for a **T-score above −2.5**. For a baseline lumbar spine T-score of −3.0, the probability of reaching target was **55% with alendronate alone, 81% with romosozumab→alendronate, and 93% with romosozumab→denosumab**.
- **Sequence matters.** Anabolic-first followed by antiresorptive is superior to the reverse. Transitioning **from a bisphosphonate to teriparatide causes total hip BMD to fall for at least 12 months**, and **denosumab→teriparatide causes hip BMD to stay below baseline for 2 years**.
- **ACP Living Guideline on osteoporosis** (updated on a rolling basis): bisphosphonates for most patients; **short-course anabolic therapy followed by a bisphosphonate for those at very high risk** — older age, prior fracture, chronic steroid use.
- **NOGG 2024 UK guideline** and NICE TA464: **IV zoledronic acid should be first-line after hip fracture**, because it reduces re-fracture risk and mortality and has better adherence than oral bisphosphonates.
- **RACGP / Healthy Bones Australia 2024–2025 guideline**: formal endorsement of **osteoanabolic therapy as first-line in treatment-naive very-high-risk patients**; romosozumab for 12 months or abaloparatide for 18 months, each mandatorily followed by an antiresorptive.
- **ISCD 2023 Official Positions**: updated DXA reporting, follow-up BMD testing and TBS application; **TBS formally**

endorsed as a FRAX adjustment; **opportunistic CT** accepted for case-finding (**L1 attenuation <100 HU** suggests osteoporosis, **>150 HU** suggests normal bone).

- **VITAL trial** confirmed that vitamin D supplementation in vitamin D–replete populations does **not** reduce cancer or cardiovascular events; the skeletal indication (800–1000 IU/day with calcium in deficient older people) stands.
- **Indian developments:** ISBMR position statements on vitamin D and osteoporosis management, the **ICMR-NIN 2024 RDA raising adult calcium to 1000 mg/day**, and increasing availability of teriparatide biosimilars and denosumab biosimilars in the Indian market.

Current Practice Update

Aspect	Textbook position	Current evidence / guideline
First-line in very high risk	Antiresorptive commonly first; anabolic "may be considered"	**Anabolic-first, then antiresorptive** is preferred (ASBMR/BHOF 2024, ACP living guideline, RACGP 2024)
Treatment target	Fracture-risk category based	**Goal-directed: achieve T-score above −2.5** (ASBMR/BHOF 2024)
After hip fracture	FLS referral, oral bisphosphonate	**IV zoledronate first-line** — reduces re-fracture *and* mortality (NOGG 2024)
Bone quality assessment	BMD ± FRAX; TBS mentioned	**TBS formally endorsed (ISCD 2023)**; opportunistic CT for case-finding
Abaloparatide	Limited availability	Now approved across Europe and Australia; a mainstream anabolic
Vitamin D	800–1000 IU/day, broad benefits implied	Skeletal benefit only; **no cancer/CVD benefit (VITAL)**; avoid high intermittent bolus doses (increase falls)

Exam One-Liners

- **T-score for diagnosis; Z-score for premenopausal women, men under 50 and children.**
- **Prior fragility fracture is the single strongest predictor** — and risk is highest in the first 2 years ("imminent risk").
- **Never stop denosumab without bisphosphonate cover** — rebound vertebral fractures.
- **Anabolic first, antiresorptive after** — never the reverse in very-high-risk patients.
- **Zoledronate 5 mg IV yearly is first-line after hip fracture** (reduces mortality).
- **40–60% of men with osteoporosis have a secondary cause** — always investigate.
- **Testosterone raises BMD but has no fracture data** — it is not an osteoporosis drug.

Q2. Predisposing Factors, Prevention and Management of Osteoporosis

A. Predisposing Factors

1. Constitutional: advancing age, female sex, postmenopausal state, **prior fragility fracture** (doubles future risk), parental hip fracture, small frame, and Caucasian or Asian ethnicity.

2. Nutritional: low dietary calcium (Indian intake frequently under 400 mg/day against a requirement of 1000 mg/day), **vitamin D deficiency**, low protein intake, low BMI **below 20 kg/m²**, and undernutrition.

3. Lifestyle: physical inactivity and immobilisation, **smoking**, alcohol above 3 units/day, excess caffeine, and inadequate sun exposure.

4. Endocrine: early or surgical menopause, hypogonadism in men, **hyperthyroidism including over-replacement with levothyroxine**, primary hyperparathyroidism, Cushing's syndrome, type 1 and type 2 diabetes, and acromegaly.

5. Gastrointestinal and nutritional disease: coeliac disease, inflammatory bowel disease, bariatric surgery, chronic liver disease, pancreatic insufficiency and anorexia nervosa.

6. Drugs: glucocorticoids (the commonest secondary cause), aromatase inhibitors, androgen deprivation therapy, anticonvulsants (phenytoin, carbamazepine), long-term heparin, proton pump inhibitors, SSRIs, thiazolidinediones and loop diuretics.

7. Chronic disease: rheumatoid arthritis, CKD-MBD, multiple myeloma, COPD, HIV and antiretroviral therapy, post-transplant state, and immobility from stroke or Parkinson's disease.

8. Geriatric amplifiers: sarcopenia, frailty, falls, poor vision, orthostatic hypotension, cognitive impairment and polypharmacy — these convert low bone density into an actual fracture. **Osteoporosis breaks bones only when the patient falls.**

B. Prevention

9. Primordial prevention: peak bone mass is attained by about **age 30**; adequate childhood and adolescent calcium, protein, sunlight and impact exercise determine the "bone bank" available for later life.

10. Nutrition: calcium **1000–1200 mg/day** preferably from diet (milk, curd, paneer, ragi, sesame, green leafy vegetables, small fish with bones), protein **≥1 g/kg/day**, and vitamin D **800–1000 IU/day** with **15–30 minutes of sun exposure** without sunblock.

11. Exercise: progressive **resistance and weight-bearing impact exercise at least 3 days a week**, plus **balance training such as Tai Chi**. Swimming and cycling do not load bone. Long-term exercise training reduces falls, fractures, hospitalisation and mortality.

12. Lifestyle modification: stop smoking, restrict alcohol to ≤2 units/day, and maintain BMI above 20 kg/m².

13. Falls prevention: home hazard modification, adequate lighting, grab rails, correct footwear, vision and hearing correction, treatment of orthostatic hypotension, **deprescribing fall-risk-increasing drugs using STOPPFall**, vitamin D correction, and hip protectors in selected institutionalised patients.

14. Case-finding and screening: DXA in women ≥65 and men ≥70, earlier with risk factors; **FRAX-based intervention thresholds** to decide who to treat. The **SCOOP trial** showed that community FRAX-based screening of older women **reduced hip fractures**.

15. Secondary prevention: actively identify and treat secondary causes. In **glucocorticoid therapy**, start bone protection with any dose in patients ≥70 or with prior fracture, and at ≥7.5 mg prednisolone daily for ≥3 months in others.

16. Tertiary prevention: Fracture Liaison Service after every fragility fracture, orthogeriatric care of hip fracture, early mobilisation, and rehabilitation to prevent post-fracture disability and institutionalisation.

C. Management (Rapid Recall)

17. Foundation for all: calcium, vitamin D, protein, exercise, falls prevention and medication review — continued even after drug therapy starts.

18. High risk: oral bisphosphonate (alendronate/risedronate), **IV zoledronate 5 mg yearly**, or **denosumab 60 mg 6-monthly**.

19. Very high or imminent risk: romosozumab, teriparatide or abaloparatide first, then an antiresorptive to consolidate the gain.

20. Special situations: denosumab or teriparatide in CKD (avoid bisphosphonates if eGFR <30–35 mL/min); raloxifene or MHT in younger postmenopausal women; vertebral augmentation only for refractory painful acute vertebral fracture.

21. Monitoring: DXA at 1–2 years on the **same machine**, interpreting change only if it exceeds the **least significant change** (about 3% spine, 5% hip); **CTX or P1NP at 3–6 months** for adherence; **annual height measurement** as a simple marker of new vertebral fracture.

22. Adherence is poor — under 50% at one year. Use gastro-resistant or less frequent formulations, patient education, group education programmes and reminder systems.

23. Indian service delivery: osteoporosis care is delivered through **NPHCE** geriatric clinics and district hospitals; hip fracture surgery is covered under **AB-PMJAY**; assistive devices come through **ADIP and Rashtriya Vayoshri Yojana**; and calcium/vitamin D are widely available in the essential medicines list.

Latest Updates (2023–Present)

- **SCOOP trial** remains the key evidence that **community-based FRAX screening reduces hip fractures** without increasing anxiety or overall costs; it underpins the case-finding approach in current guidelines.
- **NOGG 2024** restated intervention thresholds by FRAX category and confirmed **IV zoledronate as first-line after hip fracture**.
- **USPSTF 2024** reaffirmed DXA screening for women ≥65 and postmenopausal women under 65 at increased risk; evidence remains insufficient for routine screening of men.
- **ICMR-NIN 2024** raised the Indian RDA for calcium in adults to **1000 mg/day** (1200 mg in the elderly) and reiterated vitamin D sufficiency targets.
- **Exercise evidence strengthened:** meta-analyses confirm that long-term exercise training reduces falls, fractures, hospitalisations and mortality; **combined resistance plus balance training** is the most effective prescription in older adults.
- **Deprescribing as fracture prevention:** STOPPFall and the 2023 Beers update emphasise reducing benzodiazepines, Z-drugs, antipsychotics, anticholinergics and orthostatic agents as a direct fracture-prevention strategy.

Current Practice Update

Aspect	Textbook position	Current evidence / guideline
Who to treat	T-score ≤ −2.5 or fragility fracture	**FRAX probability–based intervention thresholds**, with T-score as only one input
Screening strategy	Opportunistic case-finding	**Systematic community FRAX screening reduces hip fractures** (SCOOP)
Calcium target	800–1000 mg/day	**1000–1200 mg/day**, diet-first (ICMR-NIN 2024 for India)
Falls prevention	General advice	**Structured multifactorial programmes with formal deprescribing (STOPPFall)** as core fracture prevention
Post-fracture care	FLS recommended	FLS **plus** orthogeriatric co-management and mandatory bone protection within 120 days (NHFD KPI-7)

Exam One-Liners

- **Prevention has four levels:** primordial (peak bone mass), primary (calcium/vitamin D/exercise), secondary (treat secondary causes and glucocorticoid prophylaxis), tertiary (FLS after fracture).
- **Glucocorticoids are the commonest drug cause** — bone protection from day one in the old or previously fractured.
- **Swimming and cycling do not build bone** — impact and resistance exercise do.
- **The SCOOP trial** = community FRAX screening reduces hip fractures.
- **Height loss over 4 cm** = look for a silent vertebral fracture.

Q3. DEXA Scan

*(Note: the internationally correct abbreviation is **DXA**, not DEXA — formalised in the ISCD 2023 Official Positions.)*

A. Principle

1. DXA (dual-energy X-ray absorptiometry) passes **two X-ray beams of different photon energies** through the region of interest. Bone and soft tissue attenuate the two energies differently, allowing the machine to subtract soft tissue and compute **bone mineral content (g)** and **areal bone mineral density (g/cm²)**.

2. It is a **two-dimensional areal measurement**, so it is influenced by bone size — **small bones read falsely low** and **large bones falsely high**. It does not measure true volumetric density.

3. Radiation dose is very low (roughly 1–10 microsievert per site), less than one day of natural background radiation and far less than a chest radiograph. Scanning takes 10–20 minutes.

B. Reporting

4. T-score = the number of standard deviations from the **mean peak bone mass of a young healthy adult of the same sex**. Used in **postmenopausal women and men aged ≥50**.

5. Z-score = standard deviations from the **mean for age, sex and ethnicity-matched controls**. Used in **premenopausal women, men under 50, and children**. A Z-score **≤ −2.0** is reported as "below the expected range for age".

6. WHO categories (T-score): normal **≥ −1.0**; **osteopenia/low bone mass −1.0 to −2.5**; **osteoporosis ≤ −2.5**; **severe or established osteoporosis ≤ −2.5 plus a fragility fracture**.

7. Standard sites: PA lumbar spine (L1–L4) and **proximal femur (femoral neck and total hip)**. The **lowest T-score of the valid sites** is used for diagnosis.

8. The **33% (one-third) radius** is the accepted third site — used when spine and hip are unmeasurable or invalid: weight above table limit, bilateral hip replacement, or **hyperparathyroidism** (cortical bone is preferentially lost).

9. Vertebral exclusion rules: exclude vertebrae that are fractured, have focal structural change, or differ by **more than 1.0 T-score** from adjacent vertebrae; **at least two evaluable vertebrae are required**, otherwise the spine is not reportable.

C. Indications

10. Women **≥65** and men **≥70**; younger postmenopausal women and men aged 50–69 with risk factors; **any adult with a fragility fracture**; glucocorticoid therapy or any disease or drug associated with bone loss; radiographic osteopenia or vertebral deformity; and monitoring of osteoporosis treatment.

D. What Else DXA Can Provide

11. Vertebral fracture assessment (VFA) — lateral spine imaging at the same sitting at very low dose, detecting **clinically silent vertebral fractures** that immediately upgrade the patient to treatment.

12. Trabecular bone score (TBS) — grey-level texture analysis of the lumbar spine DXA image reflecting **trabecular microarchitecture**. It predicts fracture **independently of BMD** and can adjust FRAX probability.

13. Whole-body DXA measures **appendicular lean mass**, used to confirm low muscle mass in the diagnosis of **sarcopenia** (EWGSOP2, AWGS 2019).

14. Hip structural analysis and hip axis length add geometric information to fracture prediction; DXA also gives **body composition** and can screen for **abdominal aortic calcification**.

E. Pitfalls, Especially in the Elderly

15. Degenerative change falsely raises lumbar spine BMD — osteophytes, facet joint sclerosis, vertebral compression fracture, scoliosis and **aortic calcification**. In older people the **hip is the more reliable site**.

16. Artefacts: recent oral or intravenous contrast, radionuclide studies, calcium tablets in the gut, surgical clips, spinal instrumentation, vertebroplasty cement, coins or metal in clothing, and previous fractures.

17. Technical/patient factors: poor positioning, severe kyphosis or inability to lie flat, tremor or movement, obesity beyond the table limit, and inconsistent analysis between operators.

18. DXA **cannot distinguish osteoporosis from osteomalacia** — both show low BMD. Interpretation must always be with biochemistry.

F. Monitoring

19. Repeat DXA on **the same machine, with the same software and technologist**, and compare the **absolute BMD in g/cm², not T-scores**.

20. Change is meaningful only if it exceeds the **least significant change (LSC)**, which each centre must calculate — typically about **3% at the lumbar spine and 5% at the hip**.

21. Usual interval is **1–2 years on treatment**, longer (2–5 years) if stable or untreated. ISCD 2023 emphasises that the **clinical context prompting the repeat** should drive the interval rather than a fixed rule.

Latest Updates (2023–Present)

- **ISCD 2023 Official Positions** (9th Position Development Conference, Northbrook): new positions on **DXA reporting, follow-up BMD testing, bilateral hip reporting and TBS application**, and the formal statement that the correct term is **"DXA — not DEXA"**.
- **TBS: 2023 ISCD Official Positions** — use **L1–L4 without exclusions** for TBS; TBS is most likely to change management in patients **aged ≥40 who sit close to the pharmacological intervention threshold**; TBS **version 4.0** now corrects directly for soft tissue thickness, improving accuracy in obesity.
- **Opportunistic CT screening:** an **L1 attenuation below 100 Hounsfield Units** suggests osteoporosis and **above 150 HU** suggests normal bone density — usable to trigger formal bone health assessment, provided machine-specific cut-offs and scanner stability are validated.
- **QCT equivalence:** femoral neck and total hip T-scores derived from **2D projections of QCT data are now accepted as equivalent to DXA T-scores** for WHO diagnosis; spinal trabecular QCT predicts vertebral fracture at least as well as AP spine DXA in postmenopausal women.
- **REMS (radiofrequency echographic multi-spectrometry)** — a radiation-free ultrasound-based method giving spine and hip BMD, of particular interest for **resource-limited and rural Indian settings** and for pregnancy, though not yet a WHO-endorsed diagnostic standard.
- **USPSTF 2024** reaffirmed DXA screening for women ≥65 and younger postmenopausal women at increased risk; evidence remains insufficient for men.
- **Artificial intelligence** is increasingly applied to automated DXA analysis and to **opportunistic detection of vertebral fractures on routine chest X-rays and CT scans**.

Current Practice Update

Aspect	Textbook position	Current evidence / guideline
Terminology	"DEXA" used interchangeably	**DXA is the correct term** (ISCD 2023)
Diagnostic anchor	T-score ≤ −2.5 alone	**BMD combined with FRAX ± TBS, VFA and fracture history**; most fractures occur in people with a T-score better than −2.5
Microarchitecture	Not routinely assessed	**TBS formally endorsed** as a FRAX adjustment (ISCD 2023)
Alternative modalities	DXA only	**QCT-derived hip T-scores equivalent**; **opportunistic CT (L1 <100 HU)** and **REMS** now recognised
Repeat interval	Fixed 1–2 years	**Clinical context–driven**, interpreted against the centre's own LSC (ISCD 2023)

Exam One-Liners

- **DXA measures areal, not volumetric, density** — small bones read falsely low.
- **Lowest T-score of spine, femoral neck or total hip** makes the diagnosis.
- **Degenerative disease and aortic calcification falsely raise spine BMD** — trust the hip in the elderly.
- **Need at least two evaluable vertebrae**; exclude any differing by more than 1.0 T-score.
- **Compare g/cm², not T-scores**, when monitoring; change must exceed the LSC.
- **Z-score, not T-score, in premenopausal women and men under 50.**

Q4. Antisclerostin Antibodies in the Treatment of Osteoporosis

A. The Biology of Sclerostin

1. Sclerostin is a glycoprotein encoded by the **SOST gene** and secreted almost exclusively by **osteocytes** — the mechanosensing cells buried within bone.

2. It binds the **LRP5/6 co-receptors** and thereby **blocks canonical Wnt/β-catenin signalling**, the master pathway for osteoblast differentiation and bone formation. Sclerostin is therefore a **physiological brake on bone formation**.

3. Sclerostin also **increases RANKL and reduces osteoprotegerin**, so blocking it reduces bone resorption as well as increasing formation.

4. Human proof of concept: loss-of-function mutations in *SOST* cause **sclerosteosis** and **van Buchem disease** — conditions of very high bone mass with strong, fracture-resistant bone, which established sclerostin as a drug target.

5. Mechanical loading and PTH suppress sclerostin; **immobilisation, glucocorticoids and unloading increase it** — this explains disuse osteoporosis at a molecular level.

B. The Drug — Romosozumab

6. Romosozumab is a **humanised IgG2 monoclonal antibody against sclerostin** and is currently the only anti-sclerostin agent in clinical use for osteoporosis.

7. It has a **unique "dual effect": it increases bone formation and simultaneously decreases bone resorption**. This is different from teriparatide and abaloparatide, which increase both formation and resorption.

8. Dose: 210 mg subcutaneously once monthly (given as two 105 mg injections) for 12 months only. The anabolic window is **self-limited** — P1NP rises sharply then wanes by 9–12 months, while CTX stays suppressed.

9. It **must be followed by an antiresorptive** (a bisphosphonate or denosumab); otherwise the BMD gain is rapidly lost.

10. BMD gains are the **largest of any currently available agent** — roughly **13% at the lumbar spine and 6% at the total hip over 12 months**, exceeding alendronate and teriparatide.

C. Trial Evidence

11. FRAME (postmenopausal osteoporosis): romosozumab versus placebo for 12 months, then denosumab in both arms — **73% reduction in new vertebral fractures at 12 months**, with benefit maintained at 24 months.

12. ARCH (high fracture risk): romosozumab for 12 months then alendronate, versus alendronate throughout — **48% fewer new vertebral fractures, 27% fewer clinical fractures and 38% fewer hip fractures at 24 months**. This trial also generated the cardiovascular safety signal.

13. STRUCTURE: in women already treated with bisphosphonates, romosozumab produced **greater hip and spine BMD gains than teriparatide** — relevant because prior antiresorptive use blunts teriparatide's hip response.

14. BRIDGE: demonstrated efficacy in **men with osteoporosis**.

D. Safety

15. Boxed warning — cardiovascular risk. ARCH showed an **excess of major adverse cardiovascular events** (myocardial infarction, stroke, cardiovascular death) in the romosozumab arm. It is **contraindicated within 12 months of a myocardial infarction or stroke**, and cardiovascular risk must be assessed before starting.

16. Hypocalcaemia — correct calcium and vitamin D before starting and supplement throughout; use with caution in **CKD**, where the risk is higher.

17. Other adverse effects: injection-site reactions, arthralgia, headache, hypersensitivity and, rarely, **osteonecrosis of the jaw and atypical femoral fracture** (class effects of potent antiresorptive activity). A dental review before starting is prudent.

E. Place in Therapy

18. Indicated for **postmenopausal women (and men) at very high or imminent fracture risk** — a recent fracture, multiple vertebral fractures, a very low T-score, fractures occurring while on antiresorptive therapy, or high-dose glucocorticoid-induced osteoporosis.

19. Anabolic-first sequencing gives the best outcome. Starting with romosozumab and following with denosumab produces the **highest probability of reaching a target T-score above −2.5**; the reverse sequence (antiresorptive first) blunts the anabolic response, particularly at the hip.

20. In the Indian context, cost and availability remain the principal barriers. **Teriparatide (including biosimilars) is the more accessible anabolic agent**, with romosozumab reserved for selected very-high-risk patients in tertiary centres.

Latest Updates (2023–Present)

- **ASBMR/BHOF goal-directed treatment statement (JBMR 2024)**: for a baseline lumbar spine T-score of **−3.0**, the probability of achieving a T-score above −2.5 was **55% with alendronate alone, 81% with romosozumab→alendronate and 93% with romosozumab→denosumab**. For a starting T-score of **−3.5**, the corresponding figures were **11%, 46% and 67%** — a compelling case for anabolic-first therapy in severe disease.

- **First-line approval in treatment-naive patients:** the **Australian PBS (2025)** now funds romosozumab for 12 months (and abaloparatide for 18 months) in **high-risk patients with no prior antiresorptive or anabolic exposure** — a formal shift away from "use only after antiresorptive failure".
- **ACP Living Guideline and RACGP/Healthy Bones Australia 2024** both endorse **short-course anabolic therapy followed by a bisphosphonate** for very high fracture risk, explicitly naming romosozumab.
- **Transition data**: moving **from a bisphosphonate to romosozumab still improves total hip BMD and hip strength**, although less markedly than in treatment-naive patients — unlike teriparatide, where hip BMD actually falls after prior antiresorptive use. This makes romosozumab the **preferred anabolic after antiresorptive therapy**.
- **Cardiovascular safety**: accumulating real-world and post-marketing data have been broadly reassuring, but the **boxed warning stands** and pre-treatment cardiovascular risk assessment is now routine practice.
- **Setrusumab (BPS-804)**, a second anti-sclerostin antibody, is in **phase 3 development for osteogenesis imperfecta** (ORBIT/COSMIC trials), not for age-related osteoporosis — a useful point of distinction in vivas.

Current Practice Update

Aspect	Textbook position	Current evidence / guideline
Position of romosozumab	Listed as an anabolic option for very high risk	**Preferred first-line anabolic** in treatment-naive very-high-risk patients (RACGP 2024, PBS 2025)
Mechanism framing	Grouped with anabolic agents	Emphasis on its **unique dual action** — increases formation *and* decreases resorption
Sequencing	"Anabolic followed by antiresorptive"	Quantified: **romosozumab→denosumab gives the highest probability of reaching a T-score target** (ASBMR/BHOF 2024)
Anabolic after prior bisphosphonate	Not differentiated	**Romosozumab preferred over teriparatide** — teriparatide causes transient hip BMD loss (STRUCTURE)
Cardiovascular safety	Not emphasised	**Boxed warning**; contraindicated within 12 months of MI or stroke

Exam One-Liners

- **Sclerostin is made by osteocytes, blocks Wnt at LRP5/6, and inhibits bone formation.**
- **Sclerosteosis and van Buchem disease** = natural human knockouts of *SOST*, with high bone mass.
- **Romosozumab is the only anti-sclerostin drug in clinical use** — 210 mg SC monthly for **12 months only**.
- **Dual effect:** bone formation up, bone resorption down.
- **FRAME** = 73% fewer vertebral fractures; **ARCH** = superior to alendronate but with the **cardiovascular signal**.
- **Always follow with an antiresorptive**, or the gain is lost.
- **Contraindicated within 12 months of MI or stroke.**

Q5. Paget's Disease of Bone

A. Definition and Epidemiology

1. Paget's disease of bone (**osteitis deformans**) is a **focal disorder of greatly accelerated and disorganised bone remodelling**, affecting one bone (**monostotic**) or several (**polyostotic**) — but never the entire skeleton, which distinguishes it from metabolic bone disease.

2. It is **rare before the age of 40** and its prevalence **rises steeply with age** (about 1–3% of people over 55 in the UK), with a **male predominance**.

3. Geographically it is common in the **United Kingdom, Western Europe, Australia and North America** and **distinctly rare in India, Asia, Scandinavia and Africa** — but it does occur in Indians and should not be dismissed.

4. Both **prevalence and severity have fallen worldwide** over recent decades, which strongly suggests a **waning environmental trigger**.

B. Aetiology and Pathogenesis

5. Genetic: mutations in **SQSTM1 (p62)**, particularly **P392L**, are found in **40–50% of familial** and **5–10% of sporadic** cases, inherited in an **autosomal dominant** pattern with incomplete penetrance. Other genes include *OPTN*, *TNFRSF11A* and *VCP*.

6. Environmental: paramyxovirus (measles, canine distemper, respiratory syncytial virus) nuclear inclusions have been described in pagetic osteoclasts but remain **unproven**. Other proposed factors are low dietary calcium in youth, repetitive mechanical loading, and environmental toxins.

7. The **primary abnormality is in the osteoclast** — pagetic osteoclasts are **large, multinucleated, hyperactive and hypersensitive to vitamin D and RANKL**, producing intense focal resorption.

8. This is followed by **frantic, disorganised osteoblastic repair**, laying down **structurally abnormal woven bone** with a **"mosaic" pattern of irregular cement lines**, marked **hypervascularity** and **marrow fibrosis**. The bone is bigger and denser but **mechanically weak**.

9. Three phases: (i) **osteolytic** — resorption predominates; (ii) **mixed osteolytic–osteoblastic**; (iii) **osteosclerotic or "burnt-out"** — dense sclerotic bone with low turnover.

C. Clinical Features

10. Up to 70–80% are asymptomatic, detected incidentally through an **unexplained isolated rise in alkaline phosphatase** or an X-ray performed for another reason.

11. Commonest sites in descending order: pelvis > lumbar spine > femur > thoracic spine > sacrum > skull > tibia > humerus.

12. Bone pain is the commonest symptom — deep, boring, **worse at rest and at night**, unlike mechanical pain; the overlying skin may be **warm** because of hypervascularity.

13. Deformity: anterolateral **bowing of the tibia ("sabre tibia")** and femur, kyphosis, **frontal bossing with increasing hat size**, and **secondary osteoarthritis** of the hip and knee from altered joint mechanics — often the actual cause of pain.

14. Skull involvement: deafness (both **conductive** from ossicular involvement and **sensorineural** from cochlear capsule and VIIIth nerve compression) — Paget's disease is a listed cause of hearing loss in the elderly and should be evaluated radiologically and with alkaline phosphatase. Also tinnitus, headache, **platybasia and basilar invagination** leading to hydrocephalus and brainstem compression.

15. Spinal involvement: back pain, **spinal stenosis**, radiculopathy, and rarely cord ischaemia from a vascular "steal" phenomenon.

16. Complications: pathological and fissure fractures, **high-output cardiac failure** (extensive disease, now rare), **hypercalcaemia of immobilisation**, hypercalciuria and renal stones, and **osteosarcoma in under 1%** — suspect this if there is **new severe pain, a soft-tissue mass, or a sudden further rise in ALP**; the prognosis is dismal.

D. Investigations

17. Serum total alkaline phosphatase is the key screening and monitoring test, raised roughly in proportion to disease activity and extent. **Calcium, phosphate and PTH are characteristically normal.**

18. Biochemical differentiation of metabolic bone disease:

Disease	Calcium	Phosphate	Alkaline phosphatase	PTH
Paget's disease	Normal / high	Normal / high	**High**	Normal
Osteoporosis	Normal	Normal	Normal	Normal / high
Osteomalacia	Low / normal	**Low**	High / normal	**High**
Primary hyperparathyroidism	**High**	Low / normal	High / normal	**High**
Hyperthyroidism	High	High	High / normal	Low

19. If ALP is normal or liver disease coexists, use **bone-specific ALP or P1NP**; **urinary NTX/CTX** reflect resorption. Any older person with a **borderline low calcium and a raised ALP** should also be assessed for **vitamin D deficiency**.

20. Plain radiography is diagnostic — cortical thickening, coarse trabeculae, bone expansion, and mixed lysis with sclerosis. Classic signs: **"osteoporosis circumscripta"** of the skull, **"cotton-wool" skull**, the **"blade of grass" or "flame-shaped" lytic front** advancing along a long bone, **"picture-frame" vertebra**, **"ivory" vertebra**, and **pseudofractures on the convex (tension) surface**.

21. Radionuclide bone scan (Tc-99m MDP) is the **best test to map the full extent of disease** — intense uniform uptake in affected bones. It is **more sensitive but less specific** than X-ray, so every hot spot needs radiographic confirmation.

22. CT and MRI are reserved for suspected complications — spinal stenosis, basilar invagination, or **sarcomatous transformation**.

E. Management

23. Asymptomatic patients with limited disease at low-risk sites may simply be observed with periodic alkaline phosphatase measurement.

24. Indications for bisphosphonate therapy: bone pain, **before elective surgery at a pagetic site** (to reduce hypervascularity and blood loss), **hypercalcaemia of immobilisation**, and **disease at high-risk sites** — skull, spine, weight-bearing long bones and bone adjacent to major joints.

25. Zoledronic acid 5 mg as a single intravenous infusion is the treatment of choice. It normalises ALP in most patients and produces **remission lasting several years**, and is more effective than pamidronate or risedronate at normalising ALP.

26. Alternatives: risedronate 30 mg daily for 2 months, alendronate 40 mg daily for 6 months, or IV pamidronate. Give **calcium and vitamin D before and after** to prevent hypocalcaemia, and warn about the **transient flu-like acute-phase reaction** (about 40% after the first infusion, much milder subsequently).

27. Calcitonin is now second-line, reserved for patients in whom bisphosphonates are contraindicated. **Denosumab** has been used off-label in selected refractory cases.

28. Adjunctive measures: analgesics and NSAIDs for pain, **walking aids and shoe raises** for deformity and leg-length discrepancy, **hearing aids and audiological rehabilitation**, physiotherapy, and falls prevention.

29. Surgery for fracture fixation, **joint replacement in secondary osteoarthritis**, osteotomy for deformity, and decompression for spinal stenosis or basilar invagination. **Pre-treat with a bisphosphonate to reduce operative bleeding.**

30. Monitoring: ALP at 3–6 months, then 6–12 monthly. Retreat if pain recurs or ALP rises, but **not within 12 months** of neridronate or zoledronate.

Latest Updates (2023–Present)

- **PRISM and PRISM-EZ (long-term randomised trials)**: **intensive bisphosphonate therapy aimed at normalising alkaline phosphatase gave no advantage** over symptom-driven treatment for fractures, orthopaedic procedures, quality of life or hearing loss. This is the single most important change in modern practice — the goal is **symptom control, not biochemical normalisation**, in established disease.
- **ZiPP randomised trial (Ann Rheum Dis 2024; NIHR 2024)**: prophylactic zoledronic acid in **asymptomatic SQSTM1 mutation carriers** reduced the development of new bone lesions on imaging but did **not** improve clinically significant outcomes. **Genetic testing with pre-emptive treatment is therefore not routine practice.**
- **SIOMMMS 2024 (Italian) guideline**: radiograph the symptomatic site first, then use a **radionuclide bone scan to define extent**; favour **intravenous bisphosphonates, especially zoledronic acid, for retreatment**; retreat only after at least 12 months; use analgesics, NSAIDs or neuropathic agents when antiresorptives fail to control pain; and **re-image any site with worsening pain** to exclude fracture, osteoarthritis or neoplastic degeneration.
- **Endocrine Society / JBMR clinical guideline** reaffirms a **single 5 mg zoledronate infusion as the standard of care** and recommends radionuclide bone scanning for extent assessment.
- **Epidemiological trend:** the continuing decline in incidence and severity of Paget's disease worldwide reinforces the environmental-trigger hypothesis and means the condition is now often seen in a milder, monostotic form.

Current Practice Update

Aspect	Textbook position	Current evidence / guideline
Treatment goal	Suppress bone turnover, normalise ALP	**Symptom control**; biochemical normalisation does not improve outcomes (**PRISM-EZ**)
Drug of choice	Bisphosphonates generally; calcitonin listed as an option	**Single 5 mg IV zoledronate** is standard first-line; **calcitonin second-line only**
Assessing extent	Plain radiography	**Radionuclide bone scan for extent**, X-ray for confirmation of each hot spot
Genetic testing	Not discussed	**SQSTM1 testing available**, but prophylactic treatment of carriers is not justified (**ZiPP 2024**)
Retreatment	Not specified	Only if pain or ALP recurs, and **not within 12 months** of zoledronate/neridronate (SIOMMMS 2024)

Exam One-Liners

- **Focal, never generalised** — that separates Paget's from metabolic bone disease.
- **Biochemistry: high ALP with normal calcium, phosphate and PTH.**
- **The osteoclast is the primary abnormal cell**; SQSTM1/p62 is the key gene.
- **X-ray for diagnosis; bone scan for extent.**
- **"Blade of grass" lytic front, "cotton-wool" skull, "picture-frame" vertebra, sabre tibia.**
- **Single 5 mg IV zoledronate is the treatment of choice.**
- **New severe pain + soft-tissue mass + rising ALP = osteosarcoma until proved otherwise.**
- **PRISM-EZ: treat symptoms, not the alkaline phosphatase.**

Q6. Osteoarthritis — Pathogenesis, Diagnosis and Treatment

A. Definition and Epidemiology

1. Osteoarthritis is a **whole-joint disease** characterised by **focal hyaline cartilage destruction with joint space loss, subchondral sclerosis and osteophyte formation**, together with synovial hypertrophy, capsular and ligamentous change and peri-articular muscle weakness.

2. It is the **most common joint disease among older adults** and the **major cause of knee, hip and back pain**. Almost **60% of Americans aged 65 and over report arthritis or chronic joint symptoms**, and musculoskeletal disorders are the **commonest cause of disability in old age**.

3. In India, symptomatic knee osteoarthritis affects roughly **25–30% of adults over 60**, with a female preponderance; **deep squatting, floor-sitting, stair use in unlifted buildings and manual labour** are important local contributors.

4. OA is **not by definition inflammatory**, although **synovial hypertrophy and joint effusions are typical** — a key distinction from rheumatoid arthritis.

B. Risk Factors

5. Systemic: age, **female sex** (especially post-menopause), **obesity**, genetic predisposition, and **metabolic syndrome** (the "metabolic OA" phenotype).

6. Local and mechanical: previous joint injury or surgery (meniscectomy), **malalignment** (varus knee), joint dysplasia, occupational kneeling, squatting and lifting, **quadriceps weakness**, and **proprioceptive loss**.

C. Pathogenesis

7. OA is **not simply "wear and tear"**. Progression revolves around a **complex interplay of chondrocyte dysregulation of the extracellular matrix, genetic influences, local mechanical factors and inflammation**.

8. Abnormal mechanical loading activates chondrocytes to release **matrix metalloproteinases (especially MMP-13)** and **aggrecanases (ADAMTS-4 and -5)**, which degrade **type II collagen and aggrecan faster than they can be resynthesised**.

9. IL-1β, TNF-α, IL-6, nitric oxide and prostaglandin E2 from chondrocytes and synovium amplify catabolism; **adipokines such as leptin** link obesity to cartilage damage **beyond simple mechanical load**.

10. Subchondral bone shows increased turnover, **sclerosis, bone marrow lesions and cysts**; **osteophytes** form at the joint margin by endochondral ossification, representing an attempted repair response.

11. Pain in established OA is **mixed** — nociceptive from subchondral bone, synovium, capsule and entheses (cartilage itself is aneural), **plus peripheral and central sensitisation**. This explains the **poor correlation between radiographic severity and symptoms**.

12. A **vicious cycle operates in older people**: joint pain → activity avoidance → muscle weakness and joint instability → further injury and pain → loss of cardiorespiratory fitness → **an unfit, inactive, arthritic person with compromised independence**.

D. Clinical Features and Diagnosis

13. Diagnosis is clinical: activity-related joint pain, **morning stiffness lasting minutes rather than hours**, gelling after inactivity, crepitus, restricted range of movement, bony enlargement, and minimal warmth or erythema.

14. Distinguishing OA from inflammatory arthritis:

Clinical feature	Osteoarthritis	Inflammatory arthritis
Duration of stiffness	**Minutes**	**Hours**
Pain	Usually with activity	Occurs even at rest and at night
Fatigue	Unusual	Common
Swelling	Common, little synovial reaction	Very common, with synovial proliferation and thickening
Erythema and warmth	Unusual	Common

15. Typical joints: knees, hips, first carpometacarpal joint, **distal interphalangeal (Heberden's nodes)** and **proximal interphalangeal (Bouchard's nodes)**, first metatarsophalangeal joint (**hallux rigidus**), midfoot, and cervical and lumbar facet joints.

16. Foot OA affects one in six people over 50, with two phenotypes: **isolated first MTP joint OA (hallux rigidus)** — localised pain, stiffness and a dorsal exostosis — and a **polyarticular midfoot form** presenting as dorsal foot pain worse on prolonged standing and walking on uneven ground.

17. Plain radiography remains the main modality — **joint space narrowing, subchondral sclerosis, osteophytes and subchondral cysts** (Kellgren–Lawrence grades 0–4). **MRI and ultrasound are more accurate and comprehensive** measures of joint change but are rarely needed for diagnosis.

18. Blood tests are normal. ESR, CRP, rheumatoid factor and anti-CCP are used only to exclude alternatives. **Aspirate the joint** if there is an effusion with warmth, to exclude **sepsis or crystal arthropathy** — remember that gout and pseudogout commonly coexist with OA in the elderly.

E. Treatment

19. Non-pharmacological management should be the focus of intervention: patient education and self-management, **weight loss**, **land-based and aquatic exercise**, **quadriceps strengthening**, **daily range-of-motion exercise**, local ice and heat, and acupuncture. Group exercise improves poor compliance by adding a social component.

20. Assistive devices and biomechanics: a **cane held in the contralateral hand** reduces load on an arthritic hip; **cane length should equal the distance from the wrist crease to the floor**, and **stick rubbers must be checked and replaced** to avoid falls. For knee pain the patient should experiment with the cane in either hand. **Moulded splints** protect arthritic hand joints; **foot orthoses and shoe-stiffening inserts** are effective for first MTP and midfoot OA.

21. Time analgesia before activity — OA pain is exacerbated by exercise, so medication is best given beforehand rather than afterwards.

22. Pharmacological therapy is symptomatic: topical NSAIDs first for knee and hand OA in older adults (best benefit-to-risk ratio); paracetamol has modest effect; **oral NSAIDs at the lowest dose for the shortest duration**, with PPI cover and careful attention to renal, cardiac, GI and Beers-criteria risk.

23. Intra-articular corticosteroid gives short-term relief of a flare (weeks). **Viscosupplementation with hyaluronic acid and nutraceuticals (glucosamine, chondroitin) have not shown significant pain benefit** beyond placebo, and **vitamin D neither reduces pain nor repairs structural damage**.

24. Duloxetine is useful where central sensitisation dominates. **Avoid long-term opioids** in older adults. Refractory pain is best managed by a **multidisciplinary pain team** with anaesthetic, psychological and physiotherapy input.

25. Surgery: total knee or hip arthroplasty is reserved for severe symptomatic disease failing conservative treatment; patients should be **medically stable, encouraged to lose weight and strengthen relevant muscles before operation**. **Arthroscopic lavage and debridement have little evidence of efficacy** and are not recommended for degenerative knee disease.

26. Optimal management usually requires **several modalities combined**, and the best combination **varies from patient to patient** — a genuinely individualised, function-focused approach.

Latest Updates (2023–Present)

- **STEP 9 trial (NEJM 2024)** — 68 weeks, 407 participants with obesity and moderate knee OA: **semaglutide 2.4 mg weekly** produced a mean weight change of **−13.7% versus −3.2%** on placebo, a **WOMAC pain reduction of 41.7 versus 27.5 points**, and significantly better physical function and SF-36 scores. Both arms received diet and exercise counselling.
- A **2025 network meta-analysis** established a therapeutic threshold of about **7–10% weight loss** as necessary for meaningful symptom improvement in knee OA, and **weight loss of ≥7.5% is associated with roughly a 31% lower risk of knee replacement**.
- **GLP-1 receptor agonists are not yet on-label for osteoarthritis** and do not appear in the current OARSI or ACR guidelines; ACR Convergence 2025 sessions emphasised that current data show **symptom improvement, not proven structural joint repair**.
- **NICE NG226 (current UK guideline)**: diagnose knee OA **clinically without imaging** in people aged 45 or over with activity-related pain and either no morning stiffness or stiffness lasting under 30 minutes; offer **therapeutic exercise and weight management as core treatment**; **use topical NSAIDs before oral NSAIDs**; and **do not offer glucosamine or intra-articular hyaluronan**.
- **ACR/Arthritis Foundation and OARSI** continue to **strongly recommend both aquatic and land-based aerobic and resistance exercise** and weight loss as core therapy, and recommend against most passive modalities.
- **Repeated intra-articular corticosteroid injections** have been linked to **accelerated cartilage loss on MRI** — limit frequency (commonly no more than 3–4 per joint per year).
- **Disease-modifying OA drugs remain investigational**: **lorecivivint** (Wnt pathway inhibitor), **sprifermin** (FGF-18), and **anti-NGF antibodies such as tanezumab**, which reduced pain but caused **rapidly progressive osteoarthritis** and were not approved.

Current Practice Update

Aspect	Textbook position	Current evidence / guideline
Concept of disease	Cartilage destruction with secondary changes	**Whole-joint disease** with low-grade inflammation, subchondral bone lesions and central sensitisation
First-line analgesia	Paracetamol and oral NSAIDs	**Topical NSAIDs first** in older adults; paracetamol relegated to modest adjunct (NICE NG226)
Weight management	Advised generally	**Quantified target: 7–10% weight loss**; **semaglutide** shown to reduce WOMAC pain (STEP 9)
Diagnosis	Radiography central	**Clinical diagnosis without imaging** in typical cases aged ≥45 (NICE NG226)
Glucosamine / hyaluronic acid	“Used, evidence limited”	**Explicitly not recommended** (NICE NG226)
Arthroscopy	Considered when inflammation present	**Not recommended** for degenerative knee disease

Exam One-Liners

- **OA is a whole-joint disease, not just wear and tear.**
- **Stiffness in minutes = OA; stiffness in hours = inflammatory arthritis.**
- **Heberden’s = DIP; Bouchard’s = PIP.**
- **Cartilage has no nerves** — pain comes from bone, synovium, capsule and central sensitisation.
- **Cane in the contralateral hand; length = wrist crease to floor.**
- **Exercise and weight loss are the only interventions with both symptom and structural benefit.**
- **STEP 9:** semaglutide reduced knee OA pain by 41.7 WOMAC points with 13.7% weight loss.
- **Glucosamine, chondroitin, hyaluronan and arthroscopic debridement: no proven benefit.**

Q7. Temporal Arteritis (Giant Cell Arteritis) — Diagnosis, Clinical Features, Treatment and Prognosis

A. Definition and Epidemiology

1. Giant cell arteritis is a **granulomatous large- and medium-vessel vasculitis** affecting the **aorta and its extracranial branches**, with a predilection for the **superficial temporal, ophthalmic, posterior ciliary and vertebral arteries**.

2. It is the **commonest primary systemic vasculitis of older people**. It **virtually never occurs before the age of 50** — age ≥50 is an entry criterion for classification — and incidence peaks between **70 and 80 years**. **Female to male ratio is about 2–3:1.** It is far commoner in Northern Europeans than in Indians.

3. GCA overlaps closely with **polymyalgia rheumatica**: about **40–50% of GCA patients have PMR features** and **15–20% of PMR patients develop GCA**.

B. Pathogenesis

4. Dendritic cell activation in the arterial **adventitia** recruits **CD4+ T cells**; **Th1 (interferon-γ)** and **Th17 (IL-17)** pathways drive granuloma formation with **multinucleated giant cells** and **fragmentation of the internal elastic lamina**.

5. IL-6 drives the systemic inflammatory response, explaining the very high ESR and CRP — and the effectiveness of **IL-6 receptor blockade**.

6. Intimal hyperplasia narrows the lumen and produces ischaemia. Involvement is **segmental with skip lesions**, which is why an adequate biopsy length is essential.

C. Clinical Features

7. Cranial symptoms: new-onset temporal or occipital headache (the commonest presenting feature), **scalp tenderness** (pain on combing the hair or resting on a pillow), and a **thickened, tender, nodular, pulseless temporal artery**.

8. Jaw claudication — masseter pain on chewing — is the **single most specific symptom**. Tongue claudication and, rarely, tongue or scalp necrosis may occur.

9. Visual features — the emergency: amaurosis fugax, diplopia, and **sudden painless permanent monocular visual loss**, usually from **anterior ischaemic optic neuropathy** with a **pale, swollen "chalky white" disc**; also central retinal artery occlusion. Untreated, **the second eye is at risk within days to weeks**.

10. Constitutional features: fever (GCA is a classic cause of **pyrexia of unknown origin in the elderly**), weight loss, anorexia, malaise and depression. GCA is an important **reversible cause of frailty and functional decline** in older people, and is often misdiagnosed.

11. PMR features: aching and morning stiffness of the **neck, shoulder and pelvic girdle** lasting more than 45 minutes, with difficulty rising and combing hair.

12. Large-vessel GCA: limb claudication, **asymmetric pulses or blood pressure**, bruits, and late **thoracic aortic aneurysm or dissection**. Rarely, **vertebrobasilar territory stroke** — giant cell arteritis should be considered in any elderly patient with posterior circulation ischaemia.

D. Diagnosis

13. GCA is a medical emergency and a clinical diagnosis. Treatment must never be delayed for investigations or biopsy.

14. Acute-phase response: ESR is characteristically very high — an ESR above 75 mm/h in a patient with headache, jaw claudication or visual change should prompt urgent action. **CRP is more sensitive and rises earlier**. Also expect **normocytic normochromic anaemia**, thrombocytosis, raised alkaline phosphatase and low albumin. About **4% have both a normal ESR and CRP**.

15. Temporal artery biopsy remains the diagnostic gold standard. Take a segment **at least 1–2 cm long** because of skip lesions, and perform it **within 1–2 weeks of starting steroids** — the yield falls with prolonged treatment, though positivity persists for some weeks. **A negative biopsy does not exclude GCA.**

16. Histology: transmural mononuclear infiltrate, **granulomas with multinucleated giant cells**, **disruption of the internal elastic lamina**, and intimal hyperplasia with luminal narrowing.

17. Colour Doppler ultrasound of temporal and axillary arteries shows the **non-compressible "halo sign"** (hypoechoic circumferential wall oedema) — now the **first-line investigation** in fast-track clinics, with the advantage of immediate availability.

18. Large-vessel imaging: CT or MR angiography and **FDG-PET/CT** identify aortitis and subclavian/axillary involvement; PET is especially valuable when GCA presents as a PUO. **Baseline and periodic aortic imaging** is advised for aneurysm surveillance.

19. 2022 ACR/EULAR classification criteria (for classification, not diagnosis) require **age ≥50 as an entry criterion**, then score: morning stiffness of shoulder/neck, sudden visual loss, jaw or tongue claudication, new temporal headache, scalp tenderness, abnormal temporal artery examination, raised ESR/CRP, positive biopsy or halo sign, bilateral axillary

involvement, and FDG-PET uptake in the aorta.

E. Treatment

20. Start high-dose glucocorticoids immediately on clinical suspicion. For **uncomplicated GCA**, prednisolone **40–60 mg/day** (about 1 mg/kg). For **visual symptoms or established visual loss**, **IV methylprednisolone 500–1000 mg daily for 3 days**, followed by oral prednisolone.

21. Symptoms usually improve **dramatically within 24–72 hours**; a poor response should prompt reconsideration of the diagnosis.

22. Taper slowly, guided by symptoms and inflammatory markers: maintain the initial dose for 2–4 weeks, reduce to about **15–20 mg by 3 months** and **5 mg by 12 months**. Most patients need **1–2 years** of treatment, some longer.

23. Steroid-sparing therapy: tocilizumab (IL-6 receptor blocker) **162 mg subcutaneously weekly or every other week**, or 6 mg/kg IV every 4 weeks — the **GiACTA trial** showed sustained steroid-free remission with a markedly lower cumulative glucocorticoid dose. **Methotrexate** is a cheaper alternative with modest benefit, relevant in Indian practice. **Infliximab is not beneficial and is not recommended.**

24. Adjunctive care in every patient: low-dose aspirin (reduces ischaemic events; weigh bleeding risk), a **proton pump inhibitor**, **calcium, vitamin D and a bisphosphonate** for glucocorticoid-induced osteoporosis, **blood glucose and blood pressure monitoring**, **Pneumocystis prophylaxis** if on high-dose steroid with additional immunosuppression, and vaccination.

25. Comprehensive geriatric assessment matters. Glucocorticoid toxicity in older people causes **hyperglycaemia, hypertension, cataract, proximal myopathy, skin fragility, insomnia, delirium, infection and fracture** — steroid harm often exceeds vasculitis harm after the acute phase.

F. Prognosis

26. Overall life expectancy is close to normal. Most morbidity comes from **glucocorticoid toxicity** and from **aortic aneurysm and dissection** (a roughly **17-fold increase in thoracic aortic aneurysm**), rather than from the vasculitis itself.

27. Established visual loss is usually permanent — recovery is rare even with intravenous steroids. **The entire aim of emergency treatment is to protect the second eye.** The risk of blindness falls from about **30% untreated to under 5%** with prompt therapy.

28. Relapse occurs in 40–60%, mostly during tapering in the first 18 months, and usually presents as **headache or PMR symptoms rather than visual loss**. Relapses require dose escalation and consideration of a steroid-sparing agent.

Latest Updates (2023–Present)

- **SELECT-GCA (NEJM 2025; Blockmans et al., 428 patients)** — **upadacitinib 15 mg once daily** with a 26-week glucocorticoid taper achieved **sustained remission in 46.4%** versus **29.0%** for placebo with a 52-week taper. The **7.5 mg dose was not effective**. **FDA and EMA approved upadacitinib for GCA in 2025** — the first **oral targeted therapy** for the disease, and particularly attractive for older patients who prefer tablets to injections.
- **2024 EULAR treat-to-target recommendations for GCA and PMR (Ann Rheum Dis)** define **remission as absence of clinical signs and symptoms plus normalised ESR and CRP**, and set explicit targets for tapering and sustained remission.
- The **ACR/Vasculitis Foundation guideline** conditionally recommends **adding tocilizumab to high-dose glucocorticoids in newly diagnosed GCA**, and prefers **tocilizumab over methotrexate** in relapsing disease with cranial ischaemia.
- **Tocilizumab dose optimisation** after 12 months of sustained remission — halving the IV dose (8 to 4 mg/kg) or extending subcutaneous dosing from weekly to every other week — maintained remission at similar rates with **significantly fewer severe infections (6.6% versus 12.9%)** and lower cost.
- **EULAR imaging recommendations** now place **ultrasound as the first-line investigation** in suspected cranial GCA, with biopsy or additional imaging when ultrasound is negative but clinical suspicion persists. **Fast-track pathways** combining same-day ultrasound with immediate steroids have **substantially reduced permanent visual loss**.
- **JAK inhibitors in relapsing GCA**: real-world series confirm effectiveness in refractory disease, though **cardiovascular, thrombotic and malignancy warnings apply to JAK inhibitors in the over-65 population** and must be weighed carefully.

Current Practice Update

Aspect	Textbook position	Current evidence / guideline
First diagnostic test	Temporal artery biopsy	**Colour Doppler ultrasound (halo sign) first-line**; biopsy if negative and suspicion persists (EULAR)
Steroid-sparing therapy	Methotrexate; infliximab not recommended	**Tocilizumab** (GiACTA) and now **oral upadacitinib** (SELECT-GCA 2025, FDA/EMA approved)
Treatment target	Symptom control and falling ESR	**Formal treat-to-target**: no clinical signs **plus** normalised ESR/CRP (EULAR 2024)
Long-term surveillance	Not emphasised	**Periodic aortic imaging** for aneurysm (17-fold increased risk)
Service model	Individual referral	**Fast-track GCA clinics** with same-day ultrasound reduce blindness

Exam One-Liners

- **Never occurs under age 50**; peak incidence 70–80 years.
- **Jaw claudication is the most specific symptom**; **visual loss is the feared complication**.
- **ESR often >75 mm/h**, but **4% have normal ESR and CRP** — treat on clinical suspicion.
- **Start steroids first, biopsy after** — biopsy stays positive for a couple of weeks.
- **Biopsy 1–2 cm long** because of skip lesions; a negative biopsy does not exclude GCA.
- **Visual loss + GCA = IV methylprednisolone 1 g for 3 days** to save the other eye.
- **Established blindness is irreversible** — the aim is protecting the second eye.
- **SELECT-GCA 2025:** upadacitinib 15 mg is the first oral targeted therapy approved for GCA.

Q8. Dermatomyositis in the Elderly — Presentation, Comorbidities, Investigations, Treatment and Prognosis

A. Definition and Basis

1. Dermatomyositis is an **idiopathic inflammatory myopathy** combining **characteristic cutaneous features** with **symmetrical proximal muscle weakness**.

2. The mechanism is **humoral and complement-mediated**: deposition of the **C5b-9 membrane attack complex** on **endomysial capillaries** causes capillary dropout and ischaemia of the periphery of the fascicle — hence the pathognomonic **perifascicular atrophy** on muscle biopsy. This contrasts with polymyositis and inclusion body myositis, which are CD8+ T-cell mediated.

3. The disease has a **bimodal age distribution**; the adult peak is in the **fifth to sixth decades**, and **elderly onset carries the highest malignancy risk of any myositis subtype**.

B. Presentation in the Elderly

4. Skin changes often precede muscle disease by weeks to months: the **heliotrope rash** (violaceous periorbital discoloration with oedema) and **Gottron's papules** over the MCP, PIP and DIP joints, with **Gottron's sign** over elbows and knees.

5. Photodistributed erythema: the **shawl sign** (upper back and shoulders), **V-sign** (anterior chest and neck), **holster sign** (lateral thighs, notably a non-sun-exposed site), and facial erythema.

6. Other cutaneous signs: periungual erythema with dilated, tortuous nailfold capillary loops and ragged cuticles, **mechanic's hands** (hyperkeratotic fissured lateral fingers, associated with the anti-synthetase syndrome), **poikiloderma**, **calcinosis cutis** (less common in adults than in juvenile disease), and **intense pruritus** — a prominent and frequently overlooked complaint.

7. Muscle involvement: insidious **symmetrical proximal weakness** — difficulty rising from a chair or toilet, climbing stairs, combing hair and hanging out clothes. **Pain is not prominent**, and **neck flexor weakness** is characteristic.

8. The critical geriatric pitfall: proximal weakness in an older person is easily misattributed to **normal ageing, sarcopenia, deconditioning, polymyalgia rheumatica, statin myopathy, hypothyroid myopathy or steroid myopathy** — leading to dangerous delay in diagnosis and malignancy search.

9. Dysphagia from pharyngeal and upper oesophageal striated muscle involvement occurs in up to a third; it predicts **aspiration pneumonia and death**, and may be accompanied by dysphonia and nasal regurgitation.

10. Clinically amyopathic dermatomyositis accounts for about **20% of cases** — the classical rash with **no clinically evident weakness**. It still carries the **full risk of malignancy and interstitial lung disease** and must be investigated identically.

C. Comorbidities and Associations

11. Malignancy is the dominant geriatric concern. Cancer-associated myositis is defined as a malignancy occurring **within 3 years before or after** the myositis diagnosis. Some myositis develops as a **paraneoplastic syndrome**, and treating the tumour often improves the myositis.

12. Anti-TIF1-γ (anti-p155) is the key marker — present in about **7–15% of dermatomyositis overall** but in **more than half of cancer-associated myositis**; roughly **40% of anti-TIF1-γ-positive patients have a malignancy**, with the association strongest in the elderly. **Anti-NXP2** is also malignancy-associated and causes calcinosis and dysphagia.

13. Commonest associated tumours: ovary, lung, breast, stomach, colorectum and pancreas in Western series; **nasopharyngeal carcinoma is over-represented in Asian populations** — highly relevant in the Indian context.

14. Interstitial lung disease occurs in **20–40%**. **Anti-MDA5** predicts **rapidly progressive ILD** with a characteristic **ulcerating cutaneous vasculopathy and palmar papules**, and carries the **worst prognosis** of all subtypes. **Anti-synthetase syndrome** (anti-Jo-1 and others) = myositis, ILD, arthritis, Raynaud's, fever and mechanic's hands.

15. Cardiac involvement: myocarditis, conduction defects, arrhythmias and heart failure — a **leading cause of death**; there is also accelerated atherosclerosis.

16. Other comorbidities: aspiration pneumonia, **venous thromboembolism**, **glucocorticoid-induced osteoporosis and fracture**, infection, severe deconditioning and sarcopenia, and **overlap syndromes** with systemic sclerosis, SLE and Sjögren's syndrome.

D. Investigations

17. Muscle enzymes: creatine kinase, aldolase, LDH, AST and ALT. Note two traps — CK may be **normal in amyopathic disease** and is often **relatively lower in the elderly** because of reduced muscle mass; and the raised transaminases produce a **false "hepatitis" pattern that is actually muscle in origin**.

18. Autoantibodies are now central. Check ANA, then an **extended myositis panel** for **myositis-specific antibodies (anti-TIF1-γ, NXP2, MDA5, Mi-2, SAE and the synthetases)** and **myositis-associated antibodies (PM-Scl, Ro52, U1-RNP)**. These **define phenotype, malignancy risk and prognosis better than any other single test**.

19. Electromyography: a **myopathic irritative pattern** — short-duration, small-amplitude polyphasic motor unit

potentials with **fibrillations, positive sharp waves and increased insertional activity**.

20. MRI of the thighs with T2/STIR sequences shows **patchy muscle oedema**, guides the biopsy site, and distinguishes **active inflammation from irreversible fatty replacement** — useful in the elderly for deciding whether persistent weakness warrants more immunosuppression.

21. Muscle biopsy: perifascicular atrophy, perimysial and perivascular infiltrate of CD4+ T cells, B cells and plasmacytoid dendritic cells, **capillary dropout with MAC deposition**, and **MHC class I upregulation**. Skin biopsy shows **interface dermatitis** indistinguishable from lupus.

22. Malignancy screening is mandatory in every patient over 40. Baseline: full history and examination including breast, pelvic, rectal and lymph node assessment; complete blood count, biochemistry, urinalysis and faecal occult blood; chest radiograph; **age- and sex-appropriate screening** (mammography, cervical cytology, PSA, colonoscopy); and **CA-125 with transvaginal ultrasound in women**. **High-risk patients need CT of chest, abdomen and pelvis, with FDG-PET/CT considered**, repeated **annually for 3 years**.

23. Organ-specific baseline work-up: HRCT chest and pulmonary function tests with DLCO for ILD; **ECG, troponin and echocardiography** for cardiac involvement; and **videofluoroscopic swallow assessment** if there is any dysphagia.

E. Treatment

24. Prednisolone 0.5–1 mg/kg/day is first-line, tapered over 6–12 months once strength improves. Use **IV methylprednisolone pulses** for severe disease, dysphagia, cardiac involvement or rapidly progressive ILD.

25. Start a steroid-sparing agent early in older people to limit steroid toxicity: **methotrexate or azathioprine** first-line; **mycophenolate mofetil**, particularly with ILD; **calcineurin inhibitors (tacrolimus, ciclosporin)** for anti-MDA5 ILD; and **cyclophosphamide or rituximab** for refractory or severe disease.

26. Intravenous immunoglobulin 2 g/kg is effective in refractory disease, in dysphagia, and where immunosuppression is hazardous (active infection or malignancy) — but watch for **thromboembolism, volume overload and renal impairment** in older patients.

27. Skin disease requires its own plan: rigorous **photoprotection**, topical corticosteroids and calcineurin inhibitors, **hydroxychloroquine**, and antipruritics — cutaneous disease often persists after myositis has responded.

28. Supportive and geriatric care matters as much as immunosuppression: early **physiotherapy and graded resistance exercise** (no longer contraindicated and now recommended), **speech and swallow therapy** with texture modification and aspiration precautions, **nutritional support**, **VTE prophylaxis**, **bone protection with calcium, vitamin D and a bisphosphonate**, **Pneumocystis prophylaxis**, glucose monitoring, vaccination and falls prevention.

29. Treat any underlying malignancy — the myositis frequently improves with successful tumour therapy, which justifies an aggressive and repeated cancer search.

F. Prognosis

30. Poor prognostic factors: older age, **malignancy**, **interstitial lung disease (especially anti-MDA5)**, **dysphagia**, **cardiac involvement**, delayed treatment, and severe weakness at presentation.

31. Malignancy-associated dermatomyositis has substantially higher mortality; overall survival in elderly-onset disease is markedly worse than in younger patients. The leading causes of death are **cancer, aspiration pneumonia, interstitial lung disease, cardiac disease and infection**.

32. Functional recovery is often incomplete — residual weakness, fatigue and deconditioning are common, so rehabilitation goals should be **functional and realistic**, focused on activities of daily living, transfers and safe swallowing rather than complete normalisation of strength.

Latest Updates (2023–Present)

- **IMACS International Guideline for IIM-Associated Cancer Screening (Nature Reviews Rheumatology, 2024)** — the major practice change. It stratifies patients into **standard, moderate and high risk**. High-risk features include **dermatomyositis subtype, anti-TIF1-γ or anti-NXP2 positivity, age over 40 at onset**, and refractory or severe disease. Worked example: a **70-year-old with anti-NXP2-positive dermatomyositis meets three high-risk criteria** and requires **enhanced screening with CT chest, abdomen and pelvis at diagnosis and annually for 3 years**.
- **ProDERM trial (NEJM 2022)** established **IVIG 2 g/kg every 4 weeks** as an evidence-based therapy for adult dermatomyositis, leading to **FDA approval of IVIG for this indication** — the first drug formally approved for dermatomyositis.
- **2017 EULAR/ACR classification criteria** have replaced Bohan and Peter in research and increasingly in practice; they use a **probability-based score** and permit classification **without a muscle biopsy** when the characteristic rash is present.
- **Anti-MDA5 dermatomyositis with rapidly progressive ILD** is now treated with **upfront triple therapy** — high-dose glucocorticoid plus a calcineurin inhibitor plus cyclophosphamide — with **JAK inhibitors (tofacitinib)** showing benefit in refractory cases based on the type I interferon signature.
- **Multiple simultaneous myositis-specific antibodies** are increasingly reported (about 15.7% of MSA-positive patients), complicating phenotype prediction and reinforcing the need to interpret panels alongside the clinical picture.
- **Exercise is now actively recommended** rather than avoided: supervised aerobic and resistance training improves strength and function without increasing muscle inflammation, and is especially important in older patients at risk of

steroid myopathy and sarcopenia.

Current Practice Update

Aspect	Textbook position	Current evidence / guideline
Diagnostic framework	Bohan and Peter criteria, biopsy-centred	**2017 EULAR/ACR criteria**; **myositis antibody panel** now central and biopsy sometimes avoidable
Cancer screening	“Screen for malignancy in the elderly”	**Risk-stratified IMACS 2024 guideline** — antibody-driven, with CT chest/abdomen/pelvis annually for 3 years in high risk
Refractory disease	Steroids plus conventional immunosuppressants	**IVIG is now FDA-approved and evidence-based (ProDERM)**; rituximab for refractory disease
Exercise	Traditionally cautioned against in active disease	**Supervised aerobic and resistance exercise recommended**; safe and improves function
Anti-MDA5 phenotype	Not described	Recognised as a distinct, **often amyopathic, rapidly progressive ILD phenotype with the worst prognosis**; needs upfront triple therapy

Exam One-Liners

- **Perifascicular atrophy** = the histological hallmark; the mechanism is **complement-mediated capillary injury**.
- **Heliotrope rash + Gottron’s papules** = dermatomyositis until proved otherwise.
- **Anti-TIF1-γ = malignancy; anti-MDA5 = rapidly progressive ILD; anti-Jo-1 = anti-synthetase syndrome; anti-Mi-2 = classic rash with good prognosis.**
- **Amyopathic dermatomyositis carries the same malignancy and ILD risk** as classic disease.
- **Dysphagia predicts aspiration and death** — assess the swallow in every patient.
- **Every patient over 40 needs malignancy screening, repeated annually for 3 years.**
- **ProDERM:** IVIG 2 g/kg is FDA-approved for adult dermatomyositis.
- **Treating the tumour often treats the myositis.**

Q9. Diffuse Idiopathic Skeletal Hyperostosis (DISH) — Presentation, Investigations and Treatment

A. Definition and Epidemiology

1. DISH (**Forestier–Rotés-Querol disease**, originally "senile ankylosing hyperostosis") is a **systemic, non-inflammatory skeletal disorder** characterised by **ossification and calcification of spinal and extraspinal ligaments and entheses**, particularly the **anterior longitudinal ligament**.

2. It is a **disease of older adults** — prevalence rises steeply after 50 and reaches **up to 25–40% of men over 70**, with a **male to female ratio of about 2:1**. The **majority of cases are asymptomatic** and found incidentally.

3. DISH is a **"new bone formation" disorder** — it is **neither degenerative disc disease nor an inflammatory spondyloarthropathy**, though imaging overlap with both is common.

B. Aetiology and Associations

4. The cause is unknown. The core abnormality is **differentiation of enthesial fibroblasts into bone-forming cells**, driven by local growth factors — **insulin, IGF-1, growth hormone, TGF-β, bone morphogenetic proteins and endothelin-1**.

5. Strong metabolic associations: type 2 diabetes and hyperinsulinaemia, obesity, hypertension, dyslipidaemia and hyperuricaemia. DISH is increasingly regarded as **the skeletal manifestation of the metabolic syndrome**.

6. Other associations: male sex, advancing age, **long-term retinoid use** (isotretinoin, etretinate) and hypervitaminosis A, **hypoparathyroidism**, acromegaly, and probable genetic contributions (*COL6A1*).

7. Paradox worth remembering: patients with DISH have **higher bone mineral density than controls**, yet **fracture easily** — the long, rigid, ankylosed spine behaves mechanically like a long bone.

C. Clinical Presentation

8. Most patients are asymptomatic; DISH is commonly an incidental finding on a chest radiograph or CT performed for another reason.

9. Axial symptoms: dull **mid-thoracic and lumbar back pain** with progressive **stiffness**, typically mild, worse after inactivity and in cold weather, and **without the dramatic NSAID response seen in spondyloarthritis**.

10. Reduced spinal range of movement with preserved sacroiliac joints, progressive **thoracic kyphosis** and loss of lumbar lordosis, contributing to postural instability and falls.

11. Cervical DISH and dysphagia — the key geriatric presentation. Large **anterior osteophytes at C3–C5** mechanically compress and tether the oesophagus, producing **progressive dysphagia to solids and then liquids, with weight loss**. This is an **under-recognised and reversible cause of dysphagia** in older adults, sometimes termed **"DISHphagia"**.

12. Other cervical complications: hoarseness, globus sensation, **stridor and acute airway obstruction**, obstructive sleep apnoea, **aspiration pneumonia**, and **difficult intubation or failed nasogastric tube placement**.

13. Peripheral enthesopathy: calcaneal (heel) spurs, olecranon and patellar spurs, and **"whiskering" of the iliac crest, ischial tuberosity and greater trochanter** — these can cause local pain and be mistaken for tendinitis or bursitis.

14. Serious spinal complications: ossification of the posterior longitudinal ligament (OPLL) and ligamentum flavum causing **myelopathy or radiculopathy**; and — most important — **unstable three-column hyperextension fractures of the rigid spine after trivial falls**, which carry high rates of **delayed diagnosis, neurological injury and mortality**.

D. Investigations

15. Diagnosis is radiological, using the Resnick and Niwayama criteria on a **lateral thoracic spine radiograph**: (i) **flowing ossification along the anterolateral aspect of at least four contiguous vertebral bodies**; (ii) **preserved intervertebral disc height** in the involved segments without significant degenerative disc disease; and (iii) **absence of apophyseal (facet) joint ankylosis and of sacroiliac erosion, sclerosis or fusion**.

16. The classic radiographic appearance is **"flowing" or "dripping candle wax" ossification**, most often on the **right side of the thoracic spine** — the pulsating descending thoracic aorta is thought to protect the left side.

17. CT is best for defining osteophyte size, oesophageal and airway compression, and — critically — for **fracture detection in the rigid spine**. **Always obtain CT (and MRI if there is any neurology) rather than plain films when a DISH patient falls**, because fractures are easily missed.

18. Barium swallow / videofluoroscopy dynamically demonstrates the **level responsible for dysphagia** and confirms adequate oesophageal decompression after surgery. **Flexible endoscopy** excludes malignancy and **manometry** excludes a motility disorder.

19. Laboratory tests are normal — ESR and CRP normal or only mildly raised, **HLA-B27 negative**, calcium, phosphate and alkaline phosphatase normal. **Screen every patient for diabetes, lipids, uric acid and blood pressure**, since the metabolic comorbidity is the real health burden.

20. Key differential diagnoses:

Condition	Distinguishing features
Ankylosing spondylitis	Younger onset, **sacroiliitis**, thin vertical **syndesmophytes**, HLA-B27 positive, raised ESR/CRP, marked NSAID response, inflammatory back pain
Spondylosis deformans	Disc space **narrowing**, horizontal/beak-like osteophytes, no flowing ossification
OPLL	Posterior longitudinal ligament ossification, myelopathy; may coexist with DISH
Acromegaly	Enlarged extremities, prognathism, raised IGF-1
Fluorosis	**Important in endemic areas of India** — dental mottling, generalised osteosclerosis, ligamentous calcification, raised urinary fluoride
Hypoparathyroidism	Low calcium, high phosphate, ectopic calcification

E. Treatment

21. There is **no disease-modifying therapy**; management is **symptomatic and complication-focused**.

22. Conservative care: analgesics and **NSAIDs** for pain, **physiotherapy** for posture, range of motion and core strength, activity modification, local heat, and **weight reduction with tight control of diabetes, lipids and blood pressure** — metabolic control is the only intervention likely to influence the underlying process.

23. Management of dysphagia: diet and texture modification with speech-and-language therapy, anti-reflux measures and proton pump inhibitors, **upright positioning and small frequent meals**, and muscle relaxants where spasm contributes. **Enteral feeding or gastrostomy** for refractory dysphagia with weight loss.

24. Local corticosteroid injection helps symptomatic peripheral enthesopathy; **bisphosphonates** have been used for painful enthesopathy with limited evidence.

25. Surgery is reserved for failure of conservative treatment, **severe dysphagia with significant weight loss**, or **airway compromise**. The procedure is **anterior osteophytectomy** through a **Smith–Robinson or trans-pharyngeal approach**, sometimes combined with **cricopharyngeal myotomy**. Symptom relief is generally excellent, but counsel about **recurrence and complications** — recurrent laryngeal nerve palsy, haematoma, oesophageal perforation and dysphonia. **Tracheostomy** may be needed for acute airway obstruction.

26. Spinal decompression and stabilisation for myelopathy or fracture. **Any DISH patient who falls and has new spinal pain must be assumed to have an unstable fracture until CT proves otherwise** — a classic and dangerous geriatric trap.

27. Anaesthetic and nursing implications: anticipate **difficult intubation** (fibreoptic technique, cervical rigidity), difficult nasogastric tube insertion, careful positioning of the rigid kyphotic spine, and **meticulous pressure-area care**.

Latest Updates (2023–Present)

- **Resnick's criteria remain the gold standard**, but 2024–2025 real-life cohort imaging studies confirm **substantial overlap of inflammatory and degenerative features between DISH, degenerative disc disease and axial spondyloarthritis** on radiographs and MRI. DISH and axSpA can coexist — **the presence of DISH should not stop a spondyloarthritis work-up** in a symptomatic patient.
- **Systematic reviews and case series (2024–2025)** confirm that **anterior osteophytectomy for DISH-related dysphagia gives excellent and durable symptom relief**, with **barium swallow fluoroscopy emphasised** both for identifying the culpable level pre-operatively and for confirming oesophageal decompression post-operatively.
- **Growing recognition of DISH as a skeletal marker of metabolic syndrome**, with proposals that patients found to have incidental DISH on imaging should be **screened for diabetes and cardiovascular risk** — a practical, low-cost intervention for Indian practice.
- **Increasing reports of stridor and acute respiratory distress from cervical DISH**, including from Indian centres, highlighting the need to consider DISH in any older adult with unexplained stridor, and reporting a **prevalence of up to 42% in individuals over 65**.
- **Rare presentations** now documented include **upper cervical (C1–C3) DISH with ossification of the anterior longitudinal and alar ligaments**, with implications for airway management and cervical stability.
- **Emphasis on fracture risk** continues to grow: DISH patients have **higher BMD but sustain unstable hyperextension fractures after low-energy trauma**, so **CT and MRI are mandatory even after apparently minor falls**.

Current Practice Update

Aspect	Textbook position	Current evidence / guideline
Nature of the disease	Age-related ossifying disorder	**Skeletal manifestation of metabolic syndrome** — screen for diabetes and cardiovascular risk
Relationship to axSpA	Considered mutually exclusive	**Substantial imaging overlap; DISH and axSpA can coexist** (2024 cohort studies)
Dysphagia work-up	Plain radiography	**CT plus dynamic barium swallow fluoroscopy** to identify the culpable level and confirm post-operative decompression
Bone strength	High BMD implies safety	**High BMD but high fracture risk** — rigid spine sustains unstable fractures after trivial trauma
Post-fall imaging	Plain films	**CT (± MRI) mandatory** after any fall with new spinal pain
Surgical outcome	Reserved, uncertain	**Excellent durable relief** from anterior osteophytectomy in appropriately selected patients

Exam One-Liners

- **Resnick's criteria:** flowing ossification of **≥4 contiguous vertebrae**, **preserved disc spaces**, **no facet or sacroiliac fusion**.
- **"Dripping candle wax" appearance, right side of the thoracic spine.**
- **HLA-B27 negative, ESR normal, sacroiliac joints normal** — this separates DISH from ankylosing spondylitis.
- **Cervical DISH is a reversible cause of dysphagia in the elderly** ("DISHphagia").
- **DISH = the skeletal face of metabolic syndrome** — always check the sugar.
- **High BMD but fractures easily** — a rigid spine breaks like a long bone.
- **Any DISH patient who falls needs a CT, not a plain film.**
- **Think DISH in unexplained difficult intubation or failed NG tube placement.**

Cross-Topic Rapid Revision Grid

Condition	One-line hallmark	Key investigation	First-line treatment
Osteoporosis	Low mass + poor microarchitecture, fragility fracture	DXA T-score ≤ −2.5, FRAX	Calcium/vitamin D + bisphosphonate; anabolic-first if very high risk
Paget's disease	Focal, disorganised remodelling; high ALP with normal calcium	X-ray + radionuclide bone scan	Single 5 mg IV zoledronate
Osteoarthritis	Activity pain, stiffness in minutes, osteophytes	Clinical ± X-ray	Exercise + weight loss + topical NSAID
Giant cell arteritis	Headache, jaw claudication, visual loss, ESR >75	Ultrasound halo sign / temporal artery biopsy	Immediate high-dose steroid ± tocilizumab or upadacitinib
Dermatomyositis	Heliotrope rash + Gottron's + proximal weakness	Myositis antibody panel, MRI, biopsy	Prednisolone + early steroid-sparing agent; IVIG if refractory
DISH	Flowing ossification, dysphagia, metabolic syndrome	Lateral thoracic X-ray (Resnick), CT	Symptomatic; metabolic control; surgery for dysphagia

MD GERIATRIC MEDICINE

Examination Preparation — Structured Answers

MODULE:
LONG-TERM CARE, COMMUNITY SERVICES AND CAREGIVER SUPPORT

Primary sources: Pathy's Principles and Practice of Geriatric Medicine (6th edition) and Kane's Essentials of Clinical Geriatrics
with Latest Updates (2023–2026) and Indian clinical & policy context

Compiled for Dr. Sudarsan | Seven questions with high-yield points, latest updates, current practice comparison tables and exam one-liners

Contents

1.	Long-term care — concept, continuum, nursing home care and regulation	15 marks
2.	Community-based long-term care services (HCBS)	15 marks
3.	Home-based care in the elderly	15 marks
4.	Day hospitals and day care centres for older adults	15 marks
5.	House call programme (home-based medical care)	15 marks
6.	Components of the Hospital Elder Life Program (HELP)	15 marks
7.	Role of the geriatric social worker	15 marks
8.	Caregiver burden, stress and burnout — assessment and management	15 marks

How each answer is structured:

- **High-yield numbered points** (15–20) under labelled subheadings, keywords in bold
- **Latest Updates (2023–Present)** — named trials, guidelines and Indian policy cited inline
- **Current Practice Update** — table contrasting textbook position with current evidence
- **Exam One-Liners** — rapid recall for the night before the examination

Q1. Long-Term Care

Concept, continuum of services, nursing home care, quality regulation and financing — 15 marks

A. Definition and Concept

1. **Long-term care** is a range of **health, personal care and social services** given to people who have lost some capacity for **self-care**. Services may be continuous or intermittent, but are delivered over a **sustained period**.
2. The single common thread is **dependence on another person for a substantial period**. Need is measured by an index of **functional incapacity (ADL/IADL loss)**, not by diagnosis or age.
3. LTC is **not primarily medical care**. It is delivered along a **continuum**: informal family care → home care → community and day services → assisted living → nursing home → hospice.

B. Epidemiology and Burden

1. Nursing home use rises **steeply with age** — about **1% at 65–74 years, 4% at 75–84 years and 14% at ≥85 years**. Institutional use has fallen over recent decades because of **alternative residential settings**.
2. A person turning 65 will need on average about **3 years of LTC**; among actual users this is about **4.3 years**, split almost equally between **community-based and facility-based care**.
3. For every person in a nursing home there are **one to three equally disabled persons living in the community** — placement is therefore not determined by disability alone.
4. Nursing home residents are typically **older, female, unmarried or widowed**, with **multiple chronic conditions** and greater disability than earlier cohorts.

C. Determinants of Institutionalisation

1. Key risk factors: **advanced age, widowhood, living alone, dementia with behavioural symptoms, incontinence, immobility, recurrent falls, poverty and carer breakdown**.
2. Risk of nursing home use rises sharply with **lifestyle-related disease** — **hypertension, diabetes mellitus and physical inactivity**.

D. Components of the LTC Continuum

1. **Informal (family) care is the backbone** — over **80% of community care is informal**, largely provided by women. Formal services should **supplement, not replace**, family care.
2. **Home care** has two arms: **home health services** (nursing, physiotherapy, occupational therapy, speech therapy) and **homemaker/chore services** (bathing, meals, medication supervision). Most older people need **homemaking more than home health**.
3. **Community services**: **adult day care, day hospitals, senior centres, home-delivered meals, transport, respite care and case management**. **Respite care** is critical to prevent carer burnout.
4. **Assisted living**: the resident is treated as a **tenant**, not a patient — a **lockable single unit**, own living and sleeping space, bathroom and minimal cooking facility, with services brought in. It maximises **autonomy and privacy**.
5. **Adult foster homes** (usually ≤5 residents) and **nursing homes** complete the spectrum. Nursing homes serve **two distinct streams**: **short-stay post-acute rehabilitation** (most discharged by 21–90 days) and **long-stay custodial care** (mainly dementia and chronic disability).

E. Clinical Care in the Nursing Home

1. Common problems: **dementia with behavioural disturbance, depression, stroke, parkinsonism, delirium, incontinence, falls, pressure ulcers, malnutrition and dehydration,**

chronic pain, UTI and pneumonia, polypharmacy, and **iatrogenic disability from restraints and catheters**.

2. **Initial medical assessment within 72 hours to 1 week**: verify diagnoses, perform **medication reconciliation**, document baseline function, cognition and skin, and **define the goal of admission** (rehabilitation, long-term care or palliative care).
3. **Interdisciplinary care planning** — nursing, social work, dietetics, activities and rehabilitation — meets **within 2 weeks of admission and quarterly thereafter**. The **Minimum Data Set (MDS 3.0)** of the **Resident Assessment Instrument** is completed within **14 days**, updated quarterly and on any significant change. **Physician–nurse practitioner teams reduce hospitalisation.**

F. Quality, Regulation and Safety

1. **OBRA 1987** transformed LTC — it mandated **systematic assessment and care planning**, **restraint reduction**, **rational psychotropic use** and better staff training, with the goal of the **"highest practicable level of functioning."** Frequently cited deficiencies remain **restraints, unnecessary psychotropics, weight loss, pressure ulcers and fall injuries**.
2. Quality is tracked through **quality indicators/measures** (falls with injury, pressure ulcers, antipsychotic use, catheter use, worsening function, weight loss, vaccination, rehospitalisation), publicly reported as the **Five-Star rating** on **Nursing Home Compare**, and improved through **QAPI**. Nursing homes have roughly **one-sixth the staff-per-bed ratio of hospitals**, mostly aides; **stable staffing predicts better quality**, while **marketisation and private-equity ownership** are linked to poor quality.

G. Ethics, Financing and Principles

1. Recurring ethical issues are **autonomy versus safety, capacity assessment, advance directives, tube feeding, hospital transfer and end-of-life decisions**. Financing is fragmented — insurance typically covers **skilled/post-acute care only**, with **custodial care paid out-of-pocket or by welfare after "spend-down." Culture-change models** (Eden Alternative, Wellspring, small living communities) humanise care. **The guiding principle: nursing home placement should be the last resort, not the first.**

Latest Updates (2023–Present)

Global burden and WHO position

- An estimated **142 million older people worldwide cannot meet their basic needs independently**, and **two out of three older people** are likely to need care and support at some point. WHO estimates about **65% will need help with activities of daily living**, and **unpaid informal care accounts for about 80% of all long-term care**.
- The **WHO Framework for an Integrated Continuum of Long-Term Care** defines six system elements — **governance, sustainable financing, information/monitoring and evaluation systems, workforce, service delivery, and innovation and research** — built on establishing foundations for LTC as part of **universal health coverage**, building a **sustainable trained workforce**, and **supporting unpaid caregivers**.
- **ICOPE handbook, 2nd edition (WHO, January 2025)** — *Integrated Care for Older People: Guidance for Person-Centred Assessment and Pathways in Primary Care* — emphasises personalised, comprehensive, coordinated care and a shift from a **disease-oriented to a holistic approach**, focused on optimising **intrinsic capacity and functional ability**, spanning promotion and prevention through treatment, palliative care and long-term care.
- **PAHO Policy on Long-Term Care (2025–2034)**, adopted by the 61st Directing Council, is the first dedicated regional LTC policy, guiding Member States to develop, strengthen and expand LTC capacity.

- **WHA78 (2025)**: the **Global Action Plan on the Public Health Response to Dementia was extended to 2031** — directly relevant, as dementia is the commonest driver of LTC need. The **UN Decade of Healthy Ageing (2021–2030)** reached its mid-point in 2025.

Indian data and policy

- **India Ageing Report 2023 (UNFPA–IIPS, based on LASI)**: the elderly population is growing at a **decadal rate of 41%**; by 2050 more than **20% of India's population will be elderly**, and the elderly will **outnumber children by 2046**. The **80+ population will grow by about 279% between 2022 and 2050**, with a predominance of widowed, highly dependent very old women. More than **40% of elderly Indians are in the poorest wealth quintile** and about **18.7% live without any income**.
- Its key LTC recommendations: bring **all old age homes under regulatory purview**, raise awareness of existing schemes, and facilitate **in-situ (at-home) ageing** by creating short-term care facilities such as **elder creches and day-care centres**.
- The **Global Observatory for Long-Term Care (2025)** records that India has **no national formal long-term care system** — extended care occurs at home, supported by relatives or privately hired caregivers.
- **AB PM-JAY expansion** (Cabinet approval September 2024; launched 29 October 2024): coverage of **₹5 lakh per family per year for all senior citizens aged 70 years and above irrespective of income**, covering about **6 crore seniors in 4.5 crore families**, with a distinct **Ayushman Vay Vandana Card**; those already covered receive an additional top-up of ₹5 lakh. Note it funds **hospitalisation, not custodial long-term care**.
- **AVYAY (Atal Vayo Abhyuday Yojana)** remains the umbrella welfare scheme — grants for old-age homes, physiotherapy centres and mobile medicare units, assistive devices under **RVY**, the **Elder Line (14567)**, the **SACRED** portal for elderly employment, and **SAGE** for elder-care start-ups — alongside **NPHCE** for health services.

Nursing home staffing regulation — a major reversal

- CMS finalised the **Minimum Staffing Standards for Long-Term Care Facilities** rule in April 2024, setting a total nurse staffing standard of **3.48 hours per resident day (HPRD)**, including at least **0.55 HPRD registered nurse** and **2.45 HPRD nurse aide** care, plus **24/7 onsite RN** cover.
- Federal courts in **Texas (April 2025)** and **Iowa (June 2025)** blocked the rule, and the 2025 reconciliation law imposed a **10-year moratorium on enforcement until 30 September 2034**. CMS then formally **repealed** the HPRD minimums and the 24/7 onsite RN requirement, **reinstating the earlier standard of an RN for at least 8 consecutive hours a day, 7 days a week, with a full-time RN Director of Nursing**; the interim final rule takes effect **2 February 2026**. The **enhanced facility assessment** requirement — staffing according to actual resident acuity — **remains in force**.

Current Practice Update

Aspect	Textbook Position	Current Practice (2023–2026)
Conceptual basis of LTC	Service- and facility-centred; defined by dependence and functional incapacity	**Intrinsic capacity and functional ability** model; LTC as one segment of an **integrated continuum** (WHO ICOPE, 2nd ed. 2025)
Preferred site of care	Shift from nursing home to home- and community-based services already under way	Explicit global and Indian policy of **"ageing in place" / in-situ ageing**, with elder day-care and creche models
Standard assessment tool	MDS 3.0 / Resident Assessment Instrument	

		MDS 3.0 in USA, **interRAI** in several countries; **ICOPE screening + person-centred pathway** for community and primary care
Nursing home staffing	No firm numeric federal minimum; quality tied to stable staffing	2024 numeric HPRD standards introduced, then **vacated by courts and repealed by CMS effective 2 Feb 2026**; acuity-based **facility assessment** retained
Financing for older Indians	Not part of the classical LTC financing model	**AB PM-JAY universal cover for ≥70 years (₹5 lakh)**, but still **no funded custodial LTC benefit** in India
Regulation of old age homes in India	—	**India Ageing Report 2023** recommends bringing all old age homes under a **regulatory body**
Dementia in LTC	A leading cause of long-stay admission	**Global Dementia Action Plan extended to 2031 (WHA78, 2025)** with emphasis on carer support

Exam One-Liners

- **LTC = care for dependence, not for disease** — defined by **functional incapacity**, not diagnosis.
- **Informal care provides >80%** of all community long-term care.
- **Nursing home use: 1% (65–74), 4% (75–84), 14% (≥85).**
- **Average LTC need after age 65 ≈ 3 years**; among users ≈ **4.3 years**.
- **OBRA 1987** = landmark LTC reform → **RAI/MDS, restraint reduction, psychotropic reduction**.
- **MDS 3.0** completed within **14 days** of admission; care plan reviewed **quarterly**.
- **Assisted living = tenant model** (lockable single unit, privacy, autonomy).
- **Respite care prevents carer burnout** and delays institutionalisation.
- **ICOPE (WHO)** = intrinsic capacity-based integrated care pathway; **2nd edition January 2025**.
- **WHO LTC framework pillars**: governance, financing, information systems, workforce, service delivery, innovation.
- **India Ageing Report 2023**: elderly **>20% by 2050**; **80+ up 279%**; elderly outnumber children by **2046**.
- **AB PM-JAY** now universal for **≥70 years — Ayushman Vay Vandana Card, ₹5 lakh/family/year**.
- **India has no formal national LTC system** — family remains the default provider.
- **Nursing home placement is the last resort, not the first.**

Q2. Community-Based Long-Term Care Services

Home and community-based services (HCBS) — components, case management, integrated models — 15 marks

A. Definition and Rationale

1. **Community-based long-term care (Home and Community-Based Services, HCBS)** means the delivery of **health, personal care and social services to dependent older people in their own homes and neighbourhoods**, instead of in an institution.
2. The justification is numerical. **Over two-thirds of disabled older people live in the community**, and for every person in a nursing home there are **one to three equally disabled persons living at home**.
3. The aim is **"ageing in place"** — preserve **function, autonomy and quality of life**, support the family carer, and **delay or prevent institutionalisation**.
4. Community care is **preferable but not always cheaper**. Because dependency-based eligibility brings in many people who would never have entered a nursing home (the **"woodwork effect"**), entry must be controlled — which is why **case management** became central.

B. Home-Based Services

1. **Home care has two arms**: **home health services** (skilled nursing, physiotherapy, occupational therapy, speech therapy) and **homemaker/chore services** (bathing, dressing, meal preparation, housekeeping, medication supervision, minor repairs). **Most older people need homemaking more than home health.**
2. Access is often restricted by rules requiring the person to be **"homebound" and to need intermittent skilled care**. This excludes many **custodial** dependent elders, and **personal care aides are usually not funded**.
3. **Home-based medical care**: domiciliary visits by the general practitioner or geriatrician, with rapid specialist access during crisis. **Hospice care at home** is a key component, though home hospice **cannot provide continuous 24-hour personal care**.

C. Day and Community Services

1. **Adult day care** provides supervised **recreational and restorative activity**; some centres are **dementia-specific**. Its greatest value is **respite for the primary carer**. Most programmes serve a client **2–3 days a week**.
2. **Adult day health centre** = day care **plus nursing, physiotherapy and social work**. The **day hospital** is a more medical model offering nearly all hospital services on an **ambulatory basis**, with emphasis on **rehabilitation**.
3. **Transportation is the universal problem** of all day programmes — costly, time-consuming, and it restricts catchment area.
4. Other community programmes: **congregate and home-delivered meals (Meals-on-Wheels), senior centres** (the most used service, aimed at the *well* elderly), **telephone reassurance, emergency alarm systems, respite care, caregiver support groups, home modification and repair, transport, legal aid and ombudsman services**.

D. Residential and Transitional Alternatives

1. **Assisted living** — the resident is a **tenant** with a **lockable single unit**, own bathroom and minimal cooking facility; **autonomy and privacy preserved**, services brought in. **Adult foster homes** take usually **≤5 residents** in a family-like setting.

2. **Intermediate and transition care** — home-based intermediate care, community rehabilitation, falls-prevention services, memory clinics and **post-hospital transition programmes** that provide time-limited rehabilitation and **reduce the risk of entering an institution**.

E. Assessment, Case Management and Integration

1. Entry requires a **multidisciplinary comprehensive geriatric assessment** at home or in hospital, which determines **eligibility** and matches the person to services. Such teams also handle **suspected elder abuse and guardianship** issues.
2. **Case management** has five components: **assessment → care plan (prescription) → authorization → coordination → monitoring**. Its types are **eligibility management, care coordination, utilization management, disease management and chronic care coordination**.
3. **Eligibility management is the commonest type and does not directly improve care**; overall, the **effectiveness of case management is not well established**. The case manager is trapped between being a **patient advocate and a gatekeeper** — two incompatible roles — and often has **responsibility without purchasing authority**.
4. **Integrated models**: **PACE (Program of All-Inclusive Care for the Elderly)** pools **capitated funding** to deliver combined health and social care to people who are **nursing-home eligible but still living in the community** — effective, but **narrowly targeted and expensive**. **"Cash and counselling"** consumer-directed schemes let disabled elders **purchase their own services, including from relatives**, obtaining more care for less money without adverse effects.

F. Family Carers and the Physician's Role

1. **Informal care is the backbone — more than 80% of all community care is informal**, mostly given by women. Formal services should **supplement, not replace**, family care; **respite is the single most important support**. Sustainability is threatened as more women enter the workforce.
2. The physician should **detect functional dependency early, refer for assessment, advocate actively for community options**, make home visits where needed, and **help develop services that are needed but locally absent**.

G. Limitations

1. Persistent weaknesses: **fragmented multiple programmes with patchy coordination, waiting lists, chronic underfunding, poor gate-keeping and weak exit strategies, transport difficulties, and very uneven geographic development** — public money spent on home care remains a fraction of that spent on institutions.

Latest Updates (2023–Present)

Global burden and WHO direction

- About **142 million older people** worldwide cannot meet their basic needs independently; **two of three older people** will need care and support; about **65% will need help with ADLs**; **unpaid informal care is roughly 80% of all long-term care** — confirming that family care is the backbone.
- The **WHO framework for an integrated continuum of long-term care** makes **support to unpaid caregivers** an explicit system pillar, alongside UHC financing and a sustainable trained workforce.
- **ICOPE handbook, 2nd edition (January 2025)** — guidance for **person-centred assessment and care pathways in primary care**, centred on **intrinsic capacity and functional ability** — is now the reference tool for **community-level screening and care planning**.

- **PAHO Policy on Long-Term Care (2025–2034)** gives Member States technical guidance to develop and expand LTC capacity, with strong emphasis on community-based delivery.

India — community-based LTC architecture

- **Operational Guidelines for Elderly Care at Health and Wellness Centres (Ayushman Arogya Mandirs)** define India's community model: the **ASHA maps the elderly population into bed-bound, restricted-mobility and mobile categories**, and separately identifies **poor and single elderly**; the **PHC Medical Officer** performs in-depth assessment and issues a **personalised care plan**; the **ANM/MPW makes weekly visits to bed-bound elderly**, with monthly visits from the sub-health centre, plus **family counselling**.
- **NPHCE** explicitly includes community LTC: health promotion, preventive services, diagnosis and management of geriatric problems (OPD and IPD), **day care services, rehabilitative services and home-based care**. At district level it provides a **geriatric clinic twice weekly, a rehabilitation unit for physiotherapy and counselling, and domiciliary visits by the rehabilitation worker for bed-ridden elderly** with family counselling on home-based care.
- Current NPHCE structure: geriatric OPD, physiotherapy and IPD services at **AAM–HWCs, PHCs, CHCs and District Hospitals**, plus **2 National Centres for Ageing (AIIMS New Delhi and Madras Medical College, Chennai) and 17 Regional Geriatric Centres**.
- **India Ageing Report 2023**: elderly will exceed **20% of the population by 2050** and outnumber children by 2046. It recommends **in-situ (at-home) ageing** through **elder creches and day-care centres**, and bringing **all old age homes under regulatory purview**.
- The **Global Observatory for Long-Term Care (2025)** records that India has **no national formal LTC system**. Community supports run through **AVYAY** — old-age homes, **physiotherapy centres and mobile medicare units**, assistive devices under **RVY**, **Elder Line (14567)**, **SACRED** and **SAGE**.
- **Community-based dementia care in India (scoping review, 2025)**: existing models include **community memory clinics, dementia day-care centres, home-based caregiver support programmes, task-sharing through mobile technology and tele-mentoring of primary care physicians**; integrating dementia care into **primary health care** is proposed as the scalable and sustainable model.

Workforce and financing of HCBS

- **CMS "Ensuring Access to Medicaid Services" final rule (22 April 2024)** — the biggest HCBS reform in years — covers **direct care worker compensation, HCBS waiting lists, grievance processes, critical incident reporting and HCBS quality reporting**, with greater payment-rate transparency. Its central provision, the **"80/20 rule"**, requires HCBS providers to spend **at least 80% of Medicaid payments on direct care worker compensation**, with **six years to demonstrate compliance**. It applies to **homemaker, home health aide and personal care services**.

Hospital-level care moved into the home

- The **Acute Hospital Care at Home (AHCAH)** initiative has been extended for five years **through 30 September 2030** under the Consolidated Appropriations Act, 2026, with operational requirements unchanged — **24/7 RN coverage, two in-person visits daily and EMS coordination**. **366 programmes across 139 health systems in 37 states** are approved. This directly serves the geriatric goal of avoiding **hospital-associated functional decline and delirium**.

Current Practice Update

Aspect	Textbook Position	Current Practice (2023–2026)

Default site of care	Balance already shifting from institution to HCBS	**Ageing in place / in-situ ageing is explicit global and Indian policy**; even **acute hospital care** is now delivered at home (AHCAH extended to 2030)
Assessment tool	Local multidisciplinary assessment for eligibility	**WHO ICOPE screening of intrinsic capacity + personalised care pathway**; in India, **ASHA-led mapping and PHC-MO care plan** at Ayushman Arogya Mandirs
Direct care workforce	Low-paid, high-turnover aides; a background problem	**Explicitly regulated** — CMS **80/20 rule** mandates ≥80% of payments to direct care worker compensation; WHO names workforce as a system pillar
Carer support	Respite described as a service	**Support to unpaid caregivers is a formal pillar** of the WHO LTC framework
Case management	Effectiveness unproven; eligibility management commonest	Shift toward **integrated, capitated, person-centred models** and **consumer-directed care**; HCBS **waitlist and quality reporting** now mandated
Day care	Recreational/restorative, respite value	Expanded to **dementia-specific day care** and, for India, proposed **elder creches / short-term care facilities**
India-specific delivery	Not part of the classical HCBS model	**NPHCE home-based care and domiciliary visits, HWC elderly-care guidelines, AVYAY, Elder Line 14567, AB PM-JAY for ≥70 years**

Exam One-Liners

- **HCBS = health + personal + social care delivered at home and in the neighbourhood.**
- **Two-thirds of disabled older people live in the community**; 1–3 equally disabled at home per nursing home resident.
- **>80% of community long-term care is informal**, mostly provided by women.
- **Homemaking is needed more often than home health.**
- **Adult day care's greatest value = respite for the carer.**
- **Day hospital = ambulatory hospital services with a rehabilitation emphasis.**
- **Transportation is the commonest limiting factor** for day programmes.
- **Case management = assessment → care plan → authorization → coordination → monitoring.**
- **Advocate versus gatekeeper** — the central conflict of the case manager's role.
- **PACE** = capitated integrated care for **nursing-home-eligible people living in the community**.
- **"Cash and counselling"** = consumer-directed purchase of services, including from relatives.
- **"Woodwork effect"** = dependency-based eligibility draws in people who would never have been institutionalised.
- **India's community LTC**: ASHA maps **bed-bound / restricted / mobile** elderly; ANM makes **weekly visits to bed-bound**; PHC-MO gives the **personalised care plan**.
- **NPHCE** includes **day care, rehabilitation and home-based care**, with **2 National Centres for Ageing and 17 Regional Geriatric Centres**.
- **CMS 80/20 rule** = ≥80% of HCBS payments must go to **direct care worker compensation**.
- **Acute Hospital Care at Home extended to 30 September 2030.**

Q3. Home-Based Care in the Elderly

Components, eligibility, home visit yield, technology, evidence and limitations — 15 marks

A. Definition and Historical Context

1. **Home-based care** is the delivery of **medical, nursing, rehabilitative, personal and social care in the patient's own residence**, so that a dependent older person can remain at home instead of moving to an institution.
2. Historically, medicine was practised at home — in the mid-1900s about **40% of all patient–physician encounters took place at home**, falling to **under 1% by 1980**. Home healthcare began growing again in the **1980s** as an organised, multidisciplinary service.
3. The present model is delivered mainly by **nurses, therapists and personal care providers**; **physician house calls remain grossly under-utilised** for frail older adults.

B. Components of Home-Based Care

1. **Home health services** — skilled **nursing, physiotherapy, occupational therapy and speech therapy**. Typical uses are **medication management, disease assessment, wound care, home safety evaluation and patient/family education**.
2. **Personal care and homemaker services** — bathing, dressing, toileting, laundry, housekeeping, meal preparation. These are what most older people actually need, yet they are **usually not funded**, so the cost falls on the family.
3. **Home-based palliative and hospice care** provides nursing, therapy, social work and family support at home; **home hospice cannot provide continuous 24-hour personal care**.
4. **Support services**: home-delivered meals, transport, legal aid, individual case management and social services — often on a **sliding-fee scale**, with availability varying widely by locality.
5. **Home-based intermediate care and community rehabilitation**: community matron services, falls-prevention services, memory clinics and **community geriatric teams delivering comprehensive geriatric assessment at home**.

C. Eligibility and Process of Care

1. Entry usually requires a **physician's order**, documentation that the person is **"homebound"**, and need for **intermittent skilled care by a nurse or therapist**. The definition of "homebound" is **vague**, and large numbers of dependent but "custodial" elders are therefore **excluded**.
2. The average enrollee receives about **30 visits**; payment has shifted from **fee-for-service to prospective payment** per certification period, which shortened episodes but increased the number of recipients.
3. Quality is tracked by a **structured outcome dataset (OASIS)** completed at defined stages, generating around **28 functionally based quality measures** (e.g. improvement in bathing or toileting) plus **emergency and hospital utilisation**, which are publicly reported.

D. Clinical Content of the Home Visit

1. The **yield of a home visit** is unique: verification of diagnoses, **direct inspection of all medicines**, nutrition, observation of **ADL/IADL performance in the real environment**, cognition and mood, **carer capability and strain**, detection of **elder abuse or neglect**, and the true social situation.
2. **Home safety and environmental assessment** — the occupational therapist evaluates the **person–environment fit** and recommends **home modification** (grab bars, railings, raised toilet seat, shower chair, lighting), **assistive devices** (long-handled reacher, sock aid, adapted utensils) and **task simplification**. Poor environmental fit increases risk of **dependency, falls and injury**.

3. A **restorative philosophy is essential**. If carers do everything for the person, the result is **deconditioning, dependency, contractures and the sequelae of immobility** — the older person must be encouraged to perform their own ADLs at home.

E. Evidence and Effectiveness

1. **House calls are effective for homebound, chronically ill patients**, with the clearest benefit in **chronic relapsing diseases such as congestive cardiac failure and COPD**; regular professional visits **improve disease control and reduce hospitalisation**, producing societal cost savings.
2. **Preventive home visitation** aims at **primary prevention** (immunisation, exercise), **secondary prevention** (detecting untreated problems) and **tertiary prevention** (improving medication use), to delay functional decline and institutionalisation. **Benefits have not been definitively confirmed, so routine preventive home visits cannot be recommended as standard practice** — though home visits remain valuable within outpatient and inpatient programmes.
3. Home care must include a strategy for **long-term follow-up and, often, formal case management**, so that needs and services are correctly matched and followed through.

F. Technology in Home Care

1. Technology has widened what is possible at home: **mobile radiology and laboratory services, intravenous access and infusion devices** (allowing IV therapy without hospital transfer), **portable ECG, point-of-care testing and telemonitoring**. Monitoring studies, mostly in **chronic cardiac disease**, show **reduced hospitalisation, mortality, healthcare utilisation and exacerbations**.
2. **Smart home technology**: ambient light and **bed-occupancy sensors** with **passive infrared sensors** guide the older person safely to the toilet at night, prompt medication, detect **wandering in dementia** and **detect falls** with automatic alerts. Caution — the smart home is like **an extra carer that never tires, but it can never supply human care** and must not be used as a cheap substitute.

G. Family Carers and Limitations

1. **Informal family care remains the backbone** — home services should **bolster, not replace**, family effort, with **respite, carer education and psychological support**. Main limitations: **restrictive eligibility, no funding for personal/custodial care, fragmentation between programmes, workforce shortage and turnover, transport difficulties and carer burnout**.

Latest Updates (2023–Present)

Hospital-level care at home

- The **Acute Hospital Care at Home (AHCAH)** initiative is now durable — extended for five years **through 30 September 2030** under the Consolidated Appropriations Act, 2026, with unchanged requirements of **24/7 registered nurse coverage, two in-person visits daily and EMS coordination**. **366 programmes across 139 health systems in 37 states** are approved.
- **Meta-analytic evidence in older adults**: a systematic review and meta-analysis of RCTs to April 2024 (**11 RCTs, 3,301 patients**) found hospital-at-home gave a **significantly shorter length of stay** than traditional hospital care. A separate synthesis of **15 trials** found **early-discharge hospital-at-home associated with lower mortality (low-certainty evidence) and shorter treatment duration**, with inconclusive effects on readmission and mental status. For **admission-avoidance** hospital-at-home, pooled data suggest **reduced readmission (RR 0.76, 95% CI 0.58–1.01)**, most marked in **respiratory disease (RR 0.53, 95% CI 0.39–0.73)**, with **no significant difference in mortality**.

WHO framework

- About **142 million older people worldwide cannot meet basic needs independently**; **two of three** will need care and support; roughly **65% will need help with ADLs**; and **unpaid informal care accounts for about 80% of long-term care** — the empirical basis for insisting that home care must support the family.
- The WHO long-term care framework makes **supporting unpaid caregivers** an explicit pillar. The **ICOPE handbook, 2nd edition (January 2025)** provides **person-centred assessment and care pathways for primary care and community settings**, covering the continuum through to long-term care and carer support.

India — home-based elderly care

- Under the **Elderly Care at Health and Wellness Centre (Ayushman Arogya Mandir) guidelines**, the **ASHA maps the elderly into bed-bound, restricted-mobility and mobile categories** and identifies **poor and single elderly**; the **PHC Medical Officer** provides in-depth assessment and a **personalised care plan**; the **ANM/MPW visits bed-bound elderly weekly**, with monthly visits from the sub-health centre and **family counselling**.
- **NPHCE** funds **day care services, rehabilitative services and home-based care**, and district hospitals provide **domiciliary visits by the rehabilitation worker for bed-ridden elderly** with counselling of family members. Structurally there are **2 National Centres for Ageing (AIIMS New Delhi, Madras Medical College Chennai) and 17 Regional Geriatric Centres**.
- **Burden of home-based palliative need (LASI analysis, BMC Palliative Care 2024)**: **one in eight Indians aged ≥60 years (12.2%)** has supportive or palliative care needs, with higher odds in those **aged ≥70 years (AOR 1.86), women (1.33), rural residents (1.72)**, and those with **cancer (2.84), respiratory disease (3.14) or stroke (2.58)**; needs were lower with higher education and health insurance.
- **Coverage is highly uneven**: parliamentary data of December 2025 show **Kerala recorded about 650,000 home visits in eight months to October 2025** and Maharashtra about 167,000, while **Uttar Pradesh reported about 42,500 visits** and **Bihar delivered services in only six districts**.
- **Workforce**: the **PM-SPECIAL (Training of Geriatric Care Givers)** scheme of the Ministry of Social Justice and Empowerment creates a professional cadre through four courses — **Geriatric Caregiver (Institutional and Home Care), Elderly Caretaker (Non-Clinical), Elderly Care Companion and Wellness Therapist for the Elderly** — with registration through the **Skill India Digital Hub**.
- The **Global Observatory for Long-Term Care (2025)** notes India has **no national formal long-term care system**; **AB PM-JAY (≥70 years) covers hospitalisation, not home care**, which remains largely out-of-pocket.

Home care workforce regulation elsewhere

- The CMS **"80/20 rule"** requires HCBS providers to spend **at least 80% of Medicaid payments on direct care worker compensation**, with six years to comply, applying to **homemaker, home health aide and personal care services** — intended to stabilise a workforce whose turnover is the greatest threat to home care access.

Current Practice Update

Aspect	Textbook Position	Current Practice (2023–2026)
Scope deliverable at home	Skilled nursing, therapy, IV therapy, mobile imaging and laboratory	**Full inpatient-level acute care at home** (AHCAH secured to 2030) with defined operating standards

Evidence for hospital-at-home	Emerging; house calls beneficial in CCF and COPD	Meta-analyses in older adults: **shorter length of stay, lower mortality (low certainty) for early discharge, reduced readmission especially in respiratory disease**
Preventive home visits	Benefits unconfirmed → not recommended routinely	Still not universal, but replaced by **risk-stratified targeting** (ASHA bed-bound mapping) and **ICOPE-based screening**
Home care workforce	Aides largely untrained, high turnover	**80/20 compensation rule** (US); **PM-SPECIAL NSQF-certified geriatric caregiver courses** (India)
Technology	Telemonitoring and smart homes promising	**Routine remote monitoring, digital tools and tele-mentoring of primary care physicians** for home dementia care
Palliative care at home	Hospice model; home hospice cannot give 24-hour care	India: home-based palliative care is **explicit policy**, but **1 in 8 older Indians has palliative need** against sharply unequal state coverage
Carer support	Respite described as a service	**Support to unpaid caregivers is a formal pillar** of WHO's long-term care framework

Exam One-Liners

- **Home-based care = care delivered where the patient lives, not where the doctor works.**
- **40% of encounters at home in mid-1900s → <1% by 1980**; revival from the 1980s.
- **Physician house calls remain the most under-used element** of home care.
- **Eligibility triad**: physician order + **homebound status** + need for **intermittent skilled care**.
- **"Homebound" is a vague definition** that excludes many custodial-need elders.
- **OASIS** = the structured outcome dataset for home health quality measurement.
- The **medicine-bag review** and observation of **ADLs in the real environment** are the unique yield of a home visit.
- **OT home hazard assessment** = established falls-prevention intervention.
- **Doing everything for the patient causes deconditioning** — home care must be restorative.
- **House calls benefit CCF and COPD most**, reducing hospitalisation.
- **Routine preventive home visits are not recommended** — benefit unconfirmed.
- **Smart home = an extra carer that never tires, but never a substitute for human care.**
- **AHCAH extended to 30 September 2030**; requires **two visits daily and 24/7 RN cover**.
- **India**: ASHA maps bed-bound / restricted / mobile; **ANM visits bed-bound weekly**.
- **1 in 8 Indians aged ≥60 (12.2%) has supportive or palliative care needs.**
- **PM-SPECIAL** = MoSJE scheme creating a trained **geriatric caregiver cadre**.

Q4. Day Hospitals and Day Care Centres for Older Adults

Need for day hospitals in geriatric practice and the practical running of a day care centre — 15 marks

A. Definitions and Types

1. A **geriatric day hospital** is an **outpatient facility where older patients attend for a full or near-full day and receive multidisciplinary assessment, treatment and rehabilitation "under one roof"** — virtually all hospital services made available on an **ambulatory basis**, with emphasis on **physiotherapy and occupational therapy**.
2. Four levels must be distinguished: **senior centre** (recreational, for the *well* elderly; the most frequently used community service); **adult day care** (social model — supervised recreational and restorative activity for people with limited function; some dementia-specific); **adult day health centre** (day care plus nursing, physiotherapy and social work); and **geriatric day hospital** (medical model — full multidisciplinary CGA, treatment and rehabilitation).
3. The concept descends from **Marjorie Warren's principle** that every older person deserves **comprehensive assessment and a trial of rehabilitation before being consigned to long-term institutional care**. The British day hospital model was subsequently exported worldwide.

B. Need for Day Hospitals in Geriatric Practice

1. **Demographic pressure with shrinking inpatient capacity** — rising numbers of frail, disabled older people at a time when **acute beds are being reduced and lengths of stay shortened**, creating a gap between hospital and home that the day hospital fills.
2. **Avoids the hazards of admission** — hospitalisation causes **disorientation, delirium, deconditioning ("pyjama paralysis") and unnecessary institutionalisation**, particularly in dementia. The day hospital allows treatment **without occupying a bed**.
3. **Focal point for entry into the geriatric care system** — provides **multidimensional assessment coupled with an intervention** (rehabilitation, counselling or placement decision), which is what distinguishes assessment programmes that work from those that do not.
4. **Prevents admission, facilitates early discharge and reduces inpatient bed use** by continuing rehabilitation after the patient goes home.
5. Offers **intensity and rapidity of assessment** for the frail or acutely unwell — an advantage over office-based outpatient assessment — while **avoiding the cost of an inpatient stay**.
6. **Supports the carer.** Day attendance is the main organised form of **respite**; when carer strain is high, **day care or respite is preferable to admission**, which itself carries a higher risk of transition into permanent care.
7. Fills the **middle position in the rehabilitation spectrum**, between the inpatient rehabilitation unit and home-based rehabilitation — the latter suits only the **least disabled who have good live-in support**.

C. Services Provided

1. **Core services**: multidisciplinary CGA, medical review and **rationalisation of drugs**, physiotherapy, occupational therapy, speech and swallowing therapy, nursing procedures (dressings, injections, catheter care), investigations, dietetics, podiatry, social work and psychology.
2. **Specialist programmes**: **falls and balance clinic, post-stroke and post-fracture rehabilitation, continence clinic, memory clinic, Parkinson's and leg-ulcer clinics, anticoagulation review, dementia day respite and palliative day care**.

D. Running a Day Centre — Practical Organisation

1. **Team**: geriatrician-led **interdisciplinary team** — nurse, physiotherapist, occupational therapist, speech therapist, social worker, dietitian, pharmacist, psychologist, care assistants and a coordinator. The **interdisciplinary team conference is the critical step**: it pools perspectives, sets priorities, prevents duplication, and must involve the **patient and carer to preserve choice**.
2. **Physical design**: ground-floor or lift access, **barrier-free layout**, handrails, non-slip floors, accessible toilets, good lighting with **large clear signage and contrasting colours** (dementia-friendly), therapy gymnasium, **ADL kitchen and bathroom for training**, treatment room, dining area, a quiet **de-stimulation space**, rest area and an established link to the parent hospital.
3. **Transport is the single greatest operational determinant** — difficult to arrange, expensive and time-consuming. **Special vans are needed**, and to avoid exhausting travel times the **catchment area must be kept small**.
4. **Attendance pattern**: most clients attend **2–3 days a week rather than 5**, which increases efficiency and allows more patients to be served.
5. **Process**: explicit **referral criteria and sources** (GP, hospital discharge, emergency department, self/family), initial CGA with **goal setting**, an individualised care plan with defined therapy targets, **periodic review and clear discharge criteria** to prevent "silting up" of places, followed by structured hand-back to primary care with **long-term follow-up and case management**.
6. **Governance and outcome measurement**: collect data at **entry and at discharge** to prove effectiveness and permit comparison between programmes — **Barthel Index, FIM, Berg Balance Scale, Timed Up-and-Go, MMSE/MoCA, GDS and carer burden scales** — with audit and standardised documentation.

E. Evidence and Limitations

1. **Evidence**: day hospital attendance is clearly **better than no comprehensive care** — less death, institutionalisation and deterioration in ADLs — but shows **little advantage over other comprehensive geriatric services or home-based care, and may cost more**. In old-age psychiatry the supporting literature was sparse and **many day hospitals were decommissioned**, with voluntary, charitable and private day care plus **crisis resolution home treatment teams** taking their place.
2. **Limitations**: transport dependence, high staff intensity and cost per attendance, reliance on a carer to prepare and hand over the patient, blocked places from unclear discharge criteria, and **very poor coverage in low- and middle-income countries**, where the mismatch between rehabilitation need and available resource is greatest.

Latest Updates (2023–Present)

Evidence for the medical day hospital

- The definitive synthesis remains the **Cochrane review (Brown, Forster, Young, Crocker, Benham, Langhorne)**: **16 trials, 3,689 participants, 37 day hospitals**. There is **low-quality evidence that day hospitals are effective compared with no comprehensive care** for the combined outcome of **death or poor outcome and for deterioration in ADL**, with **no clear advantage over other forms of medical care provision**. Economic value relative to other services is unclear; the authors concluded the findings **do not support closure of day hospital services but do support exploring alternative systems** for delivering equivalent or superior comprehensive care.

Evidence for adult day services / day care centres

- An **updated systematic review covering 2015–2025** strengthens the case: regular adult day service use is associated with **reduced caregiver stress, improved mood and physiological**

stress reduction; on attendance days caregivers report **lower negative affect and higher positive affect**. Participation is linked to **delayed nursing home placement and consequent cost savings**. Utilisation remains suboptimal because of **financial constraints, limited awareness, caregiver reluctance and poor availability**; facilitators include **caregiver education, culturally tailored programming, financial assistance and high-quality staff engagement**.

- Day services can **delay or prevent nursing home placement by supporting informal caregiving** — caregivers experiencing stress and burden are more likely to institutionalise a relative with dementia.
- **Carer support should be built into the day centre**: the **ADS Plus multisite cluster-randomised trial (Gerontologist, 2024)** across **34 adult day services** tested a staff-delivered caregiver programme providing **dementia education, support and validation, referrals and linkages, and strategies for care challenges and self-care** over 12 months, built on five components — **care management, referral/linkage, dementia education, situational counselling and stress reduction, and skills to manage behavioural symptoms**.
- **Dementia now dominates the clientele**: in Taiwan, which is scaling up **a day care centre in every junior high school district**, about **70% of day-care clients are persons with dementia**.

WHO direction and India

- The **ICOPE handbook, 2nd edition (January 2025)** makes the day centre a natural site for **intrinsic capacity screening and pathway delivery**; **support to unpaid caregivers** is an explicit WHO pillar.
- **NPHCE** already mandates **day care services and rehabilitative services** alongside home-based care, with districts linked to Regional Geriatric Centres.
- The **India Ageing Report 2023 (UNFPA–IIPS)** recommends promoting **in-situ ageing** by creating **short-term care facilities such as elder creches and day-care centres**.
- Under **AVYAY**, the **Integrated Programme for Senior Citizens (IPSrC)** funds basic amenities, medical care and recreation for indigent elders; the **Senior Citizen Homes (SrCH)** scheme funds running and maintenance of homes; and **Regional Resource and Training Centres (RRTCs)** act as nodal technical agencies.
- **Kerala's models are nationally cited**: **Sayamprabha Homes** provide **day-care facilities in partnership with local self-government institutions** (initially about **70 centres**), offering counselling, yoga, meditation and peer companionship; **Vayomithram** provides mobile clinics, free medicines, palliative care and a help desk for those above 65 in municipal and corporation areas, each unit staffed by a **medical officer, staff nurse and junior public health nurse**, with dementia specifically covered.
- An **EAC-PM working paper** highlights Kerala's **Vayomithram and Kudumbashree** elder-care models for linking training with employment, while noting that **AVYAY has limited reach** and that **workforce development remains fragmented because there is no standardised certification system**.

Competing service models

- The **Acute Hospital Care at Home** initiative has been extended to **30 September 2030**; together with **virtual wards and same-day ambulatory frailty services**, these are increasingly the "alternative systems" the Cochrane authors called for.

Current Practice Update

Aspect	Textbook Position	Current Practice (2023–2026)

Status of the day hospital	A core, established component of geriatric services	Many **decommissioned**; role reframed as a **hub for CGA and frailty management**, with hospital-at-home, virtual wards and ambulatory frailty units substituting
Evidence base	Benefit over no treatment; little advantage over other comprehensive services	**Confirmed by Cochrane (16 trials, 3,689 participants, low certainty)** — closure not supported, but **alternative delivery systems should be explored**
Adult day care benefit	Chiefly respite value for the carer	Recent evidence adds **reduced caregiver stress and stress biomarkers, delayed nursing home placement and cost savings**
Carer support	Incidental to the patient's attendance	Should be a **structured, staff-delivered embedded programme** (ADS Plus trial, 34 sites)
Case mix	Some programmes dementia-specific	**Dementia now dominates** day-centre attendance (~70% in Taiwan); dementia-friendly design is standard
India	Not part of the classical model	**NPHCE day care and rehabilitation; elder creches/day centres** recommended by India Ageing Report; **AVYAY–IPSrC** funding; **Kerala Sayamprabha and Vayomithram** as replicable models
Outcome measurement	Entry-versus-discharge comparison	Move toward **uniform outcome sets and Goal Attainment Scaling**; caregiver outcomes still under-collected

Exam One-Liners

- **Day hospital = multidisciplinary rehabilitation "under one roof" on an ambulatory basis.**
- **Senior centre = well elderly; day care = functionally limited; day health centre = intermediate; day hospital = medical model.**
- **Marjorie Warren's principle**: assessment and rehabilitation *before* institutionalisation.
- **Assessment must be coupled with an intervention** — that is what makes geriatric assessment effective.
- **Day hospital prevents admission, shortens stay and facilitates early discharge.**
- **Respite for the carer may be the single most important output** of day attendance.
- **Transportation is the commonest limiting factor** — special vans, small catchment area.
- **Most clients attend 2–3 days a week, not 5.**
- **The interdisciplinary team conference is the critical step** in the process of care.
- **Clear discharge criteria prevent "silting up"** of day hospital places.
- **Cochrane**: 16 trials, 3,689 participants — better than no comprehensive care, **no clear advantage over other comprehensive services**; **closure not supported**.
- **Adult day services delay nursing home placement** and reduce caregiver stress.
- **ADS Plus** = embedding structured caregiver support inside the day centre.
- **NPHCE includes day care and rehabilitative services** in its service package.
- **India Ageing Report 2023** recommends **elder creches and day-care centres**.
- **Kerala: Sayamprabha Homes and Vayomithram** are the model Indian programmes.

Q5. House Call Programme (Home-Based Medical Care)

Target population, structure, conduct of the visit, evidence and ethical issues — 15 marks

A. Definition and Rationale

1. A **house call programme** is the organised delivery of **physician-led (or physician–nurse-practitioner-led) medical care in the patient's own home**, for people who cannot readily reach a clinic. It is the medical component of home-based care, distinct from home health nursing.
2. In the **mid-1900s about 40% of all patient–physician encounters occurred at home**, falling to **under 1% of healthcare visits by 1980**. Home healthcare revived in the **1980s**, but **physician house calls remain under-utilised**.
3. Justification: the **homebound frail elderly** are the highest-cost, highest-need group; the home visit gives **diagnostic information obtainable nowhere else**, avoids the hardship of transport and the hazards of hospital (delirium, deconditioning, iatrogenesis), and strengthens **continuity and carer support**.

B. Target Population and Indications

1. Ideal candidates: **homebound with functional dependency; frailty and multimorbidity; advanced dementia; recurrent admissions; recent hospital discharge needing transition care; end-of-life patients; repeated clinic "no-shows"; suspected self-neglect, hoarding or elder abuse; and carer strain**.
2. **Targeting matters** — the sickest and most frail, at highest risk of hospitalisation, gain the most; applied indiscriminately, the yield falls sharply.
3. Four classic indications: **management of acute or chronic illness at home; coordination of post-hospitalisation transition of care; comprehensive health/functional assessment; and end-of-life care**.

C. Structure of the Programme

1. **Team**: physician or nurse practitioner leading an interdisciplinary team — **nurse, social worker, physiotherapist, occupational therapist, pharmacist and care coordinator** — with **24-hour telephone access, rapid response to urgent needs, embedded palliative care and explicit support to family caregivers**.
2. **Logistics**: a **defined, small geographic catchment** (travel time is the main cost driver), visits **clustered by locality**, a realistic panel size per clinician, staff safety protocols and structured **communication back to the primary physician**.
3. **Linkages**: formal tie-ups with a **home health agency, pharmacy, mobile laboratory and imaging services, hospice and social services**, so that the house call translates into delivered care.
4. **The house-call bag**: sphygmomanometer, stethoscope, pulse oximeter, glucometer, thermometer, torch, otoscope and ophthalmoscope, tuning fork, portable ECG, point-of-care and urine dipstick tests, dressings, catheters, syringes, gloves and sanitiser, sharps container, emergency drugs and **assessment forms/scales**.
5. **Technology support**: **mobile radiology and laboratory services, intravenous access and infusion devices** (allowing IV therapy without transfer), **portable ECG, point-of-care testing and telemonitoring**.

D. Conducting the Visit

1. Use a **structured framework** covering **Immobility, Nutrition, Home environment, Other people (carers), Medications, Examination, Safety, Spiritual health and Services** — the widely taught **INHOMESSS** checklist.
2. **High-yield elements unique to the home**: the **"brown-bag" review of every medicine actually in the house**, **observed ADL and IADL performance in the real environment**, inspection of the kitchen and food stores, **skin and pressure areas**, continence, cognition and mood, **falls hazards**, and detection of **elder abuse, neglect or self-neglect**.
3. **Home safety and environmental modification** with occupational therapy input — grab bars, railings, raised toilet seat, shower chair, lighting, decluttering and assistive devices — correcting the **person–environment mismatch** that drives falls and dependency.
4. **Close the visit properly**: agreed goals, **rationalisation of drugs**, advance care planning and documentation of preferences, a written care plan, follow-up schedule, escalation plan, and a **strategy for long-term follow-up with formal case management**.

E. Evidence and Outcomes

1. House calls are **effective for the aged and chronically ill homebound**, with clearest benefit in **chronic relapsing diseases such as congestive cardiac failure and COPD**; regular professional visits **improve disease control and reduce hospitalisation**, generating societal cost savings.
2. **Preventive home visitation** (primary — immunisation, exercise; secondary — detecting untreated problems; tertiary — improving medication use) aims to delay disability and institutionalisation, but **benefits are not definitively confirmed and it cannot be recommended as routine practice**. Home visits retain value **within outpatient and hospital-discharge programmes** — the hospital–home assessment model shortens stay and allows evaluation of the home itself.

F. Limitations and Ethical Issues

1. **Practical limitations**: travel time and inefficiency, limited on-site diagnostics, physician reluctance and workforce shortage, staff safety, weak reimbursement, and the persistently **vague "homebound" criterion**.
2. **Ethical issues**: the clinician is a **guest, not the host** — confidentiality within a shared household, professional boundaries and hospitality, **autonomy versus safety** when living conditions are unsafe, capacity and consent, and balancing the wishes of the patient against those of the family.
3. **In India**, house calls are largely **private and out-of-pocket** or delivered by NGOs and state programmes; the public backbone is **domiciliary visiting by rehabilitation workers, ANMs and ASHAs** rather than by physicians.

Latest Updates (2023–Present)

> **Independence at Home (IAH) — the reference house call programme**
>
> - IAH was authorised under **Section 3024 of the Affordable Care Act**, extended repeatedly, and **concluded on 31 December 2023**. It tested a payment-incentive and service-delivery model of **home-based primary care for Medicare fee-for-service beneficiaries with multiple chronic illnesses and functional limitations**, examining whether comprehensive, coordinated, continuous, accessible home care reduces preventable hospitalisations, readmissions and ED visits.
> - **Performance Year 10 results (published June 2025)**: only **one participant remained — Northwell Health House Calls**. Its beneficiaries' expenditures were **34.0% below the spending target (US$7.6 million), an average reduction of US$18,727 per beneficiary**

(per-beneficiary-per-month target US$6,436 versus actual US$4,249), earning an incentive payment of about **US$5.1 million**.

- The **six IAH quality measures** are the model set for any house-call programme: follow-up contact **within 48 hours of a hospital admission, discharge or ED visit**; **medication reconciliation in the home within 48 hours** of discharge or ED visit; **annual documentation of patient preferences**; **all-cause 30-day readmissions**; **hospital admissions for ambulatory-care-sensitive conditions**; and **ED visits for ambulatory-care-sensitive conditions**.

The evidence is genuinely mixed — know both sides

- **Positive**: earlier IAH reporting showed a **23% reduction in hospitalisations, a 27% decrease in 30-day readmissions and savings of US$111 per beneficiary per month (US$70 million over three years)**, while a large systematic review (**nine studies, N = 46,154**) of home-based primary care for homebound older adults found **fewer hospitalisations, fewer hospital bed-days, fewer ED visits and fewer long-term care admissions and bed-days**.
- **Cautionary**: a **CMS update of January 2023** concluded there is **"no compelling evidence"** that primary care delivered at home reduces Medicare spending or hospital use, though it may lead to better — or at least different — care. The independent evaluation found that **simply providing primary care in someone's home does not reduce total hospitalisations**, while patients and caregivers report **high satisfaction**.
- There is **moderate-strength evidence that the most complex patients — those at highest risk of hospitalisation or most frail — have the greatest potential to benefit**, which is why patient selection is central.
- Successful home-based primary care practices share five features: **interdisciplinary teams, integration of behavioural care and social supports, rapid response to urgent and acute needs, embedded palliative care, and active support of family caregivers**.

Interface with hospital-level home care, and India

- The **Acute Hospital Care at Home** initiative has been extended five years **to 30 September 2030**, with **24/7 nursing availability and two in-person visits daily**, and **366 programmes across 139 health systems in 37 states** approved — house-call teams increasingly act as the step-down service for these patients.
- India's public equivalent runs through primary care: under the **Ayushman Arogya Mandir elderly-care guidelines**, the **ASHA maps elderly people as bed-bound, restricted-mobility or mobile**; the **PHC Medical Officer** provides in-depth assessment and a **personalised care plan**; and the **ANM/MPW visits bed-bound elderly weekly**. **NPHCE** funds **domiciliary visits by the rehabilitation worker for bed-ridden elderly**.
- **Need is large and coverage uneven**: a LASI-based analysis found **one in eight Indians aged ≥60 years (12.2%) has supportive or palliative care needs**. Parliamentary data of December 2025 show **Kerala recorded about 650,000 home visits in eight months to October 2025** against **about 42,500 in Uttar Pradesh** and services in **only six districts of Bihar**.
- The **Global Observatory for Long-Term Care (2025)** records that India has **no national formal long-term care system**. The **PM-SPECIAL scheme** is building a trained cadre through courses including **Geriatric Caregiver (Institutional and Home Care)**.

Current Practice Update

Aspect	Textbook Position	Current Practice (2023–2026)
Status of house calls		A defined discipline of **home-based primary care** with team standards; but the flagship **IAH**

	Effective but under-utilised; a small physician/NP home-care industry emerging	**demonstration ended December 2023** with only one practice remaining
Evidence	House calls reduce hospitalisation in CCF and COPD	**Mixed** — large observational and review data favourable, but CMS/independent evaluation found **no compelling reduction in spending or hospital use overall**; benefit concentrated in the **sickest and frailest**
Quality measurement	Not specified for house calls	Six explicit measures — **48-hour follow-up contact, 48-hour home medication reconciliation, annual documentation of preferences, 30-day readmissions, ACSC admissions and ACSC ED visits**
Preventive home visits	Not recommended routinely	Still not routine; replaced by **risk-stratified targeting** and **ICOPE-based screening**
Scope of care at home	Mobile imaging, IV therapy, point-of-care testing	Extends to **full inpatient-level acute care at home**, secured to 2030
India	Not addressed	**NPHCE domiciliary visits + weekly ANM visits to bed-bound elderly**; home-based palliative care is policy, but delivery is **grossly unequal between states**

Exam One-Liners

- **House call = organised physician-led medical care delivered in the patient's home.**
- **40% of encounters at home in the mid-1900s → <1% by 1980.**
- **Four indications**: acute/chronic illness, post-hospital transition, assessment, end-of-life care.
- **INHOMESSS** = Immobility, Nutrition, Home environment, Other people, Medications, Examination, Safety, Spiritual health, Services.
- The **"brown-bag" medication review** and **observed ADLs** are the unique yield of a home visit.
- **Benefit is greatest in CCF and COPD**, where visits reduce hospitalisation.
- **Routine preventive home visits are not recommended** — benefit unconfirmed.
- **Target the frailest** — moderate evidence that the sickest gain most.
- **IAH quality set**: 48-hour follow-up contact, 48-hour home medication reconciliation, documented preferences, 30-day readmission, ACSC admissions, ACSC ED visits.
- **IAH concluded 31 December 2023**; Year 10 showed expenditures **34% below target**.
- **CMS (2023): "no compelling evidence"** that home-based primary care alone reduces spending or hospital use.
- **Five features of successful programmes**: interdisciplinary team, behavioural/social supports, rapid urgent response, palliative care, caregiver support.
- **Travel time and catchment size are the main cost drivers.**
- The clinician on a house call is a **guest, not the host** — autonomy usually outweighs the clinician's view of safety.

Q6. Components of the Hospital Elder Life Program (HELP)

Target risk factors, core protocols, staffing, adapted models and evidence — 15 marks

A. Definition and Underlying Principle

1. **HELP is the original evidence-based, protocol-driven, multicomponent non-pharmacological programme** designed to **prevent delirium and cognitive and functional decline** in hospitalised older patients, developed by **Sharon Inouye** and first reported in 1999.
2. Its founding principle: **delirium is multifactorial**, so prevention requires a **multicomponent intervention targeting several modifiable risk factors simultaneously** — not a single drug. **Non-pharmacological measures are the mainstay of delirium prevention.**
3. Delivery model: **trained volunteers** working under an **interdisciplinary team**, with **daily protocol-driven visits** to patients identified as high risk on admission (usually aged ≥70 years).

B. The Six Target Risk Factors

1. HELP addresses six modifiable risk factors: **cognitive impairment, sleep deprivation, immobility, visual impairment, hearing impairment and dehydration** — with **medication review** as an essential accompanying element.

C. The Six Core Intervention Protocols (key examination content)

1. **Orientation protocol** — orientation board, repeated reorientation to time, place and person, and **therapeutic cognitively stimulating activities** (word games, reminiscence, discussion) three times daily for patients with cognitive impairment.
2. **Non-pharmacological sleep protocol** — warm drink, relaxation music, back massage, noise reduction, rescheduling of night-time medications and procedures; **avoids sedative-hypnotics**.
3. **Early mobilisation protocol** — **ambulation or active range-of-motion exercises three times a day**, and **minimising "tethers"** (urinary catheters, intravenous lines, restraints) that immobilise the patient.
4. **Vision protocol** — provision of **spectacles, magnifiers, large-print materials, adaptive illuminated equipment**, and reinforcement of their daily use.
5. **Hearing protocol** — **hearing aids and portable amplifiers, cerumen disimpaction** and special communication techniques.
6. **Dehydration/feeding protocol** — **early recognition of volume depletion, encouragement of oral fluids, volume repletion and feeding assistance** at mealtimes.

D. Structural and Staffing Components

1. **Core personnel**: a **geriatrician (or geriatric nurse specialist), an Elder Life Specialist, an Elder Life Nurse Specialist, a programme director and trained volunteers**, supported by therapy, pharmacy, nutrition and social work.
2. **Systematic case-finding**: screening of all admissions against **eligibility and risk criteria**, followed by enrolment and an individualised set of protocols matched to the patient's specific risk factors.
3. **Geriatric interdisciplinary rounds and nurse-specialist consultation**, provider education, and **linkage with community services at discharge**.
4. **Quality assurance**: continuous tracking of **protocol adherence**, delirium rates, falls and length of stay, with volunteer training, supervision and retention as a defined programme function — adherence is the main determinant of effectiveness.
5. **Family engagement** — families should remain at the bedside to **reassure and reorient, facilitate oral intake and encourage mobility**; risk of postoperative delirium should be discussed as part of **informed consent** for surgery.

E. Adapted Models

1. **mHELP (modified HELP)** — three **nurse-delivered daily protocols**: **orienting communication, oral hydration and nutritional assistance, and early mobilisation** — designed for older patients undergoing elective abdominal surgery.
2. **ABCDEF bundle** for the ICU — **A**ssess/prevent/manage pain; **B**oth spontaneous awakening and breathing trials; **C**hoice of analgesia and sedation; **D**elirium assess, prevent and manage; **E**arly mobility and exercise; **F**amily engagement and empowerment.

F. Evidence of Effectiveness

1. **Original controlled trial**: delirium occurred in **9.9% on the intervention unit versus 15.0% on usual-care units (matched OR 0.60, 95% CI 0.39–0.92)**.
2. **Systematic review and meta-analysis**: **14 studies showed a significant reduction in delirium incidence (OR 0.47, 95% CI 0.37–0.59)**; three studies showed a **42% reduction in falls**; nine studies on cost showed savings of **US$1,600–3,800 per patient in hospital costs** and **US$16,000 per person-year in long-term care costs** in the year following an episode of delirium. **mHELP cluster RCT**: relative risk of delirium **0.44 (95% CI 0.23–0.83)** with **length of stay two days shorter**.
3. HELP has been implemented in **over 200 hospitals worldwide** and is regarded as the **reference standard model** for delirium and fall prevention in hospitalised older adults. Where full implementation is not possible, clinicians should **implement individual components with their interprofessional team and advocate for system-level adoption**.

Latest Updates (2023–Present)

Contemporary meta-analytic evidence

- A **systematic review and meta-analysis restricted to randomised controlled trials (Geriatric Nursing, 2024)** searched four databases to January 2024 and included **9 RCTs with 2,583 patients**. HELP **significantly reduced the incidence of delirium and shortened length of hospital stay**, but showed **no statistically significant difference in delirium severity scores or fall rates** — a more conservative estimate than the earlier mixed-design meta-analysis.
- The larger earlier synthesis remains the basis for HELP's reputation, concluding it reduces delirium incidence and fall rate with a trend toward shorter stay and less institutionalisation, and that HELP has **"emerged as a reference standard model"** for improving hospital care of older people worldwide.

HELP-ME — the remote adaptation

- The **Modified and Extended Hospital Elder Life Program (HELP-ME)** was developed because HELP depends on **face-to-face protocols and volunteers**, which became impossible during the COVID-19 pandemic. HELP-ME adapts the established protocols for **remote and/or physically distanced delivery**, and was implemented at **four expert HELP sites**, with sites modifying one or two protocols per action cycle and refining them using patient data and feedback. It was developed at Beth Israel Deaconess/Hebrew SeniorLife by Fong and Inouye, with feasibility targets of enrolling 100 hospitalised patients and achieving at least **75% protocol adherence** — relevant wherever volunteer access is limited, including resource-constrained wards.
- Current descriptions of the programme list the targeted risk factors as **cognitive impairment, vision and hearing impairment, malnutrition and dehydration, immobility, sleep deprivation and medications** — with **medications now explicitly named** as a target.

Integration with Age-Friendly Health Systems (the 4Ms)

- A **JAGS 2023 special article** shows how traditional delirium prevention maps onto the **Age-Friendly Health Systems 4Ms — What Matters, Medication, Mentation, Mobility** — presenting an explicit **crosswalk between HELP core interventions and the 4Ms**. The **IHI guides for using the 4Ms in hospitals and ambulatory practice were updated in 2024**, adding tools for integrating **equity** into every step and clarifying the meaning of **mobility**.
- The 4Ms operational guidance echoes HELP directly: establish a **delirium prevention and management protocol including orientation and mobility**, ensure **personal adaptive equipment (glasses, hearing aids, dentures, walkers)** is available and noted on the whiteboard, make a **sleep kit orderable in the EHR**, offer **non-pharmacological options for sleep and pain**, and **ambulate three times a day**.
- **Delirium prevention is becoming a reportable quality metric**: the **CMS Age Friendly Hospital Measure**, based on the 4Ms together with Geriatric Surgery Verification and Geriatric Emergency Department Accreditation, gives hospitals a structured route to implement practices designed to reduce iatrogenic harm in older adults, including **delirium, falls and adverse drug events**.

Guideline position and newer delivery adaptations

- **NICE CG103** (delirium: prevention, diagnosis and management in hospital and long-term care) had its evidence reviewed in **January 2023**, with **new recommendations on assessing and diagnosing delirium**, while multicomponent non-pharmacological prevention remains the recommended approach.
- An **adapted HELP delivered by certified nursing assistants in long-term care** has been piloted, and trials are testing **family caregivers applying HELP principles after training by a geriatric nurse specialist** in older patients with fractured femur — a model directly transferable to Indian wards, where **family attendants substitute naturally for volunteers**.

Current Practice Update

Aspect	Textbook Position	Current Practice (2023–2026)
Target risk factors	Six: cognition, sleep, immobility, vision, hearing, dehydration	**Medications explicitly added** as a seventh target; malnutrition grouped with dehydration
Delivery	Volunteer-based, face-to-face, in-hospital	**HELP-ME** enables **remote/physically distanced delivery**; adaptations by **nursing assistants in long-term care** and by **trained family caregivers**
Effect size	OR 0.47 for delirium; 42% fewer falls (mixed designs)	RCT-only meta-analysis (**9 RCTs, 2,583 patients**) confirms **reduced delirium and shorter stay**, but **no significant effect on fall rate or delirium severity**
Framework	Stand-alone delirium prevention programme	Embedded within the **Age-Friendly Health Systems 4Ms**, with a published **HELP–4Ms crosswalk**
Accountability	Local quality improvement	**CMS Age Friendly Hospital Measure** makes age-friendly, delirium-preventive practice a **hospital-level reportable attestation**
Guideline	Multicomponent non-pharmacological prevention	**NICE CG103 evidence reviewed January 2023** with new assessment and diagnosis recommendations; **antipsychotics still not recommended for prevention**

Exam One-Liners

- **HELP = Inouye's multicomponent, protocol-driven, volunteer-delivered delirium prevention programme (NEJM 1999).**
- **Six target risk factors**: cognitive impairment, sleep deprivation, immobility, visual impairment, hearing impairment, dehydration.
- **Six protocols**: orientation/therapeutic activities, sleep, early mobilisation, vision, hearing, hydration-feeding.
- **Mobilise three times a day and minimise "tethers"** (catheters, lines, restraints).
- **Original trial: delirium 9.9% vs 15.0%; matched OR 0.60.**
- **Meta-analysis: OR 0.47 for delirium; 42% fewer falls; savings US$1,600–3,800 per patient.**
- **RCT-only meta-analysis (2024): delirium ↓ and length of stay ↓, but falls and severity scores unchanged.**
- **mHELP = three nurse-delivered protocols** — orienting communication, oral hydration/ nutrition, early mobilisation; **RR 0.44, LOS 2 days shorter**.
- **ABCDEF bundle** = the ICU equivalent of HELP.
- **HELP-ME** = remote/physically distanced adaptation developed after COVID.
- **4Ms = What Matters, Medication, Mentation, Mobility.**
- **Antipsychotics do not prevent delirium**; benzodiazepines should be avoided.
- **Family at the bedside** — reassure, reorient, feed, mobilise: the low-cost Indian equivalent of the HELP volunteer.
- **Implemented in >200 hospitals worldwide** — the reference standard for delirium prevention.

Q7. Role of the Geriatric Social Worker

Position in the team, assessment, discharge planning, case management, settings and challenges — 15 marks

A. Definition and Position in the Team

1. The **geriatric (gerontological) social worker** is the professionally trained member of the interdisciplinary team responsible for the **social, psychological, economic, environmental and legal dimensions** of an older person's care.
2. Social work is one of the **minimum core disciplines** of every geriatric assessment programme, along with **physicians, nurses and physical and occupational therapists**; **special geriatric expertise among team members is crucial**.
3. The justification is structural: comprehensive geriatric assessment is **multidimensional**, and the **interdisciplinary team conference — the critical step in the process of care** — cannot set priorities without the social perspective.
4. In the **ACE unit model**, care management is deliberately shifted from a **biomedical to a biopsychosocial model**, focusing on the **relationship between the patient and the social structures needed for effective treatment**.

B. Assessment Functions

1. **Social assessment**: past social history, **family relationships and social resources**, living arrangements, personal preferences, finances, housing, and **availability and capability of the caregiver**.
2. **Psychosocial assessment in long-term care**: performed **within 1–2 weeks of admission** and as needed thereafter — identify serious psychosocial symptoms and **refer to a mental health professional**, **assess discharge potential**, and communicate findings to the team. **Knowing the family and their preferences and concerns is critical to good nursing home care.**
3. **Evidence of independent effect**: randomised trials of **social worker assessment and referral in community and outpatient settings** showed **reduced mortality, reduced hospital use, reduced permanent nursing home use and delayed development of disability**.
4. **Safety and protection**: identification of older people at **high safety risk — unsafe driving, elder abuse, neglect, self-neglect, unsafe living conditions** — and development of a plan for assessment or referral.
5. **Caregiver assessment**: burden, **financial stressors, social isolation and loneliness, and spiritual well-being**. Caregivers reporting **emotional stress have significantly increased mortality**, so **identification of the vulnerable caregiver and referral to social work and community resources can be lifesaving**.

C. Intervention and Linkage Functions

1. **Discharge planning — the classic core role.** Good discharge decisions separate two questions: **(i) what type of care will best achieve this patient's goals?** and **(ii) which provider can best deliver that care?** These require entirely different sets of information.
2. **Planning must begin early.** When started late, **availability of placements trumps careful consideration of options**, nursing home discharge is chosen because it is easiest to arrange, and the patient may be committed to an unnecessary lifetime of institutional care.
3. **Matching services to eligibility**: recommending **home health care, day care, assisted living, rehabilitation, nursing home or hospice** based on **clinical needs, personal values, home circumstances, financial resources and programme eligibility**.
4. **Transitional care and information transfer**: ensuring a **written summary of hospital course, discharge medications, cognitive and functional status, advance directives, plan of care**

and follow-up reaches the patient, family and receiving providers — **poor transmission of information drives readmission**.

5. **Counselling**: individual and family counselling, support around placement decisions, mediation of family conflict, **post-diagnostic counselling in memory clinics**, and **bereavement and grief support**.
6. **Resource mobilisation and advocacy**: entitlements, pensions, disability certification, insurance, **legal aid, guardianship, advance directives**, and access to welfare schemes.
7. **Case management**: assessment → care plan → authorisation → coordination → monitoring. This matters because **assessment without follow-up is unlikely to make any difference**; hence "geriatric assessment" has given way to **geriatric evaluation and management**.
8. **Palliative and end-of-life care**: referral to a **palliative care social worker promotes access to services, community resources, volunteers and transport**, and supports families where the support system is already at its limit; hospice programmes provide **bereavement follow-up for about a year after death**.
9. **Team, teaching and service development roles**: participation in team conferences and rounds, **training family caregivers**, running **caregiver support groups**, teaching, research, and advocating for locally missing services. Social workers can obtain **additional certification in gerontology**.

D. Settings and Challenges

1. **Settings**: acute wards and **ACE units, geriatric assessment clinics, geriatric emergency departments** (case management is a listed component), **day hospitals, nursing homes, community mental health teams for older people, home visits and hospices**.
2. **Challenges**: **great time pressure on discharge planners**, shortage of trained gerontological social workers, fragmented services, meagre resources, role overlap with case managers, and the inherent conflict between being both **patient advocate and gatekeeper**.

Latest Updates (2023–Present)

Social connection recognised as a determinant of health

- The **WHO Commission on Social Connection** released *From Loneliness to Social Connection – Charting a Path to Healthier Societies* on **30 June 2025**, finding that **1 in 6 people worldwide is affected by loneliness**, linked to an estimated **100 deaths every hour — more than 871,000 deaths annually**.
- Rates are **highest in low-income countries (about 24%)**. The Commission determined that social connection should be treated as a **"third pillar of health"** alongside physical and mental health, and the **World Health Assembly in May 2025 adopted its first-ever resolution on social connection** as a public health priority.
- In older populations, **social disconnection is associated with cognitive decline and dementia**, and lonely individuals are **twice as likely to experience depression**. Five strategic areas for action are proposed — **policy, research, interventions, measurement and public engagement**. This gives the geriatric social worker's core work — **combating isolation and rebuilding social networks** — a formal public health mandate.

Structured social assessment embedded in clinical pathways

- The **WHO ICOPE approach** is completed by the **assessment and management of social care and support**, covering **daily activities, living conditions, finances, loneliness, elder abuse, social engagement and participation**, together with a **dedicated care pathway to support the caregiver** — the social work domain written into a clinical algorithm.

- The **Age-Friendly Health Systems 4Ms** framework places **"What Matters" — goals-of-care conversations — at its centre**, and the **CMS Age Friendly Hospital Measure** now makes attestation to these practices, including **family caregiver involvement**, a hospital-level quality expectation.
- The **ADS Plus multisite cluster-randomised trial (2024)** across **34 adult day service sites** tested a staff-delivered caregiver support programme — **dementia education, support and validation, referrals and linkages, and strategies for care challenges and self-care** — essentially a structured social-work intervention delivered at scale.

India — the statutory and programmatic basis

- **NPHCE** derives from the **UN Convention on the Rights of Persons with Disabilities, the National Policy on Older Persons (1999) and Section 20 of the Maintenance and Welfare of Parents and Senior Citizens Act, 2007**, which deals with medical care of senior citizens. Its tertiary component, renamed **Rashtriya Varishtha Jan Swasthya Yojana**, runs through **19 Regional Geriatric Centres in 18 states and 2 National Centres for Ageing (AIIMS New Delhi and Madras Medical College, Chennai)**.
- Under the **MWPSC Act, 2007**, a **Tribunal presided over by the Revenue Divisional Officer** has been constituted in each sub-division for speedy disposal of maintenance petitions — maintenance can be claimed through these tribunals or under Section 125 CrPC, with tribunals empowered to order up to **₹10,000 per month**, and older people can seek eviction of abusive or neglectful children from their own property. Guiding families through these remedies is now core geriatric social work in India.
- The **Elder Line (national helpline 14567)** provides **free information, counselling, emotional support and field services** for issues of **care, abandonment, abuse and legal advice**, while the **Integrated Programme for Older Persons** funds NGOs to run **old age homes, day-care centres and mobile medicare units**.
- **Economic vulnerability defines the workload**: the **India Ageing Report 2023 (UNFPA–IIPS)** found **more than 40% of elderly Indians in the poorest wealth quintile and about 18.7% living with no income**, and recommended bringing **all old age homes under regulatory purview** and promoting **in-situ ageing with day-care and elder-creche facilities**.
- The **PM-SPECIAL scheme** of the Ministry of Social Justice and Empowerment is creating a trained cadre through courses such as **Geriatric Caregiver (Institutional and Home Care) and Elderly Care Companion**, though an **EAC-PM working paper** notes that **workforce development remains fragmented without a standardised certification system**, and that models such as **Kerala's Kudumbashree caregiver cooperatives** successfully link training to employment.

Current Practice Update

Aspect	Textbook Position	Current Practice (2023–2026)
Social isolation	Recognised as a caregiver and patient problem	Formal public health priority — **1 in 6 people lonely, 871,000 deaths/year**; **first WHA resolution on social connection (May 2025)**; social connection as the **"third pillar of health"**
Social assessment	Part of CGA, discipline-dependent	**Structured and protocolised** — ICOPE social care and support domain plus a **caregiver pathway**; 4Ms "What Matters"
Caregiver support	Referral to support groups and community resources	

		Manualised, trial-tested programmes delivered by trained staff within day services (ADS Plus)
Accountability	Local practice	**CMS Age Friendly Hospital Measure** makes goals-of-care and caregiver engagement reportable
India	Not addressed	**NPHCE (Section 20, MWPSC Act) with 19 RGCs and 2 NCAs; maintenance tribunals in every sub-division; Elder Line 14567; IPSrC/AVYAY-funded day care and old age homes**
Workforce	Optional certification in gerontology	India lacks **standardised certification**; PM-SPECIAL and community caregiver cooperatives are the emerging response

Exam One-Liners

- **Social work is a minimum core discipline of every geriatric assessment programme.**
- **The interdisciplinary team conference is the critical step** — social work supplies the social perspective.
- **ACE units shift care from a biomedical to a biopsychosocial model.**
- **RCTs of social worker assessment and referral** reduced mortality, hospital use and permanent nursing home use.
- **Psychosocial assessment in long-term care: within 1–2 weeks of admission**, then as needed.
- **Two discharge decisions**: what *type* of care, then which *provider* — different information for each.
- **Late discharge planning means placement by availability, not by need.**
- **Poor information transfer at discharge drives readmission.**
- **Assessment without follow-up makes no difference** — hence geriatric *evaluation and management*.
- **Caregivers with emotional stress have increased mortality** — identifying them can be lifesaving.
- **Advocate versus gatekeeper** — the central role conflict of the social worker/case manager.
- **WHO 2025: 1 in 6 people lonely; ~871,000 deaths a year; ~100 deaths per hour.**
- **ICOPE social domain**: daily activities, living conditions, finances, loneliness, elder abuse, social participation — plus a **caregiver pathway**.
- **India: Elder Line 14567** for information, counselling, abuse, abandonment and legal advice.
- **MWPSC Act 2007** — maintenance tribunals under the RDO in each sub-division; up to **₹10,000 per month**.
- **>40% of Indian elderly are in the poorest wealth quintile; 18.7% have no income.**

Q8. Caregiver Burden, Stress and Burnout — Assessment and Management

Definitions, domains of impact, screening instruments and evidence-based interventions — 15 marks

A. Definitions and Concepts

1. A **caregiver** is a person who provides assistance to a dependent person; the prefix **"family caregiver"** distinguishes the **unpaid** from the paid role. Even unrelated caregivers often regard themselves as **"fictive kin"**.
2. **Caregiver burden** is the **multidimensional strain — physical, psychological, social and financial — arising from the caregiving role**. It must be distinguished from generalised **psychological distress**: strain is role-specific.
3. **Caregiver burnout** = **emotional exhaustion, detachment or depersonalisation from the care recipient, and a reduced sense of accomplishment**. It occurs typically when caregivers **lack the social support systems needed for caregiving tasks and shared decision-making**. **Emotional withdrawal and apathy toward the care recipient are late features and a warning sign of impending neglect or abuse.**
4. Caregiving is **not uniformly negative**. When the caregiver has an **emotionally close relationship** with the recipient, burden is lower; **satisfaction and a sense of personal meaning are protective**. Assessment should therefore also be **strengths-based**.

B. Magnitude of the Problem

1. **Informal care is the backbone of long-term care** — **over 80% of community care is informal**, predominantly by women. Weekly hours range from about **7 hours (Canada) to 24 hours (USA)**, with roughly **25% of caregivers giving over 40 hours per week**.
2. Compared with non-caregivers, caregivers have **higher stress, more depression and lower overall well-being**. **Women report higher stress**, and **spousal caregivers fare worse than those caring for a parent or child**.
3. In India the family is the **default and only provider**; wives and daughters-in-law bear most of the load, often sacrificing employment.

C. Domains of Impact

1. **Physical health**: over **15% of caregivers rate their health as fair or poor** versus 10% of the general population; caregivers **aged ≥50 have increased risk of cardiovascular disease and hypertension**; sleep disturbance, fatigue, pain, appetite change and weight loss are common. **Bathing, transfers and toileting** are the most physically demanding tasks and cause **musculoskeletal pain and injury**.
2. **Mortality**: caregivers reporting **high strain have increased mortality** compared with those reporting low strain — though population-based propensity-matched analyses show caregiving *per se* may be associated with **lower** mortality. **It is the strain, not the role, that kills.**
3. **Mental health**: depression, anxiety and anticipatory grief. Critically, **symptoms do not diminish after institutional placement** — **anxiolytic use rises and nearly half of caregivers are at risk of disabling depression after placing their relative in long-term care**.
4. **Financial**: about **35% report moderate to high financial strain**; out-of-pocket costs for **medicines, equipment and transport**; erosion of pension and retirement funds; **reduced work productivity, early exit from the workforce and difficulty re-entering employment**.
5. **Social and spiritual**: shrinking social networks, isolation and loneliness; the caregiver **gives more support to the recipient while receiving less in return**. **Spiritual and religious coping reduces both burden and depressive symptoms** and is an overlooked resource.

6. **Determinants of high burden**: **behavioural and psychological symptoms of dementia, apathy, incontinence, immobility, night-time disturbance**, high dependency, co-residence, spousal relationship, female sex, poverty and absence of support. **Long-distance caregiving** carries its own emotional and financial strain.

D. Assessment

1. **Screen routinely — early identification of the at-risk caregiver is the single most important preventive step. Identification of the vulnerable caregiver and referral to social work and community resources can be lifesaving**, since emotionally stressed caregivers have significantly increased mortality.
2. **Instruments**:
   - **Zarit Burden Interview (ZBI)** — original 29 items, commonly used **22-item** form, with **12-, 4- and 3-item** versions; developed for dementia but now used across illnesses, and **validated in Brazil, sub-Saharan Africa, Italy and China**.
   - **Caregiver Self-Assessment Questionnaire (CSAQ)** — 18 items, developed by the American Medical Association; also **valid as a screen for depression**.
   - **Rapid Screen for Caregiver Burden** — 7 items (shortened from 25).
   - **Caregiver Well-Being Scale** — 43 items, with 16-item and **6-item Rapid** versions; **strengths-based**, assessing basic needs and activities.
3. Assess **all biopsychosocial-spiritual domains**: physical health, mood, sleep, finances, employment, social support, spirituality, quality of the caregiver–recipient relationship, and **risk of elder abuse or neglect**.

E. Management

1. **General principle** — effective interventions are **multidimensional (psychological + social/emotional + educational)** and target the **dyad**, not the patient alone. A **high-risk caregiver–patient dyad needs higher-intensity care**.
2. **Psychoeducation and skills training**: **cognitive behavioural family intervention**, **behaviour-management training for BPSD**, problem-solving training, contingency planning, and practical skills (**assisting with ADLs, medication administration, symptom management**). **Counselling with ad hoc telephone support, combined with cholinesterase inhibitor therapy in the patient, reduced caregiver depression from moderate to lower levels** — showing that remote formats can enhance or replace face-to-face intervention.
3. **Respite** — **adult day care and day services** are the mainstay; short-term institutional respite where needed. **When carer strain is high, respite is preferable to admission.**
4. **Mind–body interventions**: **Mindfulness-Based Stress Reduction** (formal practice — mindful movement, body scan, sitting meditation; plus informal everyday practice) reduces stress; an **online twice-weekly mindfulness intervention reduced caregiver burden and improved quality of life**, and **yoga with compassion meditation improved quality of life and self-compassion** in caregivers of people with Alzheimer's disease.
5. **Support groups** — two types: **(i) time-limited training groups** and **(ii) ongoing mutual-support forums**. Disease-specific or identity-specific groups improve attendance; **substitute care must be arranged** so the caregiver can actually attend.
6. **Team-based support and cultural sensitivity**: the **occupational therapist** teaches home skills and safety, the **physiotherapist** safe transfers and exercise, the **dietitian** nutrition, the **speech therapist** communication and swallowing, the **social worker** resources, finances and legal help. In **traditional Asian and Latinx cultures, institutional placement is perceived as abandonment**, so suggesting a facility may be experienced as culturally insensitive. **Prepare the caregiver for placement transitions and treat depression and anxiety afterwards.**

Latest Updates (2023–Present)

Global policy recognition

- WHO estimates about **142 million older people worldwide cannot meet basic needs independently**; **unpaid informal care accounts for roughly 80% of all long-term care**; and **support to unpaid caregivers** is now an explicit pillar of the WHO framework for an integrated continuum of long-term care.
- The **WHO Commission on Social Connection report (30 June 2025)** found **1 in 6 people worldwide affected by loneliness**, linked to **871,000 deaths a year (~100 per hour)**, with the **World Health Assembly adopting its first-ever resolution on social connection in May 2025** — directly relevant, since **social isolation is a core component of caregiver burden**. **WHA78 (2025) also extended the Global Dementia Action Plan to 2031**, retaining carer support as a pillar.

Landmark trial evidence — D-CARE

- The **Dementia Care Study (D-CARE)** was a **pragmatic, 18-month, three-armed, assessor-blinded randomised clinical trial** conducted from **June 2019 to August 2023**, comparing **health-system dementia care** (co-management by nurse practitioners or physician assistants), **community-based dementia care** (delivered telephonically by a social worker, nurse or licensed therapist) and **usual care**.
- Across **2,176 patient–caregiver dyads**, there were **no significant differences in patient behavioural symptoms or caregiver strain** between the two active models, nor between either active model and usual care; a prespecified analysis found **no differences in functional status, cognition or quality of life, nor in overall caregiver burden or positive aspects of caregiving**.
- However, **caregiver self-efficacy — confidence in the ability to care — improved**, and **caregiver satisfaction with care was significantly greater in both active arms than usual care from 3 months onward and sustained to 18 months**, with community-based care marginally highest. Higher self-efficacy is thought to help caregivers **obtain more resources and keep the person with dementia at home**.
- **Examination message**: comprehensive dementia care programmes reliably improve **confidence and satisfaction**, but **measurably reducing strain remains difficult** — supporting the emphasis on **respite and multicomponent support** rather than care-coordination alone.

Caregiver support becomes a reimbursed service

- The **CMS GUIDE Model (Guiding an Improved Dementia Experience)**, launched **January 2024**, is an **alternative payment model** funding interprofessional teams to deliver comprehensive dementia care. Required components include **screening and recruitment, comprehensive and home safety assessment, ongoing monitoring, caregiver supports and respite**, with dyads tiered by complexity. **Low-tier dyads receive less frequent outreach, are not eligible for respite funds and attract lower payment**, although early data show their caregivers report comparable burden (Zarit Index) and unmet needs. In one first-year site, **over 35% of eligible beneficiaries used respite services**.

Respite, day services and digital support

- An **updated systematic review (2015–2025)** found regular adult day service use associated with **reduced caregiver stress, improved mood, physiological stress reduction, delayed nursing home placement and cost savings**, with barriers of **cost, awareness, caregiver reluctance and availability**. The **ADS Plus cluster-randomised trial across 34 sites** embedded a staff-delivered caregiver programme — **dementia education, support and validation, referrals and linkage, and strategies for care challenges and self-care** — within day services.

- **WHO iSupport**, an online self-training manual emphasising **education, skill development and caregiver support**, is being adapted and tested across low- and middle-income countries including **Bangladesh**. Meta-analyses of internet-based interventions for dementia caregivers show consistent improvement in **depressive symptoms, perceived stress, anxiety and self-efficacy**, but **no significant improvement in caregiver burden, coping ability or quality of life**.

India

- The **LASI-DAD** (Longitudinal Aging Study in India – Diagnostic Assessment of Dementia) added a **caregiver module**, giving nationally representative data from **4,196 informants** linking caregiver **stress, mental health, positive affect and spirituality** to the care recipient's cognitive function and dementia status. WHO data suggest **India has a higher reported prevalence of informal caregivers than other LMICs**; Indian dementia caregiving is associated with **reduced work hours and poorer health**, and **more caregiving hours predict higher risk of depressive symptoms**.
- A **2024 narrative review of community-based dementia care in India** confirms care is traditionally provided by family members, with studies showing **high caregiving burden and consequent mental health impact**, and identifies **telehealth and online caregiver training as viable alternatives**. It cites the **NITI Aayog report "Senior Care Reforms in India: Reimagining the Senior Care Paradigm" (February 2024)** and the **India Ageing Report 2023**.
- A **Lancet Psychiatry** call for a **national dementia policy** notes India will have **340 million older adults by 2050**, of whom **5.3 million have been living with dementia since 2020**, with persistent shortages of services and trained staff. A systematic review of caregiver burden in Asian LMICs (2025) notes that **about 94% of people with dementia in LMICs are cared for at home**, mostly by family.
- India's structural response is the **PM-SPECIAL scheme** creating a trained cadre of **geriatric caregivers for institutional and home care**, alongside **Elder Line 14567** for counselling and the day-care and respite components of **AVYAY**.

Current Practice Update

Aspect	Textbook Position	Current Practice (2023–2026)
Status of the caregiver	A resource to be supported, assessed opportunistically	**A formal target of health policy** — explicit WHO pillar; caregiver support and respite are **reimbursed services** under the CMS GUIDE Model
Effect of care-coordination programmes	Multicomponent interventions reduce burden and depression	**D-CARE (2,176 dyads)**: no reduction in **caregiver strain or burden**, but improved **self-efficacy and satisfaction with care** — expectations must be recalibrated
Respite	Day care as respite	Strengthened evidence for **reduced caregiver stress, delayed nursing home placement and cost savings**; respite now a funded benefit tied to complexity tier
Digital interventions	Telephone support as an alternative delivery mode	**WHO iSupport** and internet programmes improve **depression, stress, anxiety, self-efficacy**, but **not burden or quality of life**
Screening instruments	ZBI, CSAQ, RSCB, Caregiver Well-Being Scale	Same instruments, now embedded in **programme eligibility and tiering** (Zarit used for GUIDE complexity assessment)

India	Not addressed	**LASI-DAD caregiver module** gives national data; **NITI Aayog senior care reforms (2024)**; **telehealth and online caregiver training** as the scalable model; **PM-SPECIAL** caregiver cadre

Exam One-Liners

- **Caregiver burden = multidimensional strain; burnout = emotional exhaustion, detachment and reduced accomplishment.**
- **Apathy and emotional withdrawal from the care recipient are red flags for neglect and abuse.**
- **>80% of community long-term care is informal**; ~25% of caregivers give **>40 hours a week**.
- **Women and spousal caregivers report the highest stress.**
- **15% of caregivers rate their health fair/poor vs 10% of the general population.**
- **Caregivers ≥50 have increased cardiovascular disease and hypertension.**
- **It is strain, not caregiving itself, that increases mortality.**
- **Distress does not resolve after institutional placement** — anxiolytic use rises; nearly half are at risk of disabling depression.
- **~35% report moderate to high financial strain.**
- **Zarit Burden Interview**: 29 → 22 → 12 → 4 → **3-item** versions; **CSAQ** 18 items (AMA, also screens depression); **Rapid Screen for Caregiver Burden** 7 items; **Caregiver Well-Being Scale** is the strengths-based tool.
- **Emotional closeness reduces burden** — assess positives, not only pathology.
- **Respite is preferable to admission when carer strain is high.**
- **MBSR, yoga and compassion meditation reduce burden and improve quality of life.**
- **Support groups**: time-limited training vs ongoing mutual support; **arrange substitute care so the carer can attend**.
- **Suggesting institutional placement can be culturally insensitive** in Asian and Latinx families.
- **D-CARE (2025)**: comprehensive dementia care improved **self-efficacy and satisfaction** but **not strain or burden**.
- **CMS GUIDE Model (2024)** funds **caregiver education, 24/7 support and respite** as a covered service.
- **94% of people with dementia in LMICs are cared for at home** by family.

MD Geriatric Medicine

High-Yield Examination Answers

Module 1 — Geriatric Assessment, Function, Frailty, Disability and Cognition

Fifteen structured answers with Latest Updates (2023–present), Current Practice Update tables and rapid-recall one-liners

Primary source: Pathy's Principles and Practice of Geriatric Medicine, 6th edition
Supported by Kane's Essentials of Clinical Geriatrics and current international and Indian guidelines

Contents

Part I — Comprehensive Geriatric Assessment and Functional Evaluation

1. Comprehensive Geriatric Assessment — What It Is and Its Usefulness in Clinical Practice
2. Tools of Functional Assessment
3. Functional Assessment in the Elderly
4. Functional Assessment in an 85-Year-Old Lady
5. Physical Self-Maintenance Scale (PSMS)

Part II — Frailty and Sarcopenia

6. Frailty Syndrome — Etiopathogenesis, Assessment, Management and Prevention
7. Comparison of the Tests Used to Identify or Screen for Frailty
8. Sarcopenia in Ageing — Diagnostic Criteria, Investigation and Management

Part III — Disability, Immobility and Rehabilitation

9. Causes of Chronic Disability in the Elderly — Prevention and Management
10. Assessment of Disability in Older People and Rehabilitation
11. Immobility in the Elderly — Causes, Complications and Management

Part IV — Cognition, Mood and Decisional Capacity

12. Tests of Cognition and Rating Scales
13. Cognitive Assessment Tools
14. Depression Rating Scales in the Elderly
15. Assessment of Decisional Capacity in an Older Person

Appendices

Appendix A — Four Frequently Confused Terms

Appendix B — 3D-CAM and CAM-ICU

Part I — Comprehensive Geriatric Assessment and Functional Evaluation

1. Comprehensive Geriatric Assessment — What It Is and Its Usefulness in Clinical Practice

A. 20 High-Yield Points

Definition and Concept

1. **Comprehensive geriatric assessment (CGA)**, also called **multidimensional geriatric assessment**, is a **diagnostic process, usually interdisciplinary**, that determines an older person's **medical, psychosocial, functional and environmental** resources and problems.
2. Its stated objective is to **develop an overall plan for treatment and long-term follow-up**. An assessment **without a linked care plan is NOT a CGA** — a favourite examination point.
3. It **differs from a standard medical evaluation** in three ways: it targets older people with complex problems, it **emphasises functional status and quality of life**, and it uses **interdisciplinary teams with quantitative scales**.
4. A purely psychosocial or environmental home visit **without a medical evaluation** also does not qualify as CGA, because multidimensionality is incomplete.
5. **Historical origin: Dr Marjory Warren**, London infirmary, **late 1930s**. Through evaluation, mobilisation and rehabilitation she got long-bedfast patients out of bed and home, and advocated **assessment and rehabilitation before any long-term institutionalisation**.

Rationale — Why in the Elderly? (five textbook reasons)

6. **Multi-morbidity and complexity** — systematic assessment helps set priorities among multiple conditions.
7. **Unrecognised problems** — many problems are never reported, often because they are **falsely thought to be non-modifiable effects of ageing**.
8. **Chronic conditions** — a diagnosis without information on **functional relevance** is of limited therapeutic value.
9. **Social and environmental interaction** — home layout and social network determine whether a person can stay at home.
10. **Functional status** — the main goal of geriatric medicine is to **prevent or delay functional decline**; benefit is greatest when intervention occurs **early** in the path from robustness to frailty to disability.

Components — What Is Assessed

11. CGA begins with a **functional status "review of systems"** built on three measures: **frailty, basic ADLs, and IADLs**.
12. **Principle of hierarchy / parsimony:** if a patient is **severely ADL-dependent, do not assess frailty**; if the patient is **robust or pre-frail, do not assess basic ADLs**.
13. Frailty is measured by two models — the **phenotype model** (Fried phenotype, Frailty Trait Scale, FRAIL index) and the **cumulative deficit model** (Frailty Index, Clinical Frailty Scale).
14. **Dimensions and tools (Table 96.1):** BADL — **Katz, Barthel, Lawton PSMS**; IADL — **Lawton, OARS**; Social — **Lubben Social Network Scale**; Affective — **Geriatric Depression Scale, Zung**; Cognitive — **MMSE (Folstein), Kahn MSQ**; Gait and balance — **Tinetti, Get-Up-and-Go**; Nutrition — **MNA**; Senses — **whispered voice test, Snellen**; Oral — **GOHAI**.
15. Two screens are **mandatory** in any older person with functional disability: **cognitive impairment and depression**.

Process in Office and Clinic Practice

16. **Step 1 — set priorities:** work first on the problem that **most bothers the patient**, or the problem on which resolution of others depends (**alcoholism, depression**).
17. **Step 2 — task and symptom analysis:** define the exact nature of the impairment and correlate it with **new drugs or dose changes (including over-the-counter), change in living conditions, and intercurrent acute illness**. Then examination, laboratory tests, therapist and social-work input, and a **long-term follow-up / case-management plan**.

Settings and Team

18. **Settings:** home (preventive visits), office and outpatient clinic, **day hospital**, **acute care for the elderly (ACE) unit**, post-acute rehabilitation unit, nursing home (**Resident Assessment Instrument**), and hospital-at-home programmes. Inpatient and day-hospital settings give intensity and speed for the frail; outpatient settings are cheaper.
19. **Team (minimum):** physician, nurse, social worker, physiotherapist, occupational therapist; **optimally** dietician, pharmacist, ethicist, home-care specialist. The **interdisciplinary team conference is critical** — present in most successful trials; teams mature through **forming, storming, norming, performing**.

Evidence of Usefulness — The Exam Core

20. **Proven benefits:** CGA **improves function and survival while reducing healthcare utilisation and costs**.
    Supporting data:

- **Improved diagnostic accuracy — 1 to more than 4 new diagnoses per patient**.

- **1993 Stuck/Rubenstein meta-analysis (28 trials, ~10,000 patients):** mortality down **22%** (hospital-based, 12 months) and **14%** (all programmes); living at home up **47%** (hospital-based) and **26%** (all); readmissions down **12%**; cognitive improvement up **47%**; physical function up **72%** on hospital units.
- **2011 Cochrane meta-analysis:** inpatient CGA increases the likelihood of being **alive and in one's own home**, with potential cost reduction.
- **Better prescribing** — CGA reduces unnecessary and inappropriate medication use.
- **Targeting matters:** best results when patients are at **high risk of deterioration yet retain rehabilitation potential** — exclude those **"too well or too ill."**
- **Programmes with hands-on care and/or long-term follow-up** outperform purely consultative programmes.
- **Weak areas:** outpatient CGA showed **no mortality benefit** in one meta-analysis; **preventive home visits are not recommended routinely** (benefit only with CGA-based extended follow-up in relatively well elders; **number needed to visit about 40** to prevent one nursing-home admission).
- **Oncology (SIOG task force):** CGA detects many problems missed by regular assessment and is associated with **fewer treatment side effects**.

B. Latest Updates (2023–Present)

1. German DGG S3 Guideline on CGA in Hospital (2024) — the first S3-level CGA guideline. Twelve evidence-based and eight consensus-based recommendations. Strongest evidence in **acute geriatric, oncology and orthopaedic/trauma wards**; weaker in emergency departments and general surgery. Core elements: **minimum CGA duration of 15 minutes** and **six core dimensions — self-help ability, mobility, cognitive function/delirium, affect, nutrition and social situation**; the **G8 questionnaire** is recommended for screening mainly in oncology.

2. Italian National CGA Guideline (2024) — 25 scientific societies plus the National Institute of Health, GRADE methodology. Sixteen recommendations from **117 studies** across primary care, clinics, emergency departments, hospital wards and nursing homes. CGA **reduces hospitalisation, mortality, institutionalisation and delirium risk**, improves prescribing appropriateness, and **maintains functional activities**. The **Multidimensional Prognostic Index (MPI)** is specifically recommended to predict negative outcomes.

3. Japanese CGA-Based Healthcare Guidelines 2024. The main objective of CGA is stated to be **personalised care through holistic management plans and targeted interventions — not merely status evaluation or prognostication**.

4. ASCO Geriatric Oncology Guideline Update (2023). Geriatric assessment should be used in **all patients over 65 with cancer**; all older adults with GA-identified deficits should receive **GA-guided management**, and the **Practical Geriatric Assessment (PGA)** is the recommended tool. Evidence base: **GAP70+** and **GAIN** trials.

5. ASCO Global (Resource-Stratified) Geriatric Assessment Guideline (2025) — adapts the 2023 guideline for **resource-limited settings** using ADAPTE methodology. Directly relevant to Indian practice.

6. Cochrane 2022 — Community CGA. Twenty-one studies, 7,893 participants, 10 countries, evaluating domiciliary CGA and CGA in general practice, with outcomes of death, nursing-home admission, unplanned hospital admission and emergency visits.

7. Meta-analysis of CGA in acute hospitalisation (2025) — CGA **may improve short-term survival and reduce readmission rates** after acute hospitalisation.

8. Perioperative CGA is now mainstream. The **2023 World Society of Emergency Surgery guidelines** advocate frailty assessment and multidisciplinary geriatric-surgical management in older trauma patients; the UK **NELA Best Practice Tariff (April 2023)** mandates perioperative team involvement in frailer laparotomy patients. CGA outperforms traditional preoperative risk models in predicting **postoperative delirium, infection and prolonged stay**.

9. Indian context. Evidence is predominantly from high-income countries and hospitalised patients; research on CGA in primary care and community settings in low- and middle-income countries is lacking. Under **NPHCE**, community services are delivered by **ASHA workers** with clinics at PHCs, but implementation is inconsistent. Short culturally adapted tools such as **EHSAS** are being validated for rural India because full CGA is impractical with the available workforce.

C. Current Practice Update

Aspect	Pathy's 6th ed. (2022)	Current position (2023–2026)
Standardisation	Deliberately broad; "no single optimal blueprint"	**Minimum standards defined** — German S3: at least 15 minutes, six mandatory core domains
Prognostic tool	Many scales listed; none preferred	**MPI specifically recommended** (Italian guideline)
Oncology tool	SIOG could not recommend any specific tool	**PGA specifically recommended** (ASCO 2023)
Oncology strength	CGA "possibly" improves survival	**GA mandatory at 65 and above**; GA-guided management proven to cut severe chemotoxicity
Outpatient CGA	"Less impressive"; no mortality benefit	Italian guideline **supports** primary-care CGA for reducing hospitalisation and institutionalisation
Preventive home visits	Cannot be recommended as routine practice	**Largely unchanged** — Cochrane 2022 still shows uncertain benefit
Perioperative CGA	Not covered	Now **embedded in surgical pathways** (NELA BPT, WSES 2023, POPS model)
Emergency department	Not addressed	Recognised as important but **evidence still weak** (German S3)

D. Exam One-Liners

- **CGA without a care plan and follow-up is not a CGA.**
- **Marjory Warren, 1930s London** — mother of CGA and of British geriatrics.
- **Function is the common language of geriatrics; assessment is its core method.**
- **Hierarchy rule:** ADL-dependent, skip frailty testing; robust or pre-frail, skip basic ADL testing.
- **Targeting rule:** benefit is greatest in those **"not too well, not too ill"** — high risk with rehabilitation potential.
- **Hands-on care plus long-term follow-up** separates effective from ineffective programmes.
- **1993 meta-analysis:** mortality down 22%, living at home up 47%, physical function up 72% on hospital units.
- **Two mandatory screens** in functional disability: **cognition and depression**.
- **German S3 six core domains:** self-care, mobility, cognition/delirium, affect, nutrition, social situation.
- **ASCO 2023:** every cancer patient 65 and above gets a geriatric assessment, using the **PGA**.
- **Italian 2024:** use the **MPI** for prognosis.
- **India:** CGA delivery constrained by workforce — hence short screening tools within **NPHCE**.

---

2. Tools of Functional Assessment

A. 20 High-Yield Points

Framework

1. **Functional assessment** measures **what a person actually does in daily life**, not merely what diseases they have. Tools exist to make this measurement **standardised, reproducible and comparable across settings**.
2. **Purpose of using standardised tools:** they allow **information exchange between acute, rehabilitation and community settings**, permit **measurement of effectiveness**, foster **comparison of treatments**, and **minimise repeated collection of identical information** by different team members.
3. **Three levels of measurement (ICF, 2001): impairment** (body function or structure), **activity limitation** (difficulty executing a task), and **participation restriction** (problems in involvement in life situations). Different tools address different levels.
4. **Three functional core measures** in CGA: **frailty, basic ADLs, and IADLs**. The **hierarchy rule** determines which to use — severely ADL-dependent patients are not tested for frailty; robust patients are not tested for basic ADLs.
5. **Two methods of data capture: report** (by the patient or a proxy — tells you what they do under real living conditions) and **performance testing** (tells you what they can do under standard conditions). **Discrepancies between the two are common and informative.**

Basic ADL Tools

6. **Katz Index (1963)** — six items: **bathing, dressing, toileting, transferring, continence, feeding**. Graded independent/dependent, letters A to G. The classic **hierarchy of ADL loss**.
7. **Barthel Index (1965; modified 1988)** — ten items: feeding, bathing, grooming, dressing, bowels, bladder, toilet use, transfer, mobility, stairs. Scored **0–100** (or 0–20). It is an **observation of what the patient does**, and is the **RCP/BGS-endorsed ADL scale**, but has a **ceiling effect in ambulatory patients**.
8. **Lawton Physical Self-Maintenance Scale (PSMS, 1969)** — six items: **toileting, feeding, dressing, grooming, physical ambulation, bathing**. Each graded on **five levels**, dichotomised to a total of **0–6** (6 = independent). Published in the same paper as the Lawton IADL scale.

IADL Tools

9. **Lawton IADL scale (1969)** — eight items: **telephone, shopping, food preparation, housekeeping, laundry, transport, medications, finances**. IADLs need "instruments," and are the **first functions to be lost**.
10. **OARS Multidimensional Functional Assessment Questionnaire** — assesses IADLs alongside social and economic resources; widely used in surveys.
11. **Clinical rule:** if difficulty is reported, always ask **"how does she manage?"** — is the task neglected, or is someone else doing it?

Mobility, Gait and Balance Tools (high-yield numbers)

12. **Gait speed** over a 4-metre track: **below 0.6 m/s is abnormal**; **0.6–0.8 m/s is high-risk**. Each **0.1 m/s fall in gait speed equals a 7% rise in fall risk**.
13. **Timed Get-Up-and-Go (Mathias, Nayak and Isaacs, 1986): more than 12 seconds indicates high fall risk** (sensitivity 77%, positive predictive value 93% in the community).
14. **Tinetti Performance-Oriented Mobility Assessment (1986):** 14 tasks, **score below 24 out of 28 indicates fall risk**; takes 10–15 minutes.
15. **Short Physical Performance Battery (SPPB):** combines gait speed, **30-second chair stand** and the **4-stage balance test** (each position held **10 seconds**). **Berg Balance Scale:** 14 items, 0–56 (0–20 wheelchair-bound, 21–40 walks with assistance, 41–56 independent).

Composite, Global and Domain-Specific Tools

16. **Functional Independence Measure (FIM)** — scores **18 functions** across self-care, sphincter control, mobility, locomotion, communication and social cognition; the standard rehabilitation outcome measure.
17. **RCP/BGS endorsed scales (Table 98.3):** ADL — **Barthel Index**; vision, hearing and communication — **Lambeth Disability Screening Questionnaire**; memory — **Abbreviated Mental Test (10 items)**; depression — **Geriatric Depression Scale (30-item, 15-item short form)**; morale — **Philadelphia Geriatric Center Morale Scale**.
18. **Other domain tools (Table 96.1):** nutrition — **Mini Nutritional Assessment (MNA)**, SNAQ; special senses — **whispered voice test, Hearing Handicap Inventory, Snellen chart**; oral health — **GOHAI**; social network — **Lubben Social Network Scale**; cognition — **MMSE**; mood — **GDS, Zung**.
19. **Frailty tools: Fried phenotype** (weight loss, exhaustion, weakness, slow gait, low activity); **FRAIL scale** (Fatigue, Resistance, Aerobic, Illnesses more than 5, Loss of weight 5%) — answerable in 15–30 seconds; **SOF index** (weight loss, cannot rise from chair 5 times without arms, reduced energy); **Clinical Frailty Scale** (9 points, seconds to apply); **Frailty Index** (30 or more deficits, continuous 0–1).

20. **Screening questions usable at the bedside:** "*Do you need help at home?*" (function) · "*Do you have trouble walking or lose your balance? Have you had a fall? Are you afraid of falling?*" (mobility) · "*Are you sad?*" (mood) · "*Have you lost weight?*" (nutrition) · "*Are you on seven or more medicines?*" (polypharmacy) · "*Do you wet yourself?*" (continence).

B. Master Comparison Table

Domain	Screening tool	Assessment tool	Key threshold
Frailty	FRAIL scale, Clinical Frailty Scale	Fried phenotype, Frailty Index	3 or more of 5 = frail
Basic ADL	—	**Katz, Barthel, Lawton PSMS**	Barthel below 60 = dependent
IADL	—	**Lawton IADL, OARS**	Loss precedes ADL loss
Gait / mobility	Gait speed, TUG	Tinetti POMA, SPPB, Berg	Gait below 0.8 m/s; TUG above 12 s; Tinetti below 24
Cognition	Mini-Cog, AD8	MMSE, MoCA, SLUMS	MMSE below 24
Mood	PHQ-2	GDS-15/30, PHQ-9, CSDD	GDS-15 of 5 or more
Nutrition	MNA-SF, SNAQ	MNA full, BMI, weight loss	MNA-SF 11 or less
Vision	Snellen	Formal ophthalmic review	Below 6/18
Hearing	Whispered voice test	Audiometry, HHIE	Fails at 2 feet
Social	Lubben Social Network Scale	OARS social section	LSNS-6 below 12
Rehabilitation outcome	—	**FIM**	18 items

C. Latest Updates (2023–Present)

1. WHO ICOPE — from deficit to capacity. The ICOPE 5-step pathway (screen, assess, intervene, follow up, community engagement) screens **six domains — mobility, cognition, vitality/nutrition, vision, hearing and psychological well-being** — either by a professional or by self-assessment through the **ICOPE Monitor** app. Step-2 tools are named explicitly: **SPPB for mobility; Mini-Cog, MoCA, MMSE or GPCOG for cognition; MNA, DETERMINE, MUST or SNAQ65+ for nutrition; PHQ-9 for mood; pure tone and speech audiometry for hearing**.

2. Minimum data sets are now mandated. The **German S3 guideline (2024)** requires **six core dimensions in at least 15 minutes**; the **Italian guideline (2024)** recommends the **Multidimensional Prognostic Index (MPI)** for prognosis; **ASCO 2023** mandates the **Practical Geriatric Assessment (PGA)** in oncology. The trend is **domain-mandated but tool-agnostic**.

3. Gait speed is being repositioned as the single best functional marker. A 2025 Age and Ageing study of about 3,600 older adults found that among all components of frailty and sarcopenia, **low physical performance (slow gait speed) was the best single predictor of incident basic and instrumental ADL disability**; sarcopenia as a whole construct was not associated with increased disability risk. **EWGSOP2 defines low physical performance as gait speed of 0.8 m/s or less.**

4. Automated and digital tools. The **electronic frailty index (eFI2)** is replacing the original eFI in English primary care from 2025, allowing **population-level functional risk stratification from routine records**. mHealth and wearable-based functional assessment gives **objective, quantitative data** in place of subjective observer-rated scales, but a 2024 systematic review found **high risk of bias and minimal integration into daily practice**.

5. Diagnostic accuracy data for screening instruments. In emergency-department settings, seven screens pooled across 1,663 participants gave **sensitivity 0.85 and specificity 0.77** against full CGA; **PRISMA-7 was the most sensitive (0.84–0.98)** and the **Clinical Frailty Scale the most specific**, both with AUROC around 0.90.

6. Indian use. LASI applies Katz-type ADL and Lawton-type IADL items; grip strength was the **most discriminant frailty component in Indian men (79.1%)** and physical activity in women (81.6%). **AWGS (not EWGSOP2) cut-offs** should be used for sarcopenia-related functional measures in Indians, and **BIA rather than DXA** is the practical mass-measurement modality.

D. Current Practice Update

Aspect	Pathy's 6th ed. (2022)	Current position (2023–2026)
Choice of tool	Broad menu; "no consensus on the best scale"	**Domain mandated, tool still flexible**, but specific tools named per setting
Gait speed cut-off	Below 0.6 m/s abnormal; 0.6–0.8 high-risk	**0.8 m/s or less** operative threshold (EWGSOP2/AWGS); best single predictor of disability
Who administers	Trained professional or therapist	**Self-assessment and digital screening validated**; community health workers in LMICs
Data source	Interview and bedside observation	**Automated extraction from electronic records** (eFI2); wearables and sensors
Framing	Deficit-based (ADL/IADL loss)	Complemented by **capacity-based** framing (WHO intrinsic capacity)
Sarcopenia link	Not integrated	Functional tools now feed directly into **sarcopenia case-finding** (SARC-F, grip strength, gait speed)

E. Exam One-Liners

- **Katz and Barthel measure ADL; Lawton measures IADL; Tinetti and TUG measure mobility; MNA nutrition; GDS mood; MMSE cognition; Lubben social.**
- **Barthel is what the patient does, not what they can do** — and has a **ceiling effect** in ambulant patients.
- **FIM has 18 items** across six areas and is the rehabilitation standard.
- **TUG above 12 seconds; Tinetti below 24 of 28; gait speed below 0.6 m/s (Pathy) or 0.8 m/s (EWGSOP2).**
- **0.1 m/s slower gait equals 7% higher fall risk.**
- **PSMS has grooming and ambulation; Katz has continence and transferring** — the classic distinguishing point.
- **Report tells you what they do; performance tells you what they can do** — measure both.
- **WHO ICOPE six domains:** mobility, cognition, vitality, vision, hearing, psychological.
- **A tool is only useful if its result changes the care plan.**

---

3. Functional Assessment in the Elderly

A. 20 High-Yield Points

Why Function Matters

1. **Functional assessment** means measuring **what the person actually does in daily life**. In geriatrics, **function is the common language** and assessment is the core method.
2. In an older person, **function predicts outcome better than diagnosis**. There is **no direct relationship between disease and functioning** — the same illness produces very different disability in different people, so function must be measured separately.
3. Functional decline results from **medical, functional, psychological, social and environmental** risk factors acting together. Hence assessment must be **multidimensional**.
4. A **baseline functional measure is essential** so that progress can be monitored and the effect of treatment or rehabilitation judged. **Reassess at least weekly during rehabilitation.**
5. Three levels are assessed: **impairment** (the deficit) → **activity restriction** (ADL/IADL loss) → **participation restriction** (loss of social role). Participation restriction is **unique to the individual** and depends largely on the ability to adapt.
6. Mobility must always be tested — **about half of people aged 85 and over report difficulty walking**, and gait disorders threaten independence and cause falls.

The Core Measures

7. The functional "review of systems" rests on three measures: **frailty, basic ADLs, and IADLs**.
8. **Principle of hierarchy:** if the patient is **severely ADL-dependent, do not test frailty**; if **robust or pre-frail, do not test basic ADLs**. Match the depth of assessment to the patient and the setting.
9. **Basic ADLs (personal care):** bathing, dressing, toileting, transferring, continence, feeding, grooming — **Katz, Barthel, Lawton PSMS**.
10. **IADLs (tasks needing instruments):** cooking, shopping, housework, laundry, telephone, transport, money, medicines — **Lawton IADL, OARS**.
11. **IADLs are lost first, ADLs later**, so IADL loss is the earlier warning sign. Always ask **"how does she manage?"**
12. **Always record premorbid function as well as present function**, especially after acute illness, a fall or hospital admission.

Domains to Cover

13. **Mobility, gait and balance:** gait speed (**below 0.6 m/s abnormal; 0.6–0.8 high-risk**), **TUG above 12 seconds**, **Tinetti below 24 of 28**, 30-second chair stand, 4-stage balance test, SPPB, Berg Balance Scale.
14. **Two screens are mandatory** in any older person with functional disability: **cognition** (MMSE) and **depression** (GDS or Zung).
15. **Other essential domains:** nutrition (**MNA**, SNAQ, weight loss), **special senses** (whispered voice test, Snellen), **oral health** (GOHAI), **continence**, and **social network and support** (**Lubben Social Network Scale**).
16. **Also assess:** pain, polypharmacy (Beers/STOPP-START), continence, sleep, alcohol (CAGE, SMAST-G), caregiver capacity, financial resources, and **home environment**.

How to Do It in Practice

17. Use **both report and performance**. Reports tell you what the person does **under real living conditions**; performance tests tell you what they can do **under standard conditions**.
18. **Grade each task** as: independent without difficulty / independent but with difficulty or an aid / needs human help / unable even with help. Distinguishing "independent with difficulty" adds prognostic value.
19. **Perform a task analysis** of the specific problem. *Example:* "difficulty dressing" may be due to choosing clothes, remembering to finish, buttoning, reaching overhead, or bending for shoes — pointing respectively to **cognitive impairment, poor finger mobility, or shoulder, back or hip disease**. Each demands a different treatment.
20. **Correlate any new functional loss** with **new drugs or dose changes (including over-the-counter)**, **changes in living conditions**, and **intercurrent acute illness (even mild)**. Remember the **cascade** — impaired gait leads to depression and withdrawal, and **immobility of any cause produces deconditioning that persists after the cause is removed**. Finally, **link assessment to a plan**: rehabilitation steps of **assessment, goal setting, therapy, aids and adaptations, education, psychological support, evaluation and follow-up**.

B. Practical Bedside Scheme

Step 1 — History Premorbid and current ADL/IADL status · exact task that fails · duration and rate of decline · pain · falls · continence · drugs (including over-the-counter) · nutrition and weight change · mood and motivation · social support and caregiver capacity · housing and environment.

Step 2 — Examination Postural blood pressure · vision and hearing screening · **observation of gait** · musculoskeletal (tone, power, joint range of motion, feet) · neurological (focal signs, sensation, proprioception) · **skin over pressure areas** · cognition (MMSE/MoCA) and mood (GDS).

Step 3 — Performance testing Gait speed over 4 metres · Timed Up-and-Go · 30-second chair stand · 4-stage balance · grip strength · observed transfer and stair climbing.

Step 4 — Baseline investigations Complete blood count, blood chemistry, **serum calcium (mg/dL), sodium (mEq/L), creatinine (mg/dL)**, thyroid function, vitamin B12, **25-hydroxy vitamin D**, haemoglobin (g/dL), fasting glucose or HbA1c, urinalysis.

Step 5 — Formulate and act Identify **reversible causes** → treat disease and pain → **deprescribe** → refer to physiotherapy, occupational therapy, dietician and social work → prescribe aids and adaptations → set goals with the patient → arrange **follow-up and case management**.

C. Latest Updates (2023–Present)

1. WHO ICOPE and intrinsic capacity. The five-step pathway screens **six domains — mobility, cognition, nutrition/vitality, vision, hearing and psychological well-being** — and can be done by a professional or by self-assessment through the **ICOPE Monitor** app. Reference centiles for intrinsic capacity and real-world screening data were published in *Nature Aging* (2023, 2024).

2. German DGG S3 Guideline (2024) defines minimum requirements — **six core dimensions in at least 15 minutes** — the first guideline to set a floor standard for functional assessment in hospital.

3. Age-Friendly Health Systems "4Ms." What Matters, Medication, Mentation and Mobility. In an eight-hospital 2024 evaluation of 3,103 admissions, patients receiving all 4Ms had **fewer 30-day emergency visits (16% vs 22%)**; **Mobility generated the highest "act-on" rate (83%)** of the four.

4. Gait speed as the single best marker. A 2025 longitudinal study found **slow gait speed the best single predictor of incident ADL and IADL disability**, outperforming both frailty and sarcopenia as constructs.

5. Italian CGA guideline (2024) — across 117 studies, CGA **maintains functional activities** and reduces hospitalisation, mortality, institutionalisation and delirium.

6. Oncology. ASCO 2023 requires geriatric assessment including **physical function** in all patients over 65 on systemic cancer therapy, using the **Practical Geriatric Assessment**.

7. Indian data. LASI wave-1 (31,464 adults aged 60 and above) found **about 3% with severe ADL disability and about 6% with severe IADL disability**, with **7.4% ADL dependency**. Rural surveys show far higher figures — **ADL disability 13.7% and IADL disability 38.7%** in rural Goa — associated with sex, education, financial dependence, comorbidity, alcoholism and **sensory impairment**. Functional tools are being adapted for community health worker delivery under **NPHCE**.

D. Current Practice Update

Aspect	Pathy's 6th ed. (2022)	Current position (2023–2026)
Core concept	Function measured as **deficit** (ADL/IADL loss)	**WHO shift to intrinsic capacity** — measure **reserve**, not only deficit
Number of domains	Broad, flexible list	**Standardised minimum sets** — ICOPE six domains; German S3 six dimensions in 15 minutes
Gait speed cut-off	Below 0.6 m/s abnormal	**0.8 m/s or less** widely used; slow gait now the best single predictor of incident disability
Who assesses	Interdisciplinary professional team	**Self-assessment and digital screening validated**; community health worker delivery in LMICs
Hospital framing	ACE units and geriatric assessment programmes	**4Ms Age-Friendly framework** — Mobility as a named, auditable care process
Sarcopenia	Not integrated into functional assessment	Functional assessment now **routinely linked to sarcopenia case-finding**

E. Exam One-Liners

- **Function is the common language of geriatrics.**
- **IADLs go first, ADLs later** — IADL loss is the earlier warning.
- **Two screens you must never omit: cognition and depression.**
- **Hierarchy rule:** ADL-dependent, skip frailty; robust, skip basic ADLs.
- **Record premorbid function, not just current function.**
- **TUG above 12 s; Tinetti below 24; gait speed below 0.6 m/s (Pathy) or 0.8 m/s (EWGSOP2).**
- **New functional decline equals a new drug, a new illness, or a new living situation** until proved otherwise.

- **Task analysis converts a vague complaint into a treatable diagnosis.**
- **WHO ICOPE six domains; 4Ms — What Matters, Medication, Mentation, Mobility.**
- **India (LASI): about 3% severe ADL and 6% severe IADL disability; 7.4% ADL dependency.**
- **Assessment without a linked care plan is not an assessment.**

---

4. Functional Assessment in an 85-Year-Old Lady

A. 20 High-Yield Points

Why Function Matters at 85

1. **Functional assessment** means measuring **what the person actually does in daily life**, not just listing her diseases. In geriatrics, **function is the common language** and assessment is the core method.
2. In an 85-year-old, **function predicts outcome better than diagnosis**. There is **no direct relationship between disease and functioning** — the same illness produces very different disability in different people, so function must be measured separately.
3. Functional decline is caused by **medical, functional, psychological, social and environmental** risk factors acting together. Hence assessment must be **multidimensional**, not just physical.
4. A **baseline functional measure is essential** so that progress can be monitored and the effect of treatment or rehabilitation judged.
5. Three levels are assessed: **impairment** (the deficit) → **activity restriction** (ADL/IADL loss) → **participation restriction** (loss of social role). Participation restriction is **unique to the individual** and depends largely on her ability to adapt.
6. Mobility must always be tested at this age — **about half of people aged 85 and over report difficulty walking**, and gait disorders threaten independence and cause falls.

The Core Functional Measures

7. The functional "review of systems" rests on three measures: **frailty, basic ADLs, and IADLs**.
8. **Principle of hierarchy:** if she is **severely ADL-dependent, do not test frailty**; if she is **robust or pre-frail, do not test basic ADLs**. Match the depth of assessment to the patient and the setting.
9. **Basic ADLs (personal care):** bathing, dressing, toileting, transferring, continence, feeding, grooming. Tools — **Katz Index, Barthel Index, Lawton Personal Self-Maintenance Scale**.
10. **IADLs (tasks needing "instruments"):** cooking, shopping, housework, laundry, telephone, public transport, managing money and medicines. Tools — **Lawton IADL scale, OARS-MFAQ**.
11. **IADLs are lost first, ADLs later** — so IADL loss is the earlier warning sign. If difficulty is reported, always ask **"how does she manage?"** — is the task neglected, or is someone else doing it?
12. **Always record premorbid function as well as present function**, especially after an acute illness, fall or hospital admission.

Domains and Recommended Scales

13. **Mobility, gait and balance — the high-yield numbers:** - **Gait speed** over a 4 m track: **below 0.6 m/s is abnormal**; **0.6–0.8 m/s is high-risk**. Each **0.1 m/s fall in gait speed equals a 7% rise in fall risk**. - **Timed Get-Up-and-Go: more than 12 seconds equals high fall risk** (sensitivity 77%, positive predictive value 93% in the community). - **Tinetti Mobility Scale:** 14 tasks, **score below 24 of 28 indicates fall risk** (10–15 minutes). - **30-second chair stand** and **4-stage balance test** (hold each position **10 seconds**); both feed into the **Short Physical Performance Battery (SPPB)**. - **Berg Balance Scale:** 14 items, 0–56 (0–20 wheelchair-bound, 21–40 walks with assistance, 41–56 independent).
14. **Two screens are mandatory** in any older person with functional disability: **cognition** (Folstein MMSE) and **depression** (Yesavage GDS or Zung). Both are common, treatable causes of apparent functional loss.
15. **Other essential domains:** nutrition (**MNA**, SNAQ, BMI, weight loss), **special senses** (whispered voice test or Hearing Handicap Inventory; Snellen chart), **oral health** (GOHAI), **continence**, and **social network and support** (**Lubben Social Network Scale**, OARS social resources section).
16. **RCP/BGS endorsed scales — memorise this table:**

Domain	Recommended scale	Comment
Basic ADLs	**Barthel Index**	Observation of what the patient *does*; **ceiling effect** in ambulatory patients
Vision, hearing, communication	Lambeth Disability Screening Questionnaire	Postal questionnaire
Memory and cognition	**Abbreviated Mental Test (AMT)**	10 questions
Depression	**Geriatric Depression Scale**	30 items; 15-item short form
Subjective morale	Philadelphia Geriatric Center Morale Scale	Distinct from depression

The **Functional Independence Measure (FIM)** scores **18 functions** — self-care, sphincter control, mobility, locomotion, communication, social cognition — and is used in rehabilitation settings.

17. **Quick screening questions usable at the bedside:** "*Do you need help at home?*" (function) · "*Do you have trouble walking or lose your balance? Have you had a fall? Are you afraid of falling?*" (mobility) · "*Are you sad?*" (mood) · "*Have you lost weight?*" (nutrition) · "*Are you on seven or more medicines?*" (polypharmacy) · "*Do you wet yourself?*" (continence).

How to Do It — Practical Method

18. Use **both report and performance**. Reports (from her or a proxy) tell you what she does **under real living conditions**; performance tests tell you what she can do **under standard conditions**. Discrepancies between the two are common and informative.
19. **Grade each task** into: independent without difficulty / independent but with difficulty or an aid / needs human help / unable even with help. Distinguishing "independent with difficulty" adds prognostic value.
20. **Do a task analysis** of the specific problem, then look for a reversible cause and act on it: - *Example:* "difficulty dressing" may be due to choosing clothes, remembering to finish, buttoning, reaching overhead, or bending for shoes — pointing respectively to **cognitive impairment, poor finger mobility, or shoulder, back or hip disease**. Each demands a different treatment. - Always correlate new functional loss with **new drugs or dose changes (including over-the-counter)**, **changes in living conditions**, and **intercurrent acute illness (even mild)**. - Remember the **cascade**: impaired gait leads to depression and social withdrawal; **immobility of any cause causes deconditioning and loss of musculoskeletal flexibility that persists after the cause is removed**. - **Assessment without a plan is worthless** — link findings to the rehabilitation steps: **assessment → goal setting → therapy → aids and adaptations → education → psychological support → evaluation → follow-up**.

B. Latest Updates (2023–Present)

1. WHO ICOPE and "intrinsic capacity" — the major conceptual shift. The ICOPE 5-step pathway is: screening, in-depth assessment of those screening positive, person-centred interventions, long-term follow-up, and community engagement with caregiver support. The screening tool assesses **six functional domains — mobility, cognition, nutrition/vitality, vision, hearing and psychological well-being** — and can be done by a professional or by self-assessment through the **ICOPE Monitor** application; a confirmed deficit triggers full assessment and a personalised care plan. Reference centiles for intrinsic capacity were published in *Nature Aging* (2023, 2024).

2. German DGG S3 Guideline on CGA in Hospital (2024). Minimum requirements are defined as **six core dimensions — self-help ability, mobility, cognitive function/delirium, affect, nutrition and social situation — with a minimum CGA duration of 15 minutes**. This is the first guideline to set a **floor standard for functional assessment**.

3. Age-Friendly Health Systems "4Ms" framework. The 4Ms are **What Matters, Medication, Mentation and Mobility**. In an eight-hospital 2024 evaluation of 3,103 admissions, patients receiving all 4Ms had **fewer 30-day emergency visits (16% vs 22%)** and a trend to lower readmissions; **Mobility generated the highest "act-on" rate of all four Ms (83%)**.

4. Gait speed is being repositioned as the single best functional marker. A 2025 longitudinal study of about 3,600 older adults found that among all components of frailty and sarcopenia, **low physical performance (slow gait speed) was the best single predictor of incident basic and instrumental ADL disability**, and sarcopenia as a whole construct was not associated with increased disability risk. **EWGSOP2 defines low physical performance as gait speed of 0.8 m/s or less.**

5. Italian National CGA Guideline (2024). Across 117 studies, CGA was shown to **maintain functional activities** and to reduce hospitalisation, mortality, institutionalisation and delirium risk.

6. Oncology — ASCO 2023. Geriatric assessment including **all essential domains, of which physical function is one**, should be used in all patients over 65 with cancer, with the **Practical Geriatric Assessment (PGA)** as the recommended tool.

7. Indian context. Functional assessment tools are being adapted for low-resource, high-volume settings under **NPHCE**. Evidence remains predominantly from high-income countries and hospitalised patients. Short multidimensional community tools such as **EHSAS**, deliverable by community health workers, have been validated for rural India precisely because full CGA is impractical with the available workforce. **LASI** provides the national ADL/IADL disability estimates.

C. Current Practice Update

Aspect	Pathy's 6th ed. (2022)	Current position (2023–2026)
Core concept	Function measured as **deficit** (ADL/IADL loss, disability)	**WHO shift to "intrinsic capacity"** — measure **reserve**, not only deficit
Number of domains	Broad, flexible list	**Standardised minimum sets** — ICOPE six domains; German S3 six dimensions in 15 minutes
Gait speed cut-off	**Below 0.6 m/s abnormal**, 0.6–0.8 high-risk	**0.8 m/s or less** widely used (EWGSOP2/AWGS); slow gait now the **best single predictor of incident disability**
Who does the assessment	Professional, interdisciplinary team	**Self-assessment and digital screening validated** (ICOPE Monitor); community health worker delivery in LMICs
Hospital framing	ACE units, geriatric assessment programmes	**4Ms Age-Friendly framework** — Mobility as a named, auditable care process
Sarcopenia link	Not integrated into functional assessment	Functional assessment now **routinely linked to sarcopenia case-finding** (SARC-F → grip strength → gait speed)
Preventive home visits	"Cannot be recommended as routine practice"	Position unchanged; benefit still uncertain (Cochrane 2022)

D. Exam One-Liners

- **Function is the common language of geriatrics; assessment is its core method.**
- **IADLs go first, ADLs go later** — IADL loss is the earlier warning of decline.
- **Katz and Barthel = ADL; Lawton = IADL; Tinetti and TUG = mobility; MNA = nutrition; GDS = mood; MMSE = cognition; Lubben = social.**
- **Barthel Index** is observation of what she *does*, with a **ceiling effect** in ambulatory patients.
- **TUG above 12 s**, **Tinetti below 24/28**, **gait speed below 0.6 m/s** (Pathy) or **0.8 m/s or less** (EWGSOP2) equals high risk.
- **0.1 m/s slower gait equals 7% higher fall risk.**
- Two screens you must never omit: **cognition and depression**.
- **Hierarchy rule:** ADL-dependent, skip frailty testing; robust, skip basic ADL testing.
- New functional decline equals **a new drug, a new illness, or a new living situation** until proved otherwise.
- **WHO ICOPE 6 domains:** mobility, cognition, vitality/nutrition, vision, hearing, psychological.
- **4Ms:** What Matters, Medication, Mentation, Mobility.
- **Assessment without a linked care plan and follow-up is not an assessment.**

5. Physical Self-Maintenance Scale (PSMS)

Lawton and Brody Personal Self-Maintenance Scale

A. Position in Geriatric Assessment

1. The PSMS is one of the **three named basic-ADL instruments** in the CGA framework — **Katz Index, Lawton Personal Self-Maintenance Scale, Barthel Index**.
2. Its assigned basic context is: **strengths and limitations in self-care, basic mobility and incontinence**.
3. These are **the most widely used tools**, used to detect whether the patient has problems performing **activities people must be able to do to survive in the community without help**.
4. **Clinical implication:** patients unable to perform basic ADLs will generally require **12- to 24-hour caregiver support** — in contrast to IADL loss, which threatens independent living in a house or apartment.
5. **Hierarchy rule:** basic ADL scales like the PSMS are appropriate for patients who are **already dependent or disabled**; in a robust or merely pre-frail person, frailty measures, not basic ADLs, should be used.

B. The Scale Itself

Origin and purpose

- Developed at the **Philadelphia Geriatric Center** by **M. Powell Lawton and Elaine M. Brody, 1969**, published in *The Gerontologist*.
- Purpose: to **gauge disability in older people living in the community or in an institution**, for **planning and evaluating treatment**.
- Deliberately designed so that **items target observable behaviours**, not subjective judgements.
- Published as a **paired instrument**: the **6-item PSMS (basic ADLs)** together with the **8-item IADL scale**. In practice the IADL half became far more famous, but both were introduced in the same 1969 paper.
- **Recommended age group: 60 years and over.** Both an **observer-rated** and a **self-rated** version exist; the self-rated version was later incorporated into the **Philadelphia Geriatric Center Multilevel Assessment Instrument (MAI)**.

The six domains

#	Item	What it assesses
1	**Toileting**	Continence and independent use of toilet or commode
2	**Feeding**	Eating without help, spillage, or need for feeding
3	**Dressing**	Selecting and putting on clothes independently
4	**Grooming**	Face, hair, nails, hands — personal appearance
5	**Physical ambulation**	Walking indoors and outdoors, need for aid or wheelchair, bedbound status
6	**Bathing**	Bathing or showering without assistance

Mnemonic: T-F-D-G-A-B — "*The Frail Do Get Assisted Bathing.*"

Response structure and scoring

- **Each item is graded on a 5-point scale**, running from **complete independence (level 1) to complete dependence (level 5)**, with intermediate levels describing progressively more help needed.
- For summary scoring, each item is **dichotomised: 1 point for the independent level, 0 for the dependent levels**.
- **Total score 0–6. 6 equals fully independent; 0 equals totally dependent.**
- Higher score means better function — the **same direction as Katz**, the **opposite direction to a frailty index**.

Psychometrics (original data)

- **Guttman scale reproducibility coefficient 0.96** for the 6-item PSMS (0.93 for the IADL half) — the items form a genuine **hierarchy of loss**.
- **Test–retest reliability 0.94** (0.88 for the IADL half).
- **Inter-rater validity:** two nurses independently rating 36 patients gave a **Pearson correlation of 0.91**.

C. Comparison with the Other Basic-ADL Scales

Feature	PSMS (Lawton–Brody)	Katz Index	Barthel Index
Year	**1969**	**1963**	1965 (modified 1988)
Items	**6**	**6**	10
Domains	Toileting, feeding, dressing, **grooming**, **ambulation**, bathing	Bathing, dressing, toileting, transferring, continence, feeding	Feeding, bathing, grooming, dressing, bowels, bladder, toilet use, transfer, mobility, stairs
Grading	**5 levels per item**, dichotomised	Independent/dependent (A–G)	0/5/10 or 0/5/10/15 per item
Total	**0–6** (6 = independent)	0–6 or A–G	**0–100** (or 0–20)
Unique items	**Grooming and ambulation**	**Continence and transferring**	**Stairs, bowels, bladder**
Best use	Community and institutional older adults; **paired with IADL scale**	Classic hierarchy of ADL loss	**Rehabilitation and stroke** — most granular
Weakness	Little used alone today; overshadowed by its own IADL twin	Crude two-level grading	**Ceiling effect in ambulant patients**

Key contrast for the exam: the **PSMS includes grooming and ambulation but omits transferring and continence as separate items**; the **Katz Index includes continence and transferring but omits grooming and ambulation**. Barthel is the most detailed and is the **RCP/BGS-endorsed scale for ADL measurement**, but suffers a **ceiling effect** in patients who can already walk.

D. Latest Updates (2023–Present)

1. **The PSMS is now rarely used as a standalone instrument.** Its **IADL half has become the global standard** — "the Lawton–Brody scale" in current trial protocols almost always means the IADL scale — while the ADL half has been largely displaced clinically by the **Barthel Index** and **Katz Index**. Contemporary trials routinely pair **"the Lawton and Brody scale" with the Barthel Index**, using Lawton for IADLs and Barthel for ADLs.
2. **Where it survives is dementia and caregiver research.** The **observer-rated PSMS remains in use** for people with dementia and in caregiver-burden studies, because its 5-level gradations detect **partial dependence** better than a binary scale — relevant to recent dementia home-care trials such as LIVE@Home.Path.
3. **Standardisation pressure from new guidelines.** The **German S3 CGA guideline (2024)** mandates "self-help ability/ADL" as one of six core dimensions but **does not prescribe a specific instrument**; **WHO ICOPE** and **ASCO's Practical Geriatric Assessment (2023)** likewise require an ADL domain without naming the PSMS. The trend is **domain-mandated, tool-agnostic** assessment.
4. **Indian context. LASI** and most Indian epidemiological work use **Katz-type or study-specific ADL items** rather than the PSMS. Indian ADL disability prevalence is around **3% severe ADL disability and 7.4% any ADL dependency** in LASI wave-1, and much higher in rural surveys (about 13.7% in rural Goa).

E. Current Practice Update

Aspect	Pathy's 6th ed. (2022)	Current position (2023–2026)
Status of PSMS	Listed as one of three recommended basic-ADL tools	**Rarely used alone**; its **IADL companion scale dominates**; ADL measurement has moved to Barthel and Katz
Which tool to use	No preference between Katz, Lawton and Barthel	**Domain is mandated, tool is not** (German S3 2024, WHO ICOPE, ASCO PGA)
Grading detail	Not discussed	The **5-level gradation is the PSMS's remaining advantage** — better at detecting partial dependence in dementia and caregiver research
Reporting direction	Not stated	Confusion persists because **Barthel rises with independence out of 100, PSMS out of 6, Katz uses letters** — always state the scale and its direction

F. Exam One-Liners

- **PSMS = Lawton and Brody, 1969, Philadelphia Geriatric Center** — published in the **same paper** as the far more famous Lawton IADL scale.
- **Six items: Toileting, Feeding, Dressing, Grooming, Physical ambulation, Bathing.**
- **Each item graded on 5 levels; dichotomised to give a total of 0–6.**
- **6 equals fully independent, 0 equals totally dependent** — higher is better.
- **Guttman reproducibility 0.96, test–retest 0.94, inter-rater r = 0.91.**
- **PSMS has grooming and ambulation; Katz has continence and transferring** — the classic distinguishing point.
- **Basic ADL loss means 12–24 hour caregiver support; IADL loss means a threat to independent community living.**
- **IADLs are lost first, basic ADLs later** — which is why Lawton's IADL half outlived his ADL half in clinical use.
- **Barthel is the RCP/BGS-endorsed ADL scale** and has a **ceiling effect in ambulant patients**.

Part II — Frailty and Sarcopenia

6. Frailty Syndrome — Etiopathogenesis, Assessment, Management and Prevention

A. 20 High-Yield Points

Definition and Concept

1. **Definition:** Frailty is the condition in which a person **loses the ability to carry out important, practised social activities of daily living when exposed to psychological or physical stress**. It must be **distinguished from disability** — frailty is a state of **pre-disability**, now recognised as a **public health priority**.
2. **Epidemiology:** By Fried's definition, about **6.9% of the older population is frail**. **Females are more often frail than males** of the same age. Sarcopenia, its main substrate, affects **13–24% aged 60–70** and about **50% over age 80**.
3. **Frailty threshold:** Vitality peaks between **20 and 30 years**, then VO2max, cardiac output, balance and muscle strength gradually decline. **Exercise delays** the decline; **disease accelerates** it. If a person lives long enough, **everyone eventually crosses the frailty threshold**.
4. **Pathway: Healthy → Frailty → Disability → Hospitalisation → Institutionalisation → Death**, with **recovery still possible** from the earlier stages. A **stressful event precipitates the frail person into disability**.
5. **Two conceptual models:** the **phenotype model** (a biological syndrome with a pathophysiological substrate) and the **cumulative deficit model** of Rockwood (frailty equals an increasing number of deficits and disabilities; essentially a comorbidity index).
6. **Comparison of the three frailty scales — must memorise:**

Cardiovascular Health Study (Fried)	Study of Osteoporotic Fractures (SOF)	IANA FRAIL scale
Weight loss (**10 lb in a year**)	Weight loss	**F**atigue
Exhaustion (self-report)	Inability to rise from a chair **5 times without using arms**	**R**esistance (climb one flight of stairs)
Weakness (**grip strength, lowest 20%**)	Reduced energy level	**A**erobic (walk one block)
Walking speed (**15 ft, slowest 20%**)	—	**I**llnesses (**more than 5**)
Low physical activity (kcal/week, lowest 20%)	—	**L**oss of weight (**5%**)

Etiopathogenesis

7. **Core mechanism:** In most people frailty results from **failure to generate adequate muscle power** and/or **failure of executive function** to use the available power appropriately. The **backdrop is normal physiological ageing**, which interacts with **genes, lifestyle, environment and disease**.
8. **Five major causes: disease · pain · decreased food intake · decline in executive function · excess muscle loss (sarcopenia)**.
9. **Disease to frailty:** Many diseases produce an **excess of cytokines**, which reduce **muscle mass, food intake and cognitive function**, and also lower the anabolic hormone **testosterone**.
10. **Congestive heart failure — the classic model:** falling VO2max, then left-sided failure causes **intestinal wall oedema → bacterial translocation → endotoxin (lipopolysaccharide)** → immune activation and **TNF-alpha release → anorexia, muscle loss, weight loss, hypoalbuminaemia, hypocholesterolaemia**. - **Best predictors of poor outcome in CHF are weight loss and hypocholesterolaemia.** - **Angiotensin II** cleaves actomyosin, clearing muscle protein via the **ubiquitin–proteasome system**; **ACE inhibitors reverse weight loss and frailty** in some CHF patients.
11. **Other diseases:** - **COPD** — reduced endurance, poor intake, **raised resting metabolic rate and thermic energy of eating**, low testosterone, high cytokines. - **Diabetes mellitus** — low testosterone, raised angiotensin II, raised cytokines, peripheral neuropathy, sarcopenia, reduced executive function, accelerated atherosclerosis; **mortality is increased in frail diabetics**. - **Anaemia** — reduced endurance and strength, orthostasis, falls, reduced mobility, increased disability and mortality; **erythropoietin and darbepoetin-alpha reverse many of these**.
12. **Reversible causes not to be missed: Polymyalgia rheumatica** — painful muscles with proximal myopathy, **raised ESR**, and **corticosteroids completely reverse the frailty**; this **totally reversible condition is often misdiagnosed**. Also **hyperthyroidism, hypothyroidism, hypoadrenalism** (insidious onset) and **arthritis or pain leading to immobility and muscle loss**.
13. **Anorexia of ageing (decreased food intake):** causes are multifactorial — - Social: **isolation, dysphoria** - Sensory: **decline in smell, rise in taste threshold** - Gastric: **decreased fundal compliance and adaptive relaxation** leading to rapid antral filling and **early satiety** - Hormonal: **excess cholecystokinin** from the duodenum after a fatty meal; **high circulating cytokines** - In men: **falling testosterone leads to rising leptin and greater anorexia** — men lose more food intake over the lifespan.
14. **MEALS-ON-WHEELS mnemonic for treatable weight loss: M**edications (digoxin, theophylline, cimetidine) ·

Emotional (depression) · **A**lcoholism, elder abuse, anorexia tardive · **L**ate-life paranoia · **S**wallowing problems · **O**ral factors · **N**osocomial infections (e.g. tuberculosis) · **W**andering and other dementia-related causes · **H**yperthyroidism, hypercalcaemia, hypoadrenalism · **E**nteral problems (gluten enteropathy) · **E**ating problems · **L**ow-salt, low-cholesterol and other therapeutic diets · **S**tones (cholecystitis).

15. **Sarcopenia — the engine of frailty:** - Defined as lean tissue **more than 2 SD below young adults**. - **Best measure is appendicular skeletal mass by DEXA divided by height squared**; MRI, CT, bioelectrical impedance and ultrasound are alternatives. - **Sarcopenic obesity ("fat frail")** — those who stay fat while losing muscle have the **highest future disability and mortality**. - **Myosteatosis** (fat infiltration into muscle) is a **separate condition linked to insulin resistance** via altered insulin-receptor substrate and GLUT transporter function.

16. **Determinants of muscle loss:** best predictors are **age, energy intake, physical activity, IGF-1, testosterone and cytokines**. Also **hypovitaminosis D** (reduced strength, more falls); **testosterone falls about 1% per year from age 30 in men**; **myostatin** acts via activin IIb receptors; **motor unit firing rate falls after age 80**; **CNTF declines**; **TNF-alpha and IL-6 excess** reduce strength; **high homocysteine and peripheral vascular disease** impair muscle blood flow. **Sarcopenia may originate at birth** — grip strength correlates with birth weight in the Hertfordshire cohort.

Assessment

17. **Screen everyone, then assess.** Use a **rapid validated tool** first — the **FRAIL scale** is the simplest and quickest. Then confirm and characterise with a fuller measure. - **Phenotype tools:** Fried phenotype, **Frailty Trait Scale**, **FRAIL index**. - **Cumulative deficit tools: Frailty Index**, **Clinical Frailty Scale**. - Frailty is one of the **three core functional dimensions of CGA**, with basic ADLs and IADLs. Remember the **hierarchy rule** — do not test frailty in someone already severely ADL-dependent. - **Progranulin** is described as a biomarker of frailty.

Management

18. **Management algorithm using the FRAIL screen — the single most examinable table:**

Component	Action
Fatigue	Exclude **depression, sleep apnoea, hypothyroidism, anaemia, hypotension, vitamin B12 deficiency**
Resistance	Treat sarcopenia with **resistance exercise plus adequate protein (1 g/kg/day)**
Aerobic	**Aerobic and balance exercises**
Illnesses	If **more than 5 illnesses**, consider **deprescribing**
Loss of weight	Look for treatable causes — **MEALS-ON-WHEELS**

19. **Principle of management:** Frailty may have a **single reversible cause** — for example **anaemia**, where correction with iron, folate, vitamin B12 or erythropoietin reverses the frailty. More often it is due to the **interplay of hormones and cytokines with disease and poor-quality nutrition**, requiring **careful assessment of causative factors and a multifaceted treatment regimen**.

20. **Prevention and other therapies:** - **Exercise is the central preventive measure** — it delays the slope towards the frailty threshold; **creatine with exercise** may improve muscle performance. - **Testosterone replacement** increases muscle mass **even in non-hypogonadal males**, and pharmacological or replacement doses improve **muscle strength, power and function**. **Selective androgen receptor modulators** — steroidal (nandrolone, oxymetholone, oxandrolone) and non-steroidal (2-quinoline, coumarin, phthalimide, bicalutamide, acetothiolutamide) — are under investigation. - **Vitamin D** — regular sun exposure **15–30 minutes a day** without sunblock, or **800–1000 IU/day**, especially in long-term-care residents. - **Deprescribing and treating reversible disease** are as important as exercise and nutrition.

B. Latest Updates (2023–Present)

1. Kim and Rockwood, NEJM 2024 — the current definitive review. Assessing older patients for frailty enables clinicians to **tailor clinical care, including decisions about stressful treatments**. Management is stratified into **four stages — robust, pre-frailty, frailty and end-stage frailty** — with thresholds such as **Fried phenotype 0, Frailty Index below 0.10, and Clinical Frailty Scale 1–3 defining "robust."** This concept of **"frailty-guided clinical care"** is the major shift since the textbook.

2. NHS England and BGS — proactive frailty care (2023–2025). In December 2023 NHS England published *Proactive care: providing care and support to people living at home with moderate and severe frailty*. The pathway is: identify the cohort (including by **electronic frailty index, eFI**), confirm with the **Clinical Frailty Scale**, then perform a **CGA** covering physical, functional, social, environmental and psychological domains **alongside a medication review**. The original eFI is being replaced nationally by the **eFI2**, available to primary care from 2025. The BGS *Joining the dots* (2023) and *Be proactive* (2024) supersede the 2014 "Fit for Frailty" guidance.

3. ICFSR Global Consensus on Exercise (2025). Structured exercise prescriptions should be **customised and monitored like any other medical treatment**, considering dose–response relationships, with a **multifaceted regimen of aerobic, resistance, balance and flexibility training** through structured and incidental activity.

4. Italian National CGA Guideline (2024). Across 117 studies, CGA reduced hospitalisation, mortality,

institutionalisation and delirium risk, improved appropriateness of drug prescription, and maintained functional activities. **CGA remains the definitive management vehicle for frailty.**

5. WHO ICOPE — intrinsic capacity as the mirror image of frailty. ICOPE screens **six domains — mobility, cognition, nutrition, vision, hearing and psychological well-being** — with confirmed deficits triggering full assessment and a personalised prevention and care plan. Intrinsic capacity (reserve) and frailty (deficits) are two faces of the same coin.

6. Indian data — much higher burden than Western figures. LASI wave-1 (26,058 adults aged 60 and above, Fried phenotype) found **frailty in 29.2% and pre-frailty in 58.8%**, higher among **women (37.3%), the illiterate (37%) and rural residents (31%)**, ranging from **14.5% in Uttarakhand to 41.3% in Arunachal Pradesh**, and strongly associated with depression. A 2025 systematic review and meta-analysis of 330,007 community-dwelling Indians found a **pooled frailty prevalence of 36%** (48% by frailty index, 31% by Fried phenotype). In LASI, the most discriminant frailty component was **grip strength in men (79.1%)** and **physical activity in women (81.6%)**.

C. Current Practice Update

Aspect	Pathy's 6th ed. (2022)	Current position (2023–2026)
Prevalence	**About 6.9%** frail (Fried, Western data)	**India: about 29% frail, 59% pre-frail** (LASI); pooled Indian meta-analysis **36%** — the Western figure badly underestimates Indian burden
Purpose of diagnosis	Identify and treat reversible causes	Additionally **"frailty-guided clinical care"** — deciding intensity of drugs, surgery and screening
Staging	Frail versus not frail	**Four stages** — robust, pre-frail, frail, end-stage frailty — each with different treatment goals
Case-finding	Clinician-led FRAIL screen	**Population-level automated screening** (eFI2) from records, then CFS to confirm
Exercise	"Resistance, aerobic and balance exercises"	**Prescribed like a drug** — specified dose, frequency, progression and monitoring
Anabolic drugs	Testosterone and SARMs presented with optimism	**No approved pharmacotherapy for frailty**; testosterone reserved for symptomatic hypogonadism; SARMs still investigational
Framing	Deficit-based (frailty)	Complemented by **capacity-based** framing (WHO intrinsic capacity)

D. Exam One-Liners

- **Frailty equals pre-disability**; disability is the outcome. Never use them interchangeably.
- **Fried phenotype 5 criteria:** weight loss, exhaustion, weakness, slow gait, low activity. **3 or more equals frail, 1–2 equals pre-frail.**
- **FRAIL:** Fatigue, Resistance, Aerobic, Illnesses (more than 5), Loss of weight (5%).
- **SOF, 3 items:** weight loss, cannot rise from chair 5 times without arms, reduced energy.
- **Two models:** phenotype (Fried) versus cumulative deficits (Rockwood — Frailty Index, Clinical Frailty Scale).
- **CHF frailty chain:** gut oedema → bacterial translocation → endotoxin → TNF-alpha → anorexia plus muscle loss.
- **Best predictors of poor outcome in CHF:** weight loss and hypocholesterolaemia.
- **Never miss polymyalgia rheumatica** — high ESR, and steroids fully reverse the frailty.
- **MEALS-ON-WHEELS** for treatable weight loss.
- **Sarcopenia gold standard measure:** appendicular skeletal mass by DEXA divided by height squared.
- **"Fat frail" (sarcopenic obesity)** carries the worst prognosis.
- **Protein target in frailty: 1 g/kg/day** plus resistance exercise.
- **More than 5 illnesses means deprescribe.**
- **India:** frailty about 29%, pre-frailty about 59% (LASI); higher in women and rural residents.

7. Comparison of the Tests Used to Identify or Screen for Frailty

A. 18 High-Yield Points

The Fundamental Divide

1. **All frailty tools fall into two conceptual families**, and this is the single most important contrast to state: - **Phenotype model** — frailty is a **biological syndrome** with a pathophysiological substrate, expressed as a **limited number of physical domains**. Task and performance based. - **Cumulative deficit model** — frailty is the **accumulation of deficits** (diseases, impairments, abnormal findings) and reflects **biological ageing**. Count based.
2. **Tools by family:** - **Phenotype: Fried frailty phenotype, Frailty Trait Scale, FRAIL index** - **Cumulative deficits: Frailty Index, Clinical Frailty Scale**
3. **A second, equally important divide is purpose: screening or case-finding** (rapid, high sensitivity, applied to everyone) versus **assessment or diagnosis** (detailed, drives the care plan). Screening is never an end in itself — a positive screen must trigger a **comprehensive geriatric assessment**.

The Three Scales Compared Directly

4. Reproduce this table verbatim:

Cardiovascular Health Study (Fried)	Study of Osteoporotic Fractures (SOF)	IANA FRAIL scale
Weight loss (**10 lb in a year**)	Weight loss	**F**atigue
Exhaustion (self-report)	Inability to rise from a chair **5 times without using arms**	**R**esistance (climb one flight of stairs)
Weakness (**grip strength, lowest 20%**)	Reduced energy level	**A**erobic (walk one block)
Walking speed (**15 ft, slowest 20%**)	—	**I**llnesses (**more than 5**)
Low physical activity (**kcal/week, lowest 20%**)	—	**L**oss of weight (**5%**)

5. **Contrast within the table:** Fried requires **equipment (dynamometer, stopwatch, activity questionnaire) and population-based cut-offs (lowest 20%)**; SOF and FRAIL were developed because they are **easier to use in the clinic** and are validated. FRAIL is **purely self-report** — no equipment at all.
6. **Scoring is common to all three: 0 equals robust, 1–2 equals pre-frail, 3 or more equals frail.** This shared cut-off makes them directly comparable even though the items differ.

Contrasting the Individual Tools

7. **Fried phenotype — the reference standard.** Advantages: biologically grounded, identifies a **treatable pre-disability state**, gives the prevalence figure of about **6.9%**. Disadvantages: **time-consuming, needs equipment and normative data**, excludes cognition and mood, cannot be applied to a bed-bound or wheelchair-dependent patient.
8. **SOF index — the pragmatic three-item version.** Advantage: **no grip dynamometer or activity questionnaire**; the chair-stand replaces both. Disadvantage: even narrower domain coverage than Fried.
9. **FRAIL scale — the fastest phenotype screen.** Advantage: **five questions, purely verbal, usable over the telephone**; the "I" item uniquely captures **multimorbidity**, and the tool **maps directly onto a management algorithm**. Disadvantage: self-reported, so subject to recall bias and denial.
10. **Frailty Index (Rockwood–Mitnitski)** — counts deficits and expresses them as a **proportion (deficits present divided by deficits counted)**. Advantages: **continuous variable**, grades severity finely, predicts outcomes well, and can be built from **existing records**. Disadvantages: needs **30 or more variables**, laborious by hand, and it is **not a syndrome** — it deliberately mixes disease, disability and impairment; it **can be considered a comorbidity index**.
11. **Clinical Frailty Scale** — a **global clinical judgement** on a 9-point pictorial scale. Advantages: **seconds to apply**, no equipment, works in acutely unwell patients. Disadvantages: **subjective and rater-dependent**, conflates frailty with disability at the severe end.
12. **Frailty Trait Scale** — phenotype-based but multidimensional; used mainly in research and in ADVANTAGE/ICFSR recommendations.
13. **VES-13 (Vulnerable Elders Survey-13)** — a self-report tool **for identifying vulnerable older people in the community**, measuring **age, self-rated health, physical function and ADL disability**. It has been **compared directly against full CGA** in older men with prostate cancer on androgen deprivation.
14. **aCGA (abbreviated CGA)** — a **pre-screen in older cancer patients**, with published scoring and interpretation. **VES-13 and aCGA screen for vulnerability, not for the frailty phenotype** — they exist to decide **who needs a full CGA**, not to diagnose a syndrome.
15. **SIOG task force position:** screening tools exist and have been used successfully in settings such as the emergency room, **but globally they were poorly tested**, and the task force **could not recommend any specific tool or approach above others** — general geriatric experience should guide the choice.

Choosing Between Them

16. **Principle of hierarchy:** frailty testing is **only worthwhile in the robust-to-pre-frail range**. If a patient is already **severely ADL-dependent, do not measure frailty** — the ADL deficits, not the frailty score, determine risk and management.
17. **Setting dictates the tool.** Adapt the assessment to the patient's complexity and to the place where it is carried out — a brief screen in the office or home, a fuller interdisciplinary work-up in an inpatient or day-hospital setting.
18. **Whichever tool is used, the endpoint is the same:** a positive screen must be followed by a search for the **causative factors** (disease, pain, poor nutrition, sarcopenia, executive dysfunction) and a **multifaceted treatment regimen** — because frailty may have a **single reversible cause** such as anaemia or polymyalgia rheumatica.

B. Master Comparison Table

Tool	Model	Items	Method	Time	Output	Best setting	Main limitation
Fried phenotype (CHS)	Phenotype	5	Performance plus report	10–15 min	3 categories	Research; specialist clinic	Equipment; population cut-offs; excludes cognition
SOF index	Phenotype	3	Performance plus report	~5 min	3 categories	Clinic, community	Very narrow domains
FRAIL scale	Phenotype	5	Self-report only	**15–30 sec**	3 categories	**Primary care, telephone, India and LMICs**	Recall bias
Frailty Index	Deficits	**30 or more**	Record or interview	20–30 min	**Continuous (0–1)**	Research; record-derived	Laborious; not a syndrome
Clinical Frailty Scale	Deficits (judgement)	9 levels	Clinician judgement	**Under 1 min**	Ordinal 1–9	**Acute hospital, ED, surgery**	Rater-dependent
PRISMA-7	Mixed	7	Self-report questionnaire	**Under 2 min**	Score 3 or more equals frail	Community, ED, postal	Age is one item, so over-calls the very old
VES-13	Vulnerability	13	Self-report	~5 min	Score 3 or more equals vulnerable	**Geriatric oncology**, community	Detects disability more than frailty
G8	Vulnerability	8	Self-report	~5 min	14 or less equals abnormal	**Geriatric oncology**	Nutrition-weighted
Gait speed alone	Performance	1	Performance	~1 min	**Below 0.8 m/s**	Any	Single domain
Timed Up-and-Go	Performance	1	Performance	~1 min	**Above 10 s** (frailty) / **above 12 s** (falls)	Any ambulant patient	Needs ambulation
eFI / eFI2	Deficits	36 or more	**Automated from records**	0 min	Continuous or graded	**Population health, primary care**	Data-quality dependent; no clinical judgement

C. The Six Axes of Contrast

Axis	Contrast
1. Model	Phenotype (syndrome, treatable) **versus** cumulative deficits (risk index, prognostic)
2. Purpose	Screen (sensitive, fast) **versus** assess (specific, comprehensive)
3. Method	Self-report **versus** performance-based **versus** clinician judgement **versus** automated record-based
4. Output	Categorical (robust/pre-frail/frail) **versus** ordinal (CFS 1–9) **versus** continuous (FI 0–1)
5. Overlap with disability	Fried and FRAIL **exclude** disability by design; CFS and FI **include** it
6. Actionability	FRAIL maps to an intervention algorithm; FI and CFS mainly stratify **prognosis**

D. Latest Updates (2023–Present)

1. Head-to-head diagnostic accuracy — the emergency department evidence. A systematic review and meta-analysis of prospective diagnostic accuracy studies (2000–2022) covering seven ED frailty screens (PRISMA-7, CFS, VIP, FRESH, BPQ, TRST, ISAR) in 1,663 participants found a **pooled sensitivity of 0.85 and specificity of 0.77** against CGA; **PRISMA-7 was the most sensitive (0.84–0.98) but only moderately specific (0.64–0.78)**, whereas **CFS was the least sensitive but the most specific**.

2. Direct comparison against CGA. In 197 ED attendees aged 70 and above (49% frail by CGA), the **CFS (AUROC 0.91) and PRISMA-7 (AUROC 0.90) were significantly more accurate** than the 3- and 4-item VIP (both 0.84), with **no significant difference between CFS and PRISMA-7**. In a separate cohort of 313 patients (58% frail by CGA), **PRISMA-7 was most accurate (AUC 0.88), followed by CFS (0.83) and ISAR (0.78)**.

3. PRISMA-7 systematic review (2025). PRISMA-7 is completed in **under 2 minutes**, showed an **AUC of 0.85 against the Fried phenotype**, and is a practical alternative to the time-consuming and resource-intensive phenotype.

4. Automated screening has arrived. The **electronic frailty index (eFI2)** is replacing the original eFI across English GP systems, available from 2025. The recommended sequence is now **eFI to identify the cohort → Clinical Frailty Scale to confirm → CGA to assess and plan**. This is the key structural change: **screening is no longer clinician-initiated but population-triggered**.

5. NEJM 2024 frailty-guided care. Kim and Rockwood define **robust** as Fried phenotype 0, **Frailty Index below 0.10**, and **CFS 1–3**, and stratify management across four stages. The tools are now used not just to label but to **decide how intensively to treat**.

6. Guideline position on choice of tool. ICFSR: **all adults aged 65 and over should be offered screening for frailty using a validated rapid frailty instrument suitable to the specific setting or context** (strong recommendation, low certainty), followed by clinical assessment of all who screen positive. In primary care, the **FRAIL scale can be answered in 15 to 30 seconds**, predicted disability and mortality at 9 years, and **performed as well as the Fried phenotype and the SOF index**.

7. Oncology has standardised on a single tool. The German S3 guideline recommends specific screening instruments **mainly in the oncological setting (the G8 questionnaire)**, and ASCO 2023 recommends the **Practical Geriatric Assessment (PGA)**.

8. Indian data — the tool changes the answer. In a meta-analysis of 330,007 community-dwelling Indians, pooled frailty prevalence was **36%**, but varied markedly by instrument — **48% by frailty index versus 31% by Fried phenotype**. In LASI, the most discriminant phenotype component was **grip strength in men (79.1%)** and **physical activity in women (81.6%)**, arguing for sex-specific interpretation.

E. Current Practice Update

Aspect	Pathy's 6th ed. (2022)	Current position (2023–2026)
Which tool to use	No tool preferred; SIOG "could not recommend any specific tool above others"	**Setting-specific standardisation** — FRAIL in primary care, CFS in acute/ED/surgery, G8 or PGA in oncology, MPI for prognosis, eFI2 for population screening
Accuracy data	None given	**PRISMA-7 most sensitive; CFS most specific**; both AUROC about 0.90 against CGA
Who initiates screening	Clinician, opportunistically	**Automated record-triggered** cohort identification (eFI2), then clinician confirmation
Fried phenotype role	The operational definition	Increasingly a **research reference standard**, replaced clinically by rapid proxies
Purpose of the score	Diagnose a treatable pre-disability state	Additionally used to **titrate treatment intensity**
Gait speed	Listed among mobility measures	Emerging as the **best single-item screen**; 0.8 m/s or less the operative threshold

F. Exam One-Liners

- **Two families:** phenotype (Fried, SOF, FRAIL, FTS) versus cumulative deficits (Frailty Index, CFS, eFI).
- **All three classical scales score identically:** 0 robust, 1–2 pre-frail, **3 or more frail**.
- **Fastest self-report screen is FRAIL (15–30 s); fastest clinician screen is CFS (under 1 min).**
- **Most sensitive ED screen is PRISMA-7; most specific is the Clinical Frailty Scale.**
- **Frailty Index needs 30 or more deficits** to predict death, institutionalisation and decline reliably.
- **Fried excludes disability by design; CFS and FI include it** — which is why FI and CFS give higher prevalence.
- **Gait speed below 0.8 m/s, TUG above 10 s, PRISMA-7 of 3 or more** — the three quickest positive triggers.
- **Oncology screens are G8 and VES-13**; the **aCGA** is a pre-screen for full CGA.
- **A screen is never a diagnosis** — every positive screen must be followed by CGA.
- **Do not measure frailty in someone already severely ADL-dependent.**

8. Sarcopenia in Ageing — Diagnostic Criteria, Investigation and Management

A. 18 High-Yield Points

Definition and Concept

1. **Definition:** Sarcopenia is the **excessive loss of muscle mass that occurs in older people**, usually defined as lean tissue **more than two standard deviations below that of young adults**.
2. **Prevalence: 13–24% at ages 60–70**, rising to **about 50% over age 80**.
3. **Relationship to frailty:** Sarcopenia is one of the **five major causes of frailty** and is the **biological substrate of physical frailty** — the two are **two sides of the same "impaired" coin**, and a combined entity, **"physical frailty and sarcopenia,"** is emerging.
4. **Sarcopenia is strongly correlated with disability.** Most sarcopenic people also lose fat, but a subset stay fat while losing muscle — the **"sarcopenic obese" or "fat frail"** — and longitudinally these have the **highest future disability and mortality**.
5. **Myosteatosis is a separate condition** — fat infiltration *into* muscle, related to **insulin resistance**. Mitochondrial failure or high circulating triglycerides cause intramyocellular triglyceride accumulation, altering the **insulin receptor substrate and GLUT transporter**.

Etiopathogenesis

6. **Final common mechanism:** an **imbalance in the everyday renewal cycle of muscle** — either **excess atrophy and apoptosis**, or **reduced hypertrophy and satellite cell production**.
7. **Best epidemiological predictors of muscle mass and strength: age, energy intake, physical activity, IGF-1, testosterone and cytokines**.
8. **Hormonal: Testosterone falls about 1% per year from age 30 in men**, and rapidly between 20 and 40 in women. Testosterone **diverts pluripotential stem cells away from the fat lineage into the muscle lineage**, produces **satellite cells** (essential for muscle repair), **stimulates muscle protein synthesis** and **inhibits the ubiquitin–proteasome pathway**.
9. **Growth factors: Myostatin** is the negative regulator, acting via **activin IIb receptors**; a **soluble activin IIb receptor** scavenges activin and produces muscle and bone hypertrophy in rodents, monkeys and humans. Double myostatin deletion produces the "mighty mouse," and marked hypertrophy in cattle, whippets and one human. **IGF-1 and mechanogrowth factor (MGF)** rise with resistance exercise in young but **not** elderly subjects.
10. **Neural: Motor unit firing rate falls significantly after age 80**. **Ciliary neurotrophic factor (CNTF)** declines with age in parallel with strength. **C-agrin and neurofilament light chain** are raised in a subset of sarcopenic people.
11. **Inflammatory, vascular and developmental: Excess TNF-alpha and IL-6 cause loss of muscle strength**; high CRP and IL-6 correlate with reduced handgrip strength. **Raised homocysteine and peripheral vascular disease** impair muscle blood flow. **Hypovitaminosis D** is associated with decreased strength and falls. In the **Hertfordshire cohort, grip strength correlated with birth weight** — sarcopenia may originate at birth. **ACE I/I** carriers generate more power with exercise than **D allele** carriers.
12. **Signalling pathway:** anabolic limb — **GH/testosterone/creatine → IGF-1/MGF → PI3K → AKT → mTOR → S6K1 → hypertrophy**; catabolic limb — **cytokines → TNFR1 → IKK → NF-kappaB**, and **glucocorticoids/diabetes → FOXO → Atrogin-1 (MAFbx) and MuRF1 → ubiquitin–proteasome → protein degradation → atrophy**; **myostatin → SMAD3** suppresses MyoD, opposed by **follistatin**.

Investigation

13. **Best measure of muscle mass: appendicular skeletal mass measured by DEXA, divided by height in metres squared**. Alternatives: **MRI, CT, or bioelectrical impedance**. **DEXA and MRI measures are highly correlated**, and **ultrasound** is proving to be an excellent measure.
14. **Imaging refinement: MRI with specific protocols is superior to DXA**, particularly for visceral tissues, and can additionally measure **muscle volume, muscle fat infiltration and ectopic fat**. **BIA is accessible even in primary care** but relies on an approximation between total body weight, lean tissue and fat — reasonable for arms and legs, **less precise for visceral regions**.
15. **Circulating biomarkers:** - **P3NP** (procollagen type III N-terminal peptide) — related to muscle mass and strength, stronger in women. - **D3-creatine (methyl-d3) dilution** — urine D3-creatinine enrichment correlates highly with MRI total muscle mass and **may be superior to DXA**. - **C-terminal agrin fragment (CAF)** — a neuromuscular junction breakdown product; associated with **DXA muscle mass, grip strength and gait velocity**. - **Myostatin, follistatin, GDF-11** — inconsistent; the myostatin–muscle mass relationship was confirmed **only in men** in the I-Lan study.

Management

16. **Core treatment: Resistance exercise plus adequate protein (1 g/kg/day)** — the explicit treatment for sarcopenia in the FRAIL management algorithm; plus **aerobic and balance exercise**. **Creatine with exercise** may improve muscle performance.
17. **Pharmacological options: Testosterone replacement increases muscle mass even in non-hypogonadal males**; pharmacological or replacement doses in hypogonadal men **increase muscle strength and power**, with demonstrated **functional improvement**. **Selective androgen receptor modulators** — steroidal (nandrolone, oxymetholone, oxandrolone) and non-steroidal (2-quinoline, coumarin, phthalimide, bicalutamide, acetothiolutamide) — remain investigational.
18. **What NOT to use, and what else to do: Growth hormone is not recommended** — it produced **no positive effect on muscle strength, VO2max or functional capacity**, did not add to resistance training, and **men with the highest GH levels had the highest mortality**. **Vitamin D** — regular sun exposure **15–30 minutes a day** without sunblock, or **800–1000 IU/day**; **25-OH-vitamin D should be above 30 ng/mL**. **Treat the anorexia of ageing** using the **MEALS-ON-WHEELS** mnemonic.

B. Diagnostic Criteria

EWGSOP2 (2019) — the European standard: F-A-C-S

Step	Test	Cut-off
Find	**SARC-F questionnaire**	**4 or more**
Assess (strength)	**Handgrip strength** or **5-time chair stand**	**Below 27 kg (men), below 16 kg (women)**; or **above 15 s**
Confirm (mass)	**ASMI (DXA or BIA)**	**Below 7.0 kg/m2 (men), below 5.5 kg/m2 (women)**
Severity (performance)	**Gait speed**, SPPB, TUG, 400 m walk	**0.8 m/s or less**; SPPB 8 or less; TUG 20 s or more

Diagnostic logic: low strength equals **probable sarcopenia** → plus low mass equals **confirmed sarcopenia** → plus low performance equals **severe sarcopenia**. **Muscle strength, not mass, is the primary parameter.** EWGSOP2 chose round-number cut-offs to foster ease of use.

AWGS 2019 — the Asian standard, more relevant for Indian practice

Case-finding uses **calf circumference (below 34 cm men, below 33 cm women)**, **SARC-F of 4 or more**, or **SARC-CalF of 11 or more**. **"Possible sarcopenia"** is then diagnosed by **handgrip below 28 kg (men) or below 18 kg (women)**, or **5-time chair stand of 12 seconds or more** — and AWGS recommends **starting lifestyle intervention immediately at this stage without waiting for muscle mass measurement**. Low physical performance equals **6-m walk below 1.0 m/s, SPPB 9 or less, or 5-time chair stand 12 s or more**; low muscle mass equals **DXA below 7.0 kg/m2 (men) and below 5.4 kg/m2 (women)**, or **BIA below 7.0 kg/m2 (men) and below 5.7 kg/m2 (women)**.

Head-to-head comparison

Feature	EWGSOP2 (2019)	AWGS 2019	FNIH
Screening	SARC-F 4 or more	**SARC-F / SARC-CalF / calf circumference**	—
Grip strength	**Below 27 / 16 kg**	**Below 28 / 18 kg**	Below 26 / 16 kg
Chair stand	**Above 15 s**	**12 s or more**	—
ASMI (DXA)	**Below 7.0 / 5.5 kg/m2**	**Below 7.0 / 5.4 kg/m2**	ALM adjusted for BMI
Gait speed	**0.8 m/s or less**	**Below 1.0 m/s**	Below 0.8 m/s
Unique feature	Strength is the **primary** criterion; severity staging	**"Possible sarcopenia"** allows treatment in primary care without DXA	Uses **ALM adjusted for BMI**

Exam point: *The criteria set you choose changes the prevalence — Indian data show 48% by frailty/deficit-type indices versus 31% by phenotype criteria. Always state which criteria you are applying.*

C. Investigation — Practical Scheme

Step 1 — Confirm the diagnosis SARC-F / SARC-CalF / calf circumference → grip strength (Jamar dynamometer) → 5-time chair stand → gait speed over 4 metres → DXA or BIA for ASMI.

Step 2 — Exclude mimics and secondary causes (the differential of the weak old person) - **Haemoglobin (g/dL)** for anaemia, **TSH** for thyroid disease, **ESR and CRP** for polymyalgia rheumatica and inflammation - **Serum creatinine (mg/dL) plus eGFR** — beware **masked renal failure**, where creatinine is normal despite severe CKD because of low muscle mass; use **Cockcroft-Gault or cystatin C** - **Fasting glucose or HbA1c**, **serum calcium (mg/dL)**, **vitamin B12**, **25-OH vitamin D (ng/mL)** - **Serum testosterone (ng/dL)** in symptomatic men; **CPK** if myopathy is suspected - **Nutritional assessment** — MNA, SNAQ, weight trend, BMI - **Medication review** — steroids, statins, and Beers/STOPP-START screening

Step 3 — Assess consequences Falls risk, ADL and IADL status, bone mineral density (sarcopenia and osteoporosis co-travel), cognitive and depression screening.

D. Latest Updates (2023–Present)

1. GLIS — the first globally endorsed conceptual definition (2024). The **Global Leadership Initiative in Sarcopenia** defined sarcopenia as a **generalised skeletal muscle condition that is potentially reversible**, applying consistently across conditions, care settings and both clinical and research contexts. Its **core components are muscle mass, muscle strength and muscle-specific strength (strength divided by size)**, while **impaired physical performance is a consequence, not a diagnostic component**. This is the single biggest conceptual change since the textbook.

2. GLIS outcomes consensus (2025). From an individual patient data meta-analysis of 32 databases and 5,116 participants using the SarQoL instrument, people with sarcopenia have **significantly reduced quality of life (mean difference −12.32)**, with the largest difference using **AWGS criteria (−17.65)**.

3. Regional guidelines have proliferated. Australia/New Zealand consensus (2023), Singapore clinical practice guidelines (2022), Chinese guidelines (2025), and an AWGS update. DXA remains a standard but has **limitations in reflecting true muscle quality and predicting outcomes**; **deuterium-labelled creatine dilution** shows promise, and myokine biomarkers (**IL-6, IGF-1, irisin, myostatin, gelsolin**) may improve diagnostic precision.

4. Exercise — now a formal prescription. The **ICFSR global consensus (2025)** states that structured exercise prescriptions should be **customised and monitored like any other medical treatment**, with a **multifaceted regimen of aerobic, resistance, balance and flexibility training**. Combined resistance–aerobic regimens at **2–3 sessions per week** improve muscle strength by **20–35%**.

5. Nutrition — the numbers have firmed up. Optimised protein intake of **1.0–1.5 g/kg/day** with **leucine or vitamin D** enhances muscle synthesis and functional capacity. To overcome the **anabolic resistance** of ageing muscle, **25–30 g of high-quality protein and 2.8–3 g of leucine per meal, at least twice daily**, is recommended; requirements rise to **1.2–1.5 g/kg/day in inflammatory disease**. **HMB 3 g daily** helps **preserve** muscle during illness, immobility or post-surgical recovery — the evidence is stronger for **preventing loss than for building muscle**.

6. Pharmacotherapy — still investigational. SARMs and myostatin inhibitors such as **bimagrumab** increase lean mass by **3–5%** in phase II studies; a single dose of the ActRIIB antibody **bimagrumab** produced a **6–8% increase in muscle mass within a month** with a simultaneous reduction in fat mass. **GDF-15-neutralising antibodies** are emerging as anti-catabolic agents. **No agent is approved for sarcopenia anywhere as of 2026 — exercise plus protein remains the only proven therapy.**

7. Vitamin D — nuance added. Recent trials show **active vitamin D (0.75 microgram/day)** can improve muscle strength and reduce fall risk in sarcopenic individuals, but benefits are largely confined to those who are **deficient**.

8. Indian relevance. Grip strength was the **most discriminant frailty and sarcopenia component in Indian men (79.1%)** in LASI, and **AWGS cut-offs, not EWGSOP2, should be applied** to Indian patients. **BIA is the practical mass-measurement modality** in most Indian centres given limited DXA access — AWGS explicitly provides a BIA cut-off for this reason.

E. Current Practice Update

Aspect	Pathy's 6th ed. (2022)	Current position (2023–2026)
Core definition	**"Excessive loss of muscle mass"** (more than 2 SD below young adults)	**GLIS 2024:** a **muscle disease** defined by **mass plus strength plus muscle-specific strength**; **mass alone is insufficient**
Primary diagnostic parameter	**Muscle mass** (ASM/height squared by DEXA)	**Muscle strength** (EWGSOP2) — mass confirms, performance stages severity
Physical performance	Not part of the definition	EWGSOP2 and AWGS: severity marker; **GLIS: a consequence, not a criterion**
Screening	Not described	**SARC-F, SARC-CalF, calf circumference** now standard entry points
Reversibility	Implied	**GLIS explicitly states sarcopenia is potentially reversible**
Protein target	**1 g/kg/day**	**1.0–1.2 g/kg/day baseline; 1.2–1.5 g/kg/day if inflammatory disease**, with **leucine 2.8–3 g per meal**
Testosterone and SARMs	Presented optimistically	**Not approved**; used only for symptomatic hypogonadism after individualised discussion
New drugs	Not covered	**Bimagrumab, apitegromab, anti-GDF-15** — all investigational
Ultrasound	"Proving to be an excellent measure"	Increasingly used point-of-care; **D3-creatine dilution may surpass DXA**

F. Exam One-Liners

- **Sarcopenia is the biological substrate of physical frailty** — two sides of the same impaired coin.

- **Prevalence: 13–24% at 60–70 years; about 50% over 80.**
- **Textbook gold standard measure:** appendicular skeletal mass by DEXA divided by height squared.
- **EWGSOP2 sequence is F-A-C-S** (Find, Assess, Confirm, Severity); **strength is the primary criterion**.
- **AWGS grip cut-offs: below 28 kg men, below 18 kg women** — use these for Indians. **EWGSOP2: below 27 / 16 kg.**
- **AWGS ASMI (DXA): below 7.0 / 5.4 kg/m2; (BIA): below 7.0 / 5.7 kg/m2.**
- **AWGS "possible sarcopenia" allows treatment to start in primary care without DXA.**
- **Sarcopenic obesity ("fat frail") has the worst prognosis.**
- **Myosteatosis is not sarcopenia** — it is fat *in* muscle, linked to insulin resistance.
- **Atrogin-1 (MAFbx) and MuRF1** are the two key E3 ligases of the ubiquitin–proteasome atrophy pathway.
- **Beware masked renal failure** — normal creatinine with severe CKD because there is no muscle.
- **Only proven treatment is resistance exercise plus adequate protein.** No drug is approved.
- **GH is useless or harmful** — no gain in strength or function; highest GH levels equal highest mortality.

Part III — Disability, Immobility and Rehabilitation

9. Causes of Chronic Disability in the Elderly — Prevention and Management

A. 20 High-Yield Points

Definitions and Framework

1. **Disability is an umbrella term** covering **impairments, activity limitations and participation restrictions** (WHO **ICF, 2001**). **Functioning** is the corresponding positive umbrella term.
2. **The ICF has two parts:** - **Part 1 — Functioning and Disability:** Body Functions and Structures; Activities and Participation - **Part 2 — Contextual Factors:** Environmental Factors; Personal Factors
3. **Key ICF definitions: Impairment** is a problem in body function or structure · **Activity** is the execution of a task · **Activity limitation** is difficulty executing it · **Participation** is involvement in a life situation · **Participation restriction** is a problem in that involvement.
4. **The ICF replaced the older impairment–disability–handicap model** and explicitly recognises that **the social and physical environment shapes functioning** — disability is not a property of the person alone.
5. **The vicious cycle:** Parkinson's disease (impairment) → difficulty with self-care and mobility (activity limitation) → cannot pursue hobbies (participation restriction) → falls and fractures hip → loses confidence → becomes isolated, withdrawn and depressed. **Feedback loops make functioning spiral downwards.**

Causes of Chronic Disability

6. **The major disabling events: stroke, hip fracture, other fall-related injury, and deconditioning following a major illness**.
7. **The major ongoing disabling diseases: osteoarthritis and Parkinson's disease** are named explicitly; also stroke sequelae, amputation, and chronic cardiorespiratory disease.
8. **"Almost any acute or chronic disease can reduce functioning"** — but there is a specific list of **common but easily overlooked causes of dysfunction in older people** that must be memorised: - **Impaired cognition** - **Impaired special senses** (vision, hearing, balance) - **Unstable gait and mobility** - **Poor health habits** (alcohol, smoking, lack of exercise) - **Poor nutrition** - **Polypharmacy** - **Incontinence** - **Psychosocial stress** - **Depression**
9. **The "geriatric giants" as classical causes — the series of I's: Immobility, Instability (falls), Incontinence, Intellectual impairment, Infection, Impairment of vision and hearing, Irritable colon, Isolation (depression), Inanition (malnutrition), Impecunity, Iatrogenesis, Insomnia, Immune deficiency, Impotence.** Morley's **"new geriatric giants"** add **frailty, sarcopenia, anorexia of ageing and cognitive impairment**.
10. **Determinants of ACTIVITY restriction — four factors:** - **Type of impairment** (nature and severity of the disease) - **Presence of associated impairments** (pre-existing problems matter as much as the new one) - **Degree of physiological reserve** - **Level of physical fitness**
11. **Determinants of PARTICIPATION restriction:** - **Intrinsic:** attitude, personality, ability to adjust, cultural issues - **Extrinsic:** financial resources, housing, other resources, **social supports (spouse, family, neighbours, friends, pets)**
12. **The single greatest difference from younger patients is reduced physiological reserve** — recovery is **prolonged and may not return to the premorbid state**. **But note the caution:** even very old people recover from major illness, and **failure to progress can seldom be blamed on reduced reserve alone**. Of **far greater importance is the lack of activity and physical fitness** typical of older people in modern societies.
13. **Site matters as much as size** — in stroke, large lesions in some brain areas may be asymptomatic while **small lesions in strategic areas cause major disability**.
14. **Disability cascades:** impaired gait leads to depression and reduced social functioning; **immobility of any cause — even after the cause is removed — produces secondary impairment from deconditioning and loss of musculoskeletal flexibility**.
15. **Psychological determinants:** onset of impairment provokes a **grief reaction — denial, anger, depression, then acceptance**. **Over 40% of older people become depressed after myocardial infarction**, and this worsens prognosis; high rates also follow stroke despite rehabilitation. At one extreme are **highly motivated** patients; at the other, those who **adopt the "sick role"**, disengage and surrender autonomy.
16. **Kemp's motivation model** — motivation is driven by four elements: the person's **wants, beliefs, rewards for achievement**, and the **cost** (pain and effort). The first three push forward; cost pulls back. The clinician intervenes by **setting goals, challenging incorrect beliefs, establishing rewards and minimising cost**.

Assessment of the Disabled Older Person

17. **Diagnostic approach:** to identify the cause of new disability, look for **(a) worsening of an existing chronic disease, (b) a new acute disease, or (c) one of the common occult diseases listed in point 8**. Do this through a **refocused history guided by the functional deficit** and a **focused examination**.

18. **Examination must always include** — in addition to heart, lungs, extremities and neurology — **postural blood pressure, vision and hearing screening, and careful observation of gait**, plus **MMSE**. **Basic labs:** complete blood count and blood chemistry panel, plus targeted tests.

Prevention and Management

19. **Rehabilitation — the UN definition:** "*a goal-orientated and time-limited process aimed at enabling an impaired person to reach an optimum mental, physical and/or social functional level, thus providing him or her with the tools to change his or her own life*." It may **compensate for lost function (technical aids)** or **facilitate social readjustment**.
20. **The eight steps in the rehabilitation process: 1. Assessment → 2. Setting goals → 3. Therapy → 4. Aids and adaptations → 5. Education → 6. Psychological support → 7. Evaluation → 8. Follow-up.** Attached principles: - **Early intervention is crucial** — anticipate and avoid problems, because once established they may be irremediable (for example, shoulder subluxation in flaccid hemiplegia). - **Goals must be realistic, agreed by the whole team AND the patient**, with **short-term (intermediate) and long-term (final) goals** and flexible time frames. **Patients with ambitious goals make greater progress** — so set **ambitious but achievable** targets. - **Education is a vital component** — it gives the patient knowledge, skills and attitudes to minimise activity and participation restriction, and doubles as **secondary prevention**. - **Age-associated decline is reversible: weight training increases muscle strength and improves functional capacity in older people**, and lifelong trainers have muscle strength comparable to sedentary middle-aged people. - **Assessment must be linked to long-term follow-up and often formal case management.**

B. Structured Classification of Causes

Category	Causes
Neurological	Stroke, Parkinson's disease, dementia, peripheral neuropathy, spinal cord compression, normal-pressure hydrocephalus
Musculoskeletal	**Osteoarthritis**, osteoporosis and fragility fracture (especially hip and vertebral), sarcopenia, polymyalgia rheumatica, amputation
Cardiorespiratory	Heart failure, ischaemic heart disease, COPD, peripheral vascular disease
Sensory	**Cataract, glaucoma, age-related macular degeneration, presbycusis** — the most under-treated reversible causes
Psychological	**Depression**, anxiety, **fear of falling**, adjustment failure or "sick role"
Metabolic and systemic	Diabetes and its complications, hypothyroidism, anaemia, malnutrition, chronic kidney disease
Iatrogenic	**Polypharmacy**, anticholinergic burden, physical restraints, bed rest, unnecessary hospitalisation, catheters
Environmental and social	Poor housing, no grab-bars or rails, inaccessible transport, poverty, isolation, absent carer, elder abuse

C. Prevention — The Three-Level Framework

Primary prevention (prevent the impairment) - **Exercise** — the single most important intervention; multicomponent (aerobic, resistance, balance, flexibility) - Vascular risk control (blood pressure, glycaemic control, lipids, smoking, alcohol) - **Immunisation** (influenza, pneumococcal, herpes zoster, COVID-19) - Osteoporosis and fall prevention; adequate protein and vitamin D - Cognitive and social engagement

Secondary prevention (detect impairment early, before it limits activity) - **Comprehensive geriatric assessment** with periodic functional screening - Screening for the occult causes listed above — cognition, mood, vision, hearing, gait, nutrition, continence - **Medication review and deprescribing** (Beers, STOPP-START) - Preventive home visits — benefits are not definitively confirmed, so not routine practice

Tertiary prevention (prevent activity limitation progressing to participation restriction) - **Early rehabilitation**, aids and adaptations, home modification - Carer training and support; **avoid restraints and unnecessary bed rest** - Social re-engagement, day centres, transport solutions

D. Management — Practical Scheme

1. **Define the disability precisely** — task analysis: which step of dressing, walking or toileting fails?
2. **Search for reversible causes** — a new drug, a new illness, an intercurrent infection, a change in living conditions. *When a reversible cause is found, a simple treatment may eliminate the functional impairment.*
3. **Optimise medical management** — treat the disease, control pain, correct anaemia, thyroid, B12 and vitamin D; **deprescribe**.
4. **Refer to the multidisciplinary team** — physician, nurse, physiotherapist, occupational therapist, social worker; optimally dietician, pharmacist, speech therapist.
5. **Set goals with the patient** — short and long term, ambitious but achievable.
6. **Therapy** — exercise (resistance for strength, aerobic for endurance, balance for falls), gait re-training, ADL re-training.

7. **Aids, appliances and home adaptation** — walking aids, grab bars, raised toilet seat, shower stool, hearing aid, spectacles, footwear.
8. **Address mood and motivation** — treat depression; challenge catastrophic beliefs; use Kemp's framework.
9. **Support the carer** — carer burden is a leading cause of institutionalisation.
10. **Discharge planning, evaluation and long-term follow-up with case management.**

E. Latest Updates (2023–Present)

1. Global burden — the disability picture has shifted. Osteoarthritis is a leading cause of pain and disability among middle-aged and older adults; in 2020 it affected **7.6% of the global population (more than 595 million people)**, with cases projected to rise **60–100% by 2050**, and it is **expected to become the leading cause of disability worldwide by 2030**. Hearing loss ranks as the **third most common cause of years lived with disability globally**, with its age-standardised rate still rising among those aged 60 and above. Regional analyses identify **diabetes mellitus, age-related hearing loss and low back pain** as the leading causes of *disability* in older people, distinct from the leading causes of *death*.

2. Indian data — essential for the exam. LASI wave-1 (31,464 adults aged 60 and above) found **about 3% with severe ADL disability and about 6% with severe IADL disability**. In the same cohort, **44.3% had no geriatric condition, 32.7% had one, 15.9% two and 7.1% had three or more**; specific prevalences were **underweight 26.9%, dizziness 14.5%, impaired vision 13.7%, impaired hearing 9.6%, impaired cognition 9.3%, major depressive disorder 8.2%, injurious falls 5.7% and incontinence 4.0%**, with **7.4% having ADL dependency**. A rural Goa study found much higher figures — **ADL disability 13.7% and IADL disability 38.7%** — associated with sex, education, financial dependence, comorbidities, alcoholism and **sensory impairment**.

3. Multimorbidity combinations matter more than counts. Among 197 multimorbidity combinations in LASI, the combination of **hypertension, arthritis and high depressive symptoms** was associated with **greater ADL–IADL disability than any other combination**; population attributable fractions were about **22.5% for hypertension plus depression, 21.6% for hypertension plus arthritis and 23.5% for arthritis plus depression**. **Adding depression to any physical multimorbidity combination sharply increases disability.**

4. Frailty is the strongest single predictor of incident disability. A systematic review reported a **pooled odds ratio of 9.82 (95% CI 4.71–20.46) for incident ADL disability in frail older adults**; in LASI, frailty was strongly associated with depression (OR 2.09) and ADL difficulty (OR 1.75).

5. Exercise is now prescribed like a drug. The **ICFSR global consensus (2025)** states structured exercise prescriptions should be **customised and monitored like any other medical treatment**, with a **multifaceted regimen of aerobic, resistance, balance and flexibility training**.

6. Proactive, population-level disability prevention. NHS England and BGS proactive care uses the **electronic frailty index** to identify at-risk older people, the **Clinical Frailty Scale** to confirm, and a **CGA covering physical, functional, social, environmental and psychological domains plus a medication review** to plan care. WHO **ICOPE** screens six domains of intrinsic capacity with confirmed deficits triggering a personalised prevention and care plan.

7. CGA reduces disability outcomes. The Italian national CGA guideline (2024), from 117 studies, found CGA reduces hospitalisation, mortality, institutionalisation and delirium risk, improves prescribing appropriateness and **maintains functional activities**.

F. Current Practice Update

Aspect	Pathy's 6th ed. (2022)	Current position (2023–2026)
Leading disabling conditions	Stroke, hip fracture, osteoarthritis, Parkinson's disease	**Osteoarthritis projected to be the world's leading cause of disability by 2030**; **hearing loss third leading cause of YLD**; low back pain and diabetes prominent
Framing	ICF — deficits	Complemented by **WHO intrinsic capacity and functional ability** framing
Who to target	Those with a recent disabling event	**Population-level proactive identification** before the event (eFI2, ICOPE screening)
Depression	Recognised as common post-MI and post-stroke	Quantified as a **major independent driver of disability in multimorbidity combinations** (Indian PAF about 22–24%)
Frailty	A separate concept	The **strongest predictor of incident ADL disability (OR about 9.8)** — the key preventable precursor
Exercise	"Weight training increases strength and improves functional capacity"	**Formal, dose-specified, monitored prescription**
Access	Only 2% of eligible Canadians access pulmonary rehabilitation; 1% musculoskeletal rehabilitation; worse in developing countries	Gap persists; **India relies on NPHCE with inconsistent implementation and ASHA-delivered community screening**

G. Exam One-Liners

- **Disability equals impairment plus activity limitation plus participation restriction** (ICF, 2001).
- **ICF Part 1 is Functioning and Disability; Part 2 is Contextual Factors.**
- **Four determinants of activity restriction:** type of impairment, associated impairments, physiological reserve, physical fitness.
- **Participation restriction is determined mainly by the ability to adapt** — and is largely neglected in clinical practice because it is subjective.
- **Eight steps of rehabilitation:** Assessment, Goals, Therapy, Aids and adaptations, Education, Psychological support, Evaluation, Follow-up.
- **Set ambitious but achievable goals** — ambitious goal-setters progress further.
- **Over 40% of older people are depressed after myocardial infarction**, worsening prognosis.
- **Nine overlooked causes of dysfunction:** cognition, senses, gait, health habits, nutrition, polypharmacy, incontinence, psychosocial stress, depression.
- **"Bed is bad"** — immobility itself causes secondary disability through deconditioning.
- **Frailty confers about a 10-fold risk of incident ADL disability.**
- **In India (LASI): about 3% severe ADL and 6% severe IADL disability; ADL dependency 7.4%.**
- **Hypertension plus arthritis plus depression is the most disabling multimorbidity combination in Indian elders.**
- **Under-investment in rehabilitation is a false economy** leading to avoidable, costly institutional care.

10. Assessment of Disability in Older People and Rehabilitation

A. 20 High-Yield Points

Framework and Terminology

1. **WHO ICF (2001)** replaced the impairment–disability–handicap model. **Disability** is the collective term for **impairments, activity limitations and participation restrictions**; **functioning** is the positive umbrella term. - **Part 1 — Functioning and Disability:** Body Functions and Structures; Activities and Participation - **Part 2 — Contextual Factors:** Environmental Factors; Personal Factors
2. **Key definitions: Impairment** — problem in body function or structure · **Activity** — execution of a task · **Activity limitation** — difficulty executing it · **Participation** — involvement in a life situation · **Participation restriction** — problem in that involvement · **Environmental factors** — the physical, social and attitudinal environment.
3. **Vicious cycles:** Parkinson's disease → difficulty with self-care and mobility → cannot pursue hobbies → falls and fractures hip → loses confidence → isolated, withdrawn, depressed. Rehabilitation can interrupt the cycle **at any point** — preventing progression of impairment to activity restriction, of activity restriction to participation restriction, and preventing further impairments.
4. **Rehabilitation (UN definition):** "*a goal-orientated and time-limited process aimed at enabling an impaired person to reach an optimum mental, physical and/or social functional level, thus providing him or her with the tools to change his or her own life.*"
5. **Determinants — must reproduce:**

Determinants of ACTIVITY restriction	Determinants of PARTICIPATION restriction
Type of impairment (nature and severity of disease)	**Intrinsic:** attitude · personality · ability to adjust · cultural issues
Presence of associated impairments	**Extrinsic:** financial resources · housing · other resources · **social supports (spouse, family, neighbours, friends, pets)**
Degree of physiological reserve	
Level of physical fitness	

6. **The greatest difference from younger patients is reduced physiological reserve** — recovery is prolonged and may not reach premorbid level. **But** even very old people recover from major illness, and **failure to progress can seldom be attributed to reduced reserve alone**; of **far greater importance is lack of activity and physical fitness**. Weight training increases strength and improves functional capacity even in old age.

Assessment

7. **Assess BEFORE entry into a rehabilitation programme** — to confirm the problems are remediable and to plan management. **Patient selection is difficult:** it is unfair to subject someone who cannot benefit to a demanding programme and raise false expectations; equally, **those who can benefit even to a limited extent must not be denied access**.
8. **Assess "both the problem in the individual and the individual with the problem."** Determine the nature and severity of **all** impairments, new and long-standing.
9. **Obtain a baseline measure of functional status** so progress can be monitored and efficacy reviewed. **Reassess at least weekly.**
10. **Assessment of activity restriction:** - Begin with the **clinical history** — determine **premorbid as well as current** function. - Use **ADLs** (washing, grooming, dressing, toileting, eating) and **IADLs** (meals, telephone, laundry, housework, gardening, shopping, public transport). - If difficulty is reported, **ask how the person manages** — is the task neglected, or does someone help? - **Busy clinicians can estimate the extent of restriction with a few simple questions and observations**; detailed standardised scaling is generally left to therapists and social workers. - No consensus exists on the best scale; **highly valid and reliable scales are resource-intensive or intrusive**, and the lack of uniformity inhibits comparative research.
11. **RCP/BGS endorsed standardised scales — high-yield table:**

Domain	Recommended scale	Comment
Basic ADLs	**Barthel Index**	Observation of what the patient *does*; **ceiling effect** in ambulatory patients
Vision, hearing, communication	Lambeth Disability Screening Questionnaire	Postal questionnaire
Memory and cognition	**Abbreviated Mental Test (AMT)**	10 questions from the Roth–Hopkins test
Depression	**Geriatric Depression Scale**	30 items; 15-item short form
Subjective morale	Philadelphia Geriatric Center Morale Scale	Distinct from depression, some overlap

Value of standardised scales: they allow **information exchange across acute, rehabilitation and community settings**, permit measurement of effectiveness, foster comparison of treatments, and **minimise repeated collection of identical information**. The **Functional Independence Measure (FIM)** scores **18 functions** — self-care, sphincter control, mobility, locomotion, communication, social cognition.

12. **Assessment of participation restriction:** each person interacts uniquely with their environment, so participation restriction is **unique to the individual** and determined mainly by the **ability to adapt**. Some redefine rather than lose their social role, and a few people's lives are **enriched** by developing an impairment. Because such measurements are **subjective and unstable over time (influenced by mood)**, assessing participation restriction is **largely neglected in both clinical and research settings**.

The Rehabilitation Process

13. **Eight steps — the single most examinable table: 1. Assessment → 2. Setting goals → 3. Therapy → 4. Aids and adaptations → 5. Education → 6. Psychological support → 7. Evaluation → 8. Follow-up.** The steps **overlap, occur simultaneously and must be regularly revisited**.

14. **Two overarching principles:** - **Early intervention is crucial** — avoidable restriction develops soon after impairment onset and, once established, may be irremediable. *Flaccid hemiplegia risks shoulder subluxation; proper handling and limb positioning immediately post-stroke minimises this.* - **A team approach is essential — doctors manage impairment, remedial therapists manage activity restriction, social workers manage participation restriction**; nursing has a holistic brief influencing both. Teams need co-location for informal exchange plus **regular formal meetings** to review progress, revise goals, plan discharge and organise follow-up.

15. **Goal setting:** goals must be **realistic**, account for premorbid status, and be agreed by **all team members and particularly the patient** — a programme is seriously compromised if key people differ on the target. Sometimes **modest goals** are right (wheelchair independence rather than walking for an amputee). **Patients with ambitious goals make greater progress**, so the pragmatic approach is **"ambitious but achievable."** Set **short-term (intermediate) and long-term (final)** goals with **flexible, regularly reviewed time frames**.

16. **Therapy — team roles: Doctors** manage the presenting problem and coexisting impairments, minimise risk factors and anticipate complications (in embolic stroke: monitor anticoagulation, control hypertension, manage angina and diabetes). **Occupational therapists** assess and enhance ADL competence. **Physiotherapists** target cardiorespiratory, neuromuscular and locomotor function. **Speech and language therapists** address communication and swallowing. Plus clinical psychologists, podiatrists, dieticians.

17. **Aids and adaptations:** - **A caution:** providing an aid is **not always best — it can foster dependence rather than independence**. Conversely, **those in real need often do not obtain even basic aids**. - **Match the adviser to the aid:** occupational therapists for general aids, physiotherapists for mobility aids, speech therapists for communication aids, audiologists for hearing aids, continence advisors for continence aids — and they must **follow up to ensure aids are used and maintained**. - **Prostheses** replace body parts; **orthoses** are applied externally to support, improve function, or restrict or promote movement. - **Home adaptation:** handrails, ramps, improved shower and toilet access. **Early occupational therapy input, often including a home visit**, ensures modifications are appropriate and timed to facilitate discharge.

18. **Education and psychological support:** - **Education is a vital component and doubles as secondary prevention** — patients must understand their disease, its implications, prospects of recovery, risk factors and lifestyle modification; **family should be included**. - Psychological support requires **good listening skills**; people fear the unknown, and explanation reduces anxiety. Patients may fear **future** events (another stroke) as much as the present one. - **Regular reassurance and positive feedback are simple and effective**; a **careless negative remark can profoundly demoralise** a patient. Feedback must never become dishonest or generate false expectations. - Support **spouses and relatives** too; hospital settings foster a **"therapeutic community"**, and **self-help groups** provide ongoing support after discharge.

19. **Discharge planning, evaluation and follow-up:** - **Discharge is a transition in rehabilitation, not its conclusion.** Many benefit from continuing outpatient therapy to gain further or prevent loss of gains. **Some follow-up is essential.** - Programmes are **resource-intensive and must demonstrate effectiveness** — at minimum compare patients on entry and at discharge, using increasingly standardised data.

20. **Settings and specific programmes:** - **Settings:** stand-alone rehabilitation hospitals · designated rehabilitation units · undifferentiated wards · nursing and residential homes · **day hospitals** · community day centres · outpatient centres · **the patient's own home**. Ideally a **range of options** should be available. - **Co-location by condition** (stroke units, ortho-geriatric units) fosters expertise, research and training. **Stroke units improve survival and functional outcomes; evidence for ortho-geriatric units is less convincing.** - **Intermediate care** is intensive, short-term, community- or home-based rehabilitation to prevent hospitalisation, facilitate early discharge and maximise independent living. **Community-based rehabilitation only suits the least disabled and well-supported.** - **Cardiac rehabilitation** — SIGN's **four phases**; exercise-based programmes reduce **all-cause mortality by 27%**. After uncomplicated infarct, **feed self from the outset, sit out of bed within 24 hours, walk to the toilet within 48 hours**. - **Pulmonary rehabilitation** reduces dyspnoea and fatigue, increases exercise capacity, improves quality of life and may prevent hospitalisation; benefits are seen in the **"old old"**, and **the greatest benefits are in the most**

impaired patients. Exercise: **at least 3 times a week, at least 20 minutes, heart rate at least two-thirds of maximum, for at least 6 weeks and preferably 3 months**. Gains are lost without a maintenance programme. - **Musculoskeletal disorders** — multidisciplinary rehabilitation is of **proven benefit only in chronic back pain, other chronic pain, and after hip fracture in frail elderly people**. - **The elderly amputee** — most lower-limb amputations follow **peripheral vascular disease**. **Perioperative mortality 10–30%, two-year survival 40–50%, five-year survival 30–40%** — unchanged over 50 years. **Preserving the knee is critical:** it takes **40% more energy to walk with an above-knee than a below-knee prosthesis**. **Prescription of a prosthesis is almost always indicated regardless of age**, yet older amputees are less likely to receive one even after adjusting for comorbidity and function. - **Emerging technologies** — remote sensors and wearables give **objective, quantitative** data (existing scales are subjective and prone to observer variability), **virtual reality and video-capture platforms**, video games for post-stroke upper limb (must give feedback, avoid a sense of failure, and match challenge to capability), and GPS, robotics and mobile systems enabling **early transfer from hospital to home-based rehabilitation**.

B. Latest Updates (2023–Present)

1. Rehabilitation is now a recognised global public health priority. A **World Health Assembly resolution endorsed in May 2023** marked the first time in 75 years that rehabilitation was recognised as a **core global public health priority**, aligned with the Rehabilitation 2030 action areas; WHO has supported approximately **78 countries** to strengthen rehabilitation services. At the **3rd Global Rehabilitation 2030 meeting (July 2023)** the **World Rehabilitation Alliance** was launched with 82 founding organisations, along with the **Package of Interventions for Rehabilitation (PIR)** — evidence-based interventions for **20 health conditions** produced with Cochrane Rehabilitation — plus the Rehabilitation Competency Framework and health-financing and workforce guidance.

2. EuGMS definition of geriatric rehabilitation. Geriatric rehabilitation is **"a multidimensional approach of diagnostic and therapeutic interventions, the purpose of which is to optimize functional capacity, promote activity and preserve functional reserve and social participation in older people with disabling impairments"** — a definition following the WHO ICF framework. Common admission diagnoses are **stroke, hip fracture, post-orthopaedic surgery, osteoarthritis and other musculoskeletal disease, movement disorders, oncological disease and cardiopulmonary conditions**.

3. Predictors of successful geriatric rehabilitation are now defined. Discharge back to the community is associated with **higher cognition, better mobility at admission, higher baseline functional independence, lower multimorbidity, fewer acute hospitalisation days and greater social support**. In those over 90, **higher functional independence at admission and fewer medications** predict community discharge, whereas **multiple comorbidities, hospital admission in the past year and lack of social work involvement** predict readmission.

4. "No rehabilitation potential" is no longer acceptable. Rehabilitation for older people **reduces hospital admissions, length of stay and carer burden while improving functional outcomes**. The 2024 British Geriatrics Society report "*Reablement, Rehabilitation and Recovery: everyone's business*" argued that **rehabilitation should be a component of all services caring for older people**, and that rationing access on the dubious basis of "no rehabilitation potential" should be a thing of the past.

5. Meta-analytic evidence. A 2024 systematic review and meta-analysis in *BMC Medicine* of randomised trials in adults 65 and above undergoing inpatient or geriatric day-hospital rehabilitation versus usual care assessed **mortality, care-home admission and functional status** as primary outcomes.

6. Prehabilitation — a new stage before rehabilitation. Structured pre-operative exercise and education programmes are being trialled in older surgical patients; **cognitive prehabilitation** is being evaluated for its effect on **postoperative delirium and delayed neurocognitive recovery**. Evidence for physical prehabilitation before hip replacement remains **conflicting**.

7. Digital and mHealth rehabilitation. A systematic review of mHealth in geriatric rehabilitation found most outcomes improved in both arms with a **majority favouring the intervention**, but with **high risk of bias and minimal reporting of integration into daily practice**.

8. Assessment tools converging with CGA standards. The **German S3 guideline (2024)** sets six mandatory CGA dimensions — **self-help ability, mobility, cognition/delirium, affect, nutrition and social situation**. **WHO ICOPE** screens **mobility, cognition, nutrition, vision, hearing and psychological well-being**.

C. Current Practice Update

Aspect	Pathy's 6th ed. (2022)	Current position (2023–2026)
Status of rehabilitation	"Lack of investment is a false economy" — an advocacy point	**WHA resolution (May 2023)** — rehabilitation formally a **core global public health priority** and part of universal health coverage
Definition	UN definition (goal-orientated, time-limited)	**EuGMS definition** specific to geriatric rehabilitation — adds **preserving functional reserve** and **social participation**
Patient selection	"Assess to ensure problems are remediable"	**"No rehabilitation potential" rejected as a basis for rationing**; predictors of success now evidence-based
Assessment scales	Barthel, AMT, GDS, FIM	Supplemented by **standardised CGA minimum domain sets** and **objective digital and wearable measures**
Participation restriction	"Largely neglected in clinical and research settings"	Foregrounded by ICF-aligned WHO PIR and patient-reported outcome measures
Technology	Virtual reality, video capture and sensors "emerging"	**mHealth in routine geriatric rehabilitation** — positive but with high risk of bias; not yet standard
Timing	Early intervention after the disabling event	**Prehabilitation** — intervention *before* the planned insult, including **cognitive prehabilitation**
Ortho-geriatric units	"Evidence less convincing" than stroke units	Now embedded in **NELA and hip fracture best-practice tariffs** with mandated perioperative geriatric input

D. Exam One-Liners

- **ICF 2001:** disability equals impairment plus activity limitation plus participation restriction.
- **Doctors treat impairment; therapists treat activity restriction; social workers treat participation restriction.**
- **Eight steps:** Assessment, Goals, Therapy, Aids and adaptations, Education, Psychological support, Evaluation, Follow-up.
- **Two cardinal principles: early intervention and the team approach.**
- **Set ambitious but achievable goals** — disagreement about goals wrecks programmes.
- **Barthel Index** is observation of what the patient *does*; **ceiling effect** in ambulatory patients.
- **FIM has 18 functions** across self-care, sphincter control, mobility, locomotion, communication and social cognition.
- **Participation restriction is subjective, mood-dependent and therefore largely unmeasured.**
- **An aid can foster dependence** — but those in real need often go without.
- **Prosthesis replaces a body part; orthosis supports an existing one.**
- **Stroke units improve survival and function; ortho-geriatric unit evidence is weaker.**
- **Cardiac rehabilitation reduces all-cause mortality by 27%**; sit out of bed within 24 hours and walk to the toilet within 48 hours after uncomplicated myocardial infarction.
- **Pulmonary rehabilitation: at least 3 times a week, at least 20 minutes, at least two-thirds maximum heart rate, at least 6 weeks (preferably 3 months)** — greatest benefit in the most impaired.
- **An above-knee prosthesis costs 40% more energy than below-knee** — preserve the knee.
- **Discharge is a transition, not the end, of rehabilitation.**

11. Immobility in the Elderly — Causes, Complications and Management

A. 20 High-Yield Points

Definition and Significance

1. **Definition:** Immobility is the state in which an individual has a **limitation in independent, purposeful physical movement** of the body or of one or more lower extremities. A complementary definition is the **restriction of time spent out of bed or chair** by **medical orders, restrictive devices, chemical restraints (for example antipsychotics), lack of mobility aids, or lack of human assistance or encouragement**.
2. **Why it matters:** Immobility triggers a **cascade of further disease, pain, disability and impaired quality of life**. Small improvements in mobility reduce complications, improve wellbeing and **cut the cost and burden of caregiving**. Immobility and sedentary time are independently associated with **all-cause mortality**.
3. **Scale of the problem: One-third of older patients are restricted to bed or chair rest during hospitalisation. 15–76%** of hospitalised elders have diminished performance in at least one ADL at discharge; of these, **only half recover function at three months**, and for many the decline means **permanent loss of independent living**. **Functional decline is a profound marker of morbidity and mortality**, causing longer stays, higher costs and increased nursing-home placement.
4. **"Spiralling immobility" (Tinetti and Ginter)** — a **catch-22**: an older person perceived to be at risk of falling is restrained to prevent falls, and then **cannot ambulate again safely because of the immobilising consequences of the restraint itself**.

Causes

5. **Musculoskeletal: arthritides (degenerative joint disease is the commonest)**, osteoporosis, **fractures (especially hip and femur)**, podiatric problems, Paget's disease. Degenerative joint disease pain and joint changes cause contractures and progressive immobility if untreated.
6. **Neurological: stroke**, **Parkinson's disease**, neuropathies, **normal-pressure hydrocephalus**, dementias, cerebellar dysfunction.
7. **Cardiovascular: severe congestive heart failure**, coronary artery disease with frequent angina, **peripheral vascular disease with claudication** — all limit activity through **lack of cardiovascular endurance**. In older diabetics, peripheral vascular disease causes claudication, neuropathy and altered balance together.
8. **Pulmonary:** severe **chronic obstructive lung disease**.
9. **Sensory: impaired vision**, **decreased kinaesthetic sense**, **decreased peripheral sensation**.
10. **Environmental: forced immobility in hospitals and nursing homes**, **inadequate mobility aids**, cluttered environments, uneven surfaces, unsuitable chair or bed height, **stairs**, absent handrails and grab bars.
11. **Psychological: depression**, **fear of falling**, apathy, **lack of motivation**, and **health beliefs** — particularly the belief that **rest and immobility aid recovery**.
12. **Iatrogenic and caregiving: well-meaning caregivers who do things *for* the patient rather than optimise their function** — inappropriate wheelchair use, bathing and dressing people who could do it themselves — directly produce deconditioning.
13. **Drugs: sedatives and hypnotics** (drowsiness, dizziness, delirium, ataxia); **typical antipsychotics** (extrapyramidal rigidity); **antihypertensives** (orthostatic hypotension, bradycardia, dizziness).
14. **Other: deconditioning after prolonged bed rest**, malnutrition, **severe systemic illness such as widespread malignancy**, and acute and chronic pain.

Complications — System-Wise

15. **The complete complication table:**

System	Complications
Skin	**Pressure ulcers**
Musculoskeletal	Muscular **deconditioning and atrophy** · reduced fibre size, diameter and capillarity · **contractures** · **disuse osteoporosis** · degenerative joint disease · balance and coordination problems
Cardiovascular	**Deconditioning** · **orthostatic hypotension** · **deep vein thrombosis and pulmonary embolism**
Pulmonary	Decreased tidal volume and ventilation · **atelectasis** · **aspiration pneumonia** (supine position)
Gastrointestinal	Anorexia · **constipation** · **faecal impaction and incontinence**
Genitourinary	**Urinary infection** · **retention** (cannot void lying down; impaction obstructs flow) · **bladder calculi** · incontinence
Metabolic	**Negative nitrogen and calcium balance** · **decreased plasma volume** · **impaired glucose tolerance** · **altered drug pharmacokinetics** · hypercalcaemia
Psychological	**Sensory deprivation** · **delirium** · **depression** · anxiety

16. **Key mechanistic points:** - **Muscle atrophy severity is proportional to the duration and magnitude of activity limitation**; unchecked it causes **permanent muscle damage**. - Immobility causes a **rapid and sustained increase in bone resorption with decreased bone formation**; reduced bone mass then makes any fall more likely to fracture. - **Deconditioned cardiovascular reflexes plus reduced plasma volume produce postural hypotension**, which impairs rehabilitation and predisposes to **falls, stroke and myocardial infarction**. - **Contracture** is fixation of muscle *and* joint in flexion from structural shortening of muscles adjacent to a major joint, leading to **permanent loss of range of motion**; highly prevalent in nursing homes. - **"The bed is a very dangerous place for an older person."**

Assessment

17. **History:** nature and duration of the disability · contributing medical conditions · **pain** · **drugs affecting mobility** · **motivation and psychological factors** · **environment, physical and social, especially caregiving interactions** · the patient's and carer's **perceived** cause of immobility. **Nutrition:** check **protein status and 25-hydroxy vitamin D (ng/mL)**, both linked to muscle weakness, poor performance, balance problems and falls.
18. **Examination: skin** (bony prominences, areas pressed by bed, chair, splint, shoe or device) · **cardiopulmonary status including postural blood pressure and pulse** · **musculoskeletal** (muscle tone and strength, joint range of motion, foot deformities and lesions) · **neurological** (focal weakness, sensory and perceptual deficits, cognition). **Levels of mobility — assess and re-assess serially: bed mobility → transfers (bed to chair, toilet) → wheelchair mobility → standing balance → gait → stair climbing**, noting **pain with movement, fear, resistance and endurance**. Use **standardised repeated muscle-strength grading (MRC 0–5)** to gauge progress.

Management

19. **General principles:** - **Treat the underlying cause** and **relieve pain** — pain and fear of falling must be addressed *before* engaging the patient in exercise. - **Careful assessment of function and underlying capability**; **set realistic goals**. - **Prevent secondary disabilities and complications of immobility.** - **Evaluate the environment; adapt the environment to the patient and the patient to the environment.** - **Ongoing motivation of both the patient and the caregivers is critical.** - **Restorative philosophy of care** — maintain the highest level of function and increase time spent in physical activity; a **core element of geriatric practice**.
20. **Team-based management:**

Discipline	Role
Physiotherapy	**Objectives:** relieve pain · evaluate, maintain and improve **joint range of motion** · improve **strength, endurance, motor skills and coordination** · improve **gait and stability** · assess need for and teach use of **assistive devices**. **Modalities: active (isometric, isotonic) and passive exercise**, **sitting exercise programmes**, heat (hot packs, paraffin, diathermy, ultrasound), **hydrotherapy**, **TENS**
Occupational therapy	Focus on **functional abilities, especially ADLs and IADLs**; detailed mobility assessment; **aids, adaptations and home modification**
Physician	Treat underlying disease; **deprescribe sedatives, typical antipsychotics and offending antihypertensives**; correct anaemia, thyroid disease, B12 and vitamin D deficiency; **analgesia**
Nursing	Two-hourly turning · skin care · **early mobilisation, sitting out for meals, own clothes** · continence and bowel care · **avoid restraints and catheters**
Dietician	Protein and calorie adequacy; **vitamin D and calcium (mg/dL)**

System-specific prevention: - **Skin — prevention is the cornerstone.** Three contributory factors: **pressure, shearing forces and friction**. Shearing occurs when the **head of the bed is elevated and the torso slides down**, transmitting pressure to the sacrum. **More than 90% of pressure ulcers occur in the lower body** — sacral and coccygeal, ischial tuberosities and greater trochanter. Risk scored by **Braden or Norton scale**. **The area of damage below the ulcer is often much larger than the ulcer itself.** Management equals pressure relief (**alternating pressure mattresses and overlays are equally effective**), regular cleansing and drying, nutrition and hydration. **The most important factor is simply the attention the skin receives.** - **Chest:** upright positioning, breathing exercises, incentive spirometry, aspiration precautions. - **Venous thromboembolism:** early mobilisation, graduated compression stockings, thromboprophylaxis. - **Bowel and bladder:** timed toileting, avoid indwelling catheters, treat constipation and impaction. -

Metabolic and musculoskeletal: daily passive and active range-of-motion exercises to prevent contractures, weight-bearing where possible, adequate protein and vitamin D. - **Psychological:** treat depression, provide sensory stimulation and orientation, prevent delirium. - **Avoid restraints:** physical restraints and **full-enclosure siderails** are associated with numerous negative outcomes including **strangulation and asphyxiation**, and **research demonstrates they can be safely eliminated**. Success requires **long-term, organisation-level education plus clear policy backed by regulation** — short educational programmes alone do not work.

B. Latest Updates (2023–Present)

1. "Hospital-associated deconditioning (HAD)" is now a named, defined syndrome. A **2025 BMJ Open scoping review** set out to establish **agreed diagnostic criteria for hospital-acquired deconditioning** and to distinguish it from other immobility-linked syndromes — the central limitation of previous reviews being the absence of any consensus definition. Current working figures: **muscle deconditioning begins within two days**, with a **2–5% decline in muscle mass per day** of leg inactivity and **up to 20% loss of baseline strength in one week**.

2. "End PJ Paralysis" — the strongest recent implementation evidence. The Alberta multicomponent inpatient ambulation initiative, analysed across **32,884 discharge abstracts** using interrupted time series (2024), showed **length of stay reduced by 1.8 days**, **inpatient falls reduced by 2.2 events**, and an increasing percentage discharged home. The interventions **safely mitigated immobility-induced complications including deconditioning and hospital-acquired disability**.

3. BGS "Sit up, get dressed, keep moving!" remains the endorsed UK campaign, now paired with the **Royal College of Physicians' 2024 National Audit of Inpatient Falls report, "Don't stop moving: optimising safety while staying active in hospital"** — explicitly rejecting immobilisation as a falls-prevention strategy. **Fear of falls must not be a barrier to mobilisation**; patients must be supported to maintain independence **from the moment of arrival**.

4. Cochrane 2024 updated the evidence on **falls prevention interventions in older people**, relevant because immobilisation is still wrongly used as a falls-prevention measure.

5. International Pressure Injury Guideline — 4th edition (2025). NPIAP, EPUAP and PPPIA, with **more than 240 clinical experts and consumer representatives** and GRADE evidence-to-decision methodology. Released in **staged online chapters** — prevention from February 2025, treatment after September 2025 — with refreshed quick-reference guides, superseding the 2019 edition.

6. Pressure injury — points that have changed: - Terminology: **"pressure injury"** replaces "pressure ulcer or sore"; **reverse staging is not valid**. - **Adjunctive therapies (negative pressure wound therapy, hyperbaric oxygen, laser, electrotherapy, therapeutic ultrasound) have no good evidence of improving healing.** - **Diagnosing infection:** classic signs (purulent exudate, erythema, heat, oedema) and foul odour are **not helpful**; **increasing pain is the most useful sign (likelihood ratio 11–20)**. Confirm with a **quantitative swab using the Levine technique** — rotate the swab over 1 x 1 cm for 5 seconds with enough pressure to express fluid. - **"Skin failure"** is now recognised — in overall patient decline with irreversible conditions, wounds on bony prominences are **not preventable pressure injuries**, and care should prioritise **infection risk reduction and pain control**.

7. Age-Friendly Health Systems 4Ms. Mobility is one of the four Ms. In a 2024 eight-hospital evaluation, **Mobility generated the highest "act-on" rate of all four Ms (83%)**, and patients receiving all 4Ms had **fewer 30-day emergency visits (16% vs 22%)**.

C. Current Practice Update

Aspect	Textbook position	Current position (2023–2026)
Concept	"Immobility" as a geriatric syndrome or presenting problem	Reframed as **hospital-associated deconditioning / hospital-acquired disability** — a *preventable adverse event*, with a 2025 push for formal diagnostic criteria
Speed of onset	"Deconditioning can occur rapidly"	**Quantified: begins within 2 days; 2–5% muscle mass loss per day; up to 20% strength loss in 1 week**
Falls versus mobility	Restraints and bed rest historically used for "safety"	**RCP 2024: "Don't stop moving"** — immobilisation is *not* falls prevention
Intervention model	Individual physiotherapy or occupational therapy referral	**Whole-system campaigns** — End PJ Paralysis, Sit Up Get Dressed Keep Moving, 4Ms Mobility, with measurable outcomes
Pressure ulcers	2019 international guideline; Braden and Norton; stages I–IV	**4th edition International Guideline (2025)**; "pressure injury"; adjunctive therapies unsupported; **increasing pain is the best sign of infection**; concept of **"skin failure"**
Restraints	Described as declining but persistent	Evidence confirms **safe elimination**; requires **long-term organisational education plus policy plus regulation**

D. Exam One-Liners

- **"The bed is a very dangerous place for an older person."**
- **One-third of hospitalised older patients are on bed or chair rest**; 15–76% lose an ADL by discharge and **only half**

recover by 3 months.

- **Deconditioning starts within 48 hours** — 2–5% muscle loss per day, about 20% strength loss per week.
- **Spiralling immobility** — restrained to prevent falls, then cannot walk, then more falls.
- **Commonest musculoskeletal cause is degenerative joint disease**; commonest neurological cause is stroke.
- **Three drug classes that immobilise:** sedative-hypnotics, typical antipsychotics, antihypertensives.
- **Complications by system:** skin (pressure injury) · musculoskeletal (atrophy, contractures, disuse osteoporosis) · cardiovascular (orthostasis, DVT and PE) · respiratory (atelectasis, aspiration pneumonia) · gastrointestinal (constipation, impaction) · genitourinary (UTI, retention, calculi) · metabolic (negative nitrogen and calcium balance, impaired glucose tolerance) · psychological (delirium, depression, sensory deprivation).
- **Contracture equals muscle plus joint fixation in flexion** and permanent loss of range of motion.
- **Pressure injury triad: pressure plus shear plus friction**; **more than 90% occur in the lower body**; risk scored by **Braden or Norton**.
- **Alternating pressure mattresses and overlays are equally effective** — what matters most is the attention the skin receives.
- **Increasing pain, not pus or erythema, is the best sign of chronic wound infection.**
- **Assess mobility in a ladder:** bed mobility, transfers, wheelchair, standing balance, gait, stairs.
- **Restraints can be safely eliminated** — but only with sustained organisational education and policy.

Part IV — Cognition, Mood and Decisional Capacity

12. Tests of Cognition and Rating Scales

A. 20 High-Yield Points

Principles

1. **Purpose of cognitive testing:** to establish **strengths and weaknesses of various functions relative to a standardised, norm-referenced scoring system**, so that the **nature and extent of cognitive deficits** can be determined, informing diagnosis and management and acting as a **comparator for past and future assessments**.
2. **A cognitive screen is not a diagnosis.** The MMSE and similar tools give an overall assessment of cognitive function, but **their use alone is not sufficient to establish a diagnosis of Alzheimer's disease or any dementia**.
3. **Cognitive screening is one of only two mandatory screens** (with depression) in any older person with functional disability.
4. **Key aspects of the mental status examination:** state of consciousness · general appearance and behaviour · orientation · **memory (short and long term)** · language · visuospatial function · **executive control functions** · other cognitive functions (calculations, proverb interpretation) · **insight and judgement** · thought content · mood and affect.
5. **Test all three components of memory: immediate recall** (repeating digits), **recent memory** (recalling three objects after a few minutes), and **remote memory** (details of early life).
6. **Executive function** involves the **planning, sequencing and execution of goal-directed activities**, and is **critical to the ability to perform IADLs**. Screen with the **clock-drawing test** (a component of the Mini-Cog) or **verbal fluency** (listing 12 animal names in one minute is normal).
7. **Interpretation depends on education and age.** The MMSE score is **dependent on sociocultural level and subject to a ceiling effect** — a crucial caveat in the Indian population, where illiteracy is common.
8. **Assess premorbid intellectual status** where possible (for example the National Adult Reading Test) to interpret other test scores correctly.
9. **Adapt tests to deficits.** In language impairment or dyspraxia, **recognition memory tests reduce the demand for expressive language** required by recall tests.
10. **Always include a standardised measure of mood** — depression is the commonest mimic of cognitive impairment.

Screening and Assessment Tools

11. **Recommended tools in primary care (Table 72.2):** - **Delirium: Confusion Assessment Method (CAM)** - **Dementia screening: AD8**, **Mini-Cog** - **Dementia assessment: SLUMS**, **MMSE**, **MoCA**
12. **Mini-Cog** screens **both memory (three-item recall) and executive function (clock drawing)** — quick, and relatively education-independent.
13. **MoCA and Addenbrooke's Cognitive Examination-Revised (ACE-R)** are described as **well-validated screening options**; ACE-R **incorporates the MMSE and clock-face drawing**.
14. **MMSE severity bands in Alzheimer's disease: mild 20–24, moderate 10–19, severe below 9.**
15. **Global staging scales combining cognition and ADLs: Global Deterioration Scale (GDS, Reisberg)** and the **Clinical Dementia Rating (CDR) scale**.
16. **Severe Impairment Battery (SIB)** was developed for subjects **too cognitively impaired for standard testing**.
17. **Memory-specific screens:** the **five-word test** and the **Memory Impairment Screen (MIS)** assess **anterograde verbal memory** at an initial consultation. For detailed evaluation, **free and cued recall** paradigms — the **Grober and Buschke test (Free and Cued Selective Reminding Test)** and the **California Verbal Learning Test** — distinguish mild Alzheimer's disease from healthy subjects with **more than 90% precision**, even when MMSE scores are similar.
18. **Domain-specific battery (Table 62.4 — Cardiff memory clinic):**

Domain	Tests
Premorbid ability	**National Adult Reading Test (NART)**
Cognitive screen / battery	**MMSE, MoCA, RBANS, Frontal Assessment Battery (FAB)**
Attention and concentration	Digits forward, digits backward, digit span
Language	**Boston Naming Test**, Irving Names Learning Test
Memory	Story recall (immediate and delayed), Kendrick tests, **California Verbal Learning Test**
Executive function	**Trail-Making Test A and B**, clock drawing
Visuospatial function	**Rey–Osterrieth Complex Figure**, clock drawing
Mood	**GDS-15**

Delirium Instruments

19. **Confusion Assessment Method (CAM)** — the standard; **sensitivity 94%, specificity 89%** with high inter-rater reliability. **Inattention** is tested at the bedside by **months of the year backwards, days of the week backwards, or**

digit span 3 or 4 backwards. - **3D-CAM:** a structured **three-minute** screen — **sensitivity 96%, specificity 86% in patients with dementia**; **93% and 96% in those without**. - **Best single item: "months of the year backwards"** (sensitivity 83%, specificity 69%). Best two items: months backwards plus "what is the day of the week?" (sensitivity 93%, specificity 64%). - **CAM-ICU** for intubated patients — inattention tested by squeezing on the letter A in "SAVEAHAART"; level of consciousness by **RASS score other than 0**.

Practical Conduct of Testing

20. Tests must be **acceptable to the person and contain nothing belittling**. **Consent must be obtained**, the purpose explained, and **sensory and physical limitations accommodated** — adequate lighting, earphone amplifiers, suitable seating, minimal distractions. Provide **realistic reassurance with positively phrased feedback that highlights strengths as well as weaknesses**, and at the end **allow patients to comment on their own performance**. Ideally an experienced psychologist or neuropsychologist performs formal testing, though **much useful information can be obtained by any suitably trained professional**. Introduce questions **non-threateningly**, because many patients with early deficits respond defensively.

B. Comparison of the Main Cognitive Tools

Tool	Items / time	Domains	Main strength	Main limitation
AMT (Hodkinson)	10 questions, 2 min	Orientation, memory	Very quick; RCP/BGS endorsed	Crude; misses executive and visuospatial deficits
Mini-Cog	3 min	3-word recall + clock drawing	**Relatively education-independent**; screens executive function	Screen only
AD8	8 items, informant	Change over time	Informant-based, detects change	Needs a reliable informant
MMSE	30 points, 10 min	Orientation, registration, attention, recall, language, construction	Most widely known; severity bands defined	**Education- and culture-dependent; ceiling effect; poor for executive function**; copyrighted
MoCA	30 points, 10–15 min	Adds executive, abstraction, visuospatial, attention	**Superior for mild cognitive impairment and executive dysfunction**	Lower specificity; education adjustment (+1 if 12 years or less)
SLUMS	30 points, 10 min	Adds executive and clock tasks	Free to use; education-stratified cut-offs	Less international validation
ACE-R / ACE-III	100 points, 20 min	Five domains; incorporates MMSE	Differentiates **Alzheimer's from frontotemporal dementia**	Long
RBANS	30 min	Repeatable battery	Serial testing without practice effect	Requires training
Frontal Assessment Battery	6 items	Frontal/executive	Detects subcortical and frontal deficits	Narrow
CAM / 3D-CAM / CAM-ICU	3–5 min	Delirium	**Gold standard for delirium**	Needs training; misses hypoactive cases if hurried
GDS (Reisberg) / CDR	Interview	Global staging	Combines cognition and function	Time-consuming
SIB	40 min	Severe dementia	Usable when MMSE floor is reached	Specialist tool

C. Latest Updates (2023–Present)

1. 2024 NIA-AA Revised Criteria — Alzheimer's disease is now defined biologically. Diagnosis rests on **Core 1 biomarkers** — a positive **plasma p-tau217**, a positive **amyloid PET**, or abnormal **CSF ratios (p-tau181/Aβ42, t-tau/Aβ42, Aβ42/40)**. Cognitive testing now defines the **clinical stage**, not the disease itself. This is the single biggest conceptual change in the field.

2. Blood-based biomarkers have entered clinical use. Diagnostic models combining **plasma p-tau217 with age and ApoE genotype** achieve **AUC around 0.94** for Alzheimer's disease. In very elderly nursing-home and hospital populations, **plasma p-tau181, p-tau217 and GFAP differ significantly between cognitively normal and probable Alzheimer's groups**, while **Aβ42, Aβ40 and the Aβ42/40 ratio do not** — an important practical point, since amyloid ratios perform poorly in the oldest old.

3. Cognitive screens are being repositioned as triage. Rather than diagnosing, MMSE and MoCA increasingly serve to **decide who proceeds to biomarker testing**. Clinical trials now use combinations such as **CDR global score 0.5–1.0 plus MMSE 21 or above plus a positive Core 1 biomarker** for entry.

4. Mild behavioural impairment (MBI) — late-onset persistent neuropsychiatric symptoms — has emerged as an **early**

indicator of dementia risk that may precede measurable cognitive decline, and is being studied alongside plasma p-tau217.

5. Digital and mobile cognitive assessment. Mobile cognitive assessment systems have demonstrated **diagnostic equivalence to MMSE and MoCA** in Alzheimer's screening; future direction is **composite risk scores combining digital tests, wearables and plasma p-tau217**. Regulatory and ethical frameworks for data security, algorithmic transparency and equitable access are still being established.

6. Standardised inclusion in assessment frameworks. Cognition/delirium is one of the **six mandatory core dimensions** of the German S3 CGA guideline (2024); **cognition** is one of the **six WHO ICOPE domains**, with **Mini-Cog, MoCA, MMSE or GPCOG** named as step-2 assessment tools; **Mentation** is one of the **4Ms** of Age-Friendly Health Systems.

7. Indian context. In LASI wave-1, **impaired cognition was present in 9.3%** of adults aged 60 and above. Culture- and education-fair instruments are essential: **Hindi Mental State Examination (HMSE)**, **Vernacular MoCA versions**, **Rowland Universal Dementia Assessment Scale (RUDAS)**, **Indian Council of Medical Research Neuro-Cognitive Tool Box**, and **AIIMS Comprehensive Neuropsychological Battery in Hindi** are used in place of a directly translated MMSE, because **the standard MMSE penalises illiteracy heavily**.

D. Current Practice Update

Aspect	Pathy's 6th ed. (2022)	Current position (2023–2026)
Basis of Alzheimer's diagnosis	Clinical, supported by cognitive tests and imaging	**Biological — 2024 NIA-AA criteria** based on Core 1 biomarkers
Role of MMSE/MoCA	Screening and severity grading	**Staging and triage** to biomarker testing; no longer diagnostic on their own
Biomarkers	CSF amyloid and tau "in some centres"	**Plasma p-tau217 in routine diagnostic pathways**; AUC around 0.94 with age and ApoE
Amyloid ratios	Presented as useful	**Aβ42/40 performs poorly in the oldest old**; p-tau217 and GFAP superior
Delivery	Face-to-face testing by trained staff	**Mobile and computerised assessment shown equivalent to MMSE/MoCA**
Preclinical detection	Subjective cognitive decline and MCI	Adds **mild behavioural impairment (MBI)** as an early neurobehavioural marker
Indian practice	MMSE with education caveat	**Education-fair tools mandatory** — HMSE, RUDAS, vernacular MoCA, ICMR NCTB

E. Exam One-Liners

- **A cognitive screen is not a diagnosis** — MMSE alone cannot diagnose Alzheimer's disease.
- **MMSE bands:** mild 20–24, moderate 10–19, severe below 9.
- **MMSE is education- and culture-dependent with a ceiling effect** — the key limitation in India.
- **MoCA beats MMSE for mild cognitive impairment and executive dysfunction.**
- **Mini-Cog = 3-word recall plus clock drawing** — screens memory and executive function in three minutes.
- **Clock drawing and verbal fluency screen executive function**, which drives **IADL** performance.
- **CAM sensitivity 94%, specificity 89%; 3D-CAM takes three minutes.**
- **Best single delirium item: months of the year backwards.**
- **ACE-R incorporates the MMSE and clock drawing** and helps separate Alzheimer's from frontotemporal dementia.
- **Free and cued recall (Grober–Buschke) separates mild Alzheimer's from normal with over 90% precision** even at similar MMSE scores.
- **Global staging: Reisberg GDS and CDR** combine cognition with function.
- **SIB is for patients too impaired for standard testing.**
- **2024 NIA-AA: Alzheimer's disease is now a biological diagnosis** — plasma **p-tau217**, amyloid PET, or CSF ratios.
- **Always pair cognitive testing with a mood scale**, and obtain consent and premorbid ability first.

13. Cognitive Assessment Tools

A. 20 High-Yield Points

Classification — the Framework to Open With

1. Cognitive assessment tools fall into **four functional groups**, and stating this classification first earns marks: - **Delirium instruments** — CAM, 3D-CAM, CAM-ICU, 4AT - **Brief screening tools** — Mini-Cog, AD8, AMT, GPCOG, MIS, six-item screener - **Cognitive screening batteries** — MMSE, MoCA, SLUMS, ACE-III, RUDAS - **Detailed neuropsychological batteries and staging scales** — RBANS, CVLT, Trail-Making, FAB, Boston Naming, Rey–Osterrieth; CDR and Reisberg GDS for staging
2. **A second axis is the informant dimension** — **performance-based** (patient does the task: MMSE, MoCA) versus **informant-based** (a relative reports change: **AD8, IQCODE**). Informant tools detect **change from a personal baseline** and are invaluable when education confounds performance tests.
3. **The tool must match the question:** *Is this delirium?* → CAM. *Is there any cognitive problem?* → Mini-Cog or AD8. *How severe and in which domain?* → MoCA, MMSE, SLUMS. *Which dementia is it?* → ACE-III with a full battery. *How advanced is it?* → CDR or Reisberg GDS.

Delirium Tools

4. **CAM** — sensitivity **94%**, specificity **89%**, high inter-rater reliability. Four features: **acute onset and fluctuating course, inattention, disorganised thinking, altered level of consciousness** (features 1 and 2 plus either 3 or 4).
5. **Inattention testing at the bedside: months of the year backwards, days of the week backwards, digit span 3 or 4 backwards**; or the examiner may simply observe **inappropriate distraction or loss of the thread of conversation**.
6. **3D-CAM** — three-minute structured version: **sensitivity 96%, specificity 86% in patients with dementia**; **93% and 96% without dementia**. **CAM-ICU** for intubated patients uses the "SAVEAHAART" letter-A task and the **RASS**.

Brief Screens

7. **Mini-Cog — three-item recall plus clock drawing**; screens **memory and executive function** in about three minutes and is **relatively education-independent**, making it the most practical screen in Indian outpatient settings.
8. **AD8** — an **eight-item informant questionnaire** detecting change over time; useful when the patient's education level makes performance testing unreliable.
9. **Abbreviated Mental Test (Hodkinson, 10 items)** — the **RCP/BGS-endorsed** memory and cognition screen; extremely fast but crude.
10. **Memory Impairment Screen and the five-word test** assess **anterograde verbal memory** specifically at a first consultation.

Screening Batteries

11. **MMSE (Folstein, 1975)** — 30 points across orientation, registration, attention/calculation, recall, language and construction. **Interpretation is complex because thresholds depend on education and age**; it has a **ceiling effect** and **tests executive function poorly**. Severity bands: **mild 20–24, moderate 10–19, severe below 9**.
12. **MoCA (Nasreddine, 2005)** — 30 points; adds **trail-making, cube copy, clock drawing, verbal fluency, abstraction and delayed recall**, so it detects **mild cognitive impairment and executive/subcortical dysfunction far better than the MMSE**. **Add 1 point if formal education is 12 years or less.**
13. **SLUMS (Saint Louis University Mental Status)** — 30 points; free of charge, with **separate cut-offs for those with and without a high-school education**.
14. **ACE-R / ACE-III** — 100 points across **attention/orientation, memory, verbal fluency, language and visuospatial function**; ACE-R **incorporates the MMSE and clock-face drawing**, and the **VLOM ratio helps separate Alzheimer's disease from frontotemporal dementia**.
15. **RUDAS (Rowland Universal Dementia Assessment Scale)** — designed to be **culturally and linguistically fair**, needing no literacy; highly relevant to multilingual, variably literate Indian populations.

Detailed and Domain-Specific Tools

16. **Domain mapping (Table 62.4):** premorbid ability — **NART**; screening battery — **MMSE, MoCA, RBANS, FAB**; attention — **digit span forward and backward**; language — **Boston Naming Test**; memory — **story recall, California Verbal Learning Test**; executive — **Trail-Making A and B, clock drawing**; visuospatial — **Rey–Osterrieth Complex Figure**; mood — **GDS-15**.
17. **Free and cued recall tests** — the **Grober and Buschke test** and the **CVLT** — separate mild Alzheimer's disease from normal ageing with **more than 90% precision**, even when MMSE scores are identical, because they demonstrate a **hippocampal amnestic pattern (cueing does not improve recall)**.
18. **Staging scales: Clinical Dementia Rating (CDR)** and the **Reisberg Global Deterioration Scale** combine cognition with ADL impact. The **Severe Impairment Battery** is used when impairment is too severe for standard testing.

Rules of Use

19. **Practical conduct:** obtain **consent**, explain the purpose, ensure **adequate lighting, hearing amplification, suitable seating and no distractions**, introduce questions **non-threateningly**, phrase feedback **positively**, and give the patient a chance to comment on their own performance at the end. Assess **premorbid intellectual status** first, and **always add a mood scale**.
20. **Two cardinal caveats:** - **Depression and delirium must be excluded before any score is attributed to dementia** — screening tools become unreliable in delirium, and depression is the commonest reversible mimic. - **No cognitive screening test is appropriate for determining decision-making capacity.** Screens of **executive function** such as the **clock-drawing test** or the **Executive Interview** correlate better with decisional capacity than the MMSE, but capacity requires its own task-specific assessment.

B. Comparison Table — Choosing the Right Tool

Clinical question	Tool of choice	Time	Notes
Is this delirium?	**CAM / 3D-CAM / CAM-ICU**	3–5 min	Sensitivity 94%, specificity 89%
Rapid screen in a busy clinic	**Mini-Cog**	3 min	Education-independent; memory + executive
Screen when the patient cannot be tested	**AD8 or IQCODE** (informant)	3 min	Detects change from baseline
General cognitive assessment	**MoCA**	10–15 min	Best for MCI; add 1 point if education 12 years or less
Severity grading in known dementia	**MMSE**	10 min	Mild 20–24, moderate 10–19, severe below 9
Free alternative with education cut-offs	**SLUMS**	10 min	Education-stratified
Differentiating dementia subtypes	**ACE-III**	20 min	VLOM ratio; Alzheimer's vs frontotemporal
Illiterate or multilingual patient	**RUDAS, HMSE, vernacular MoCA**	10 min	Culture- and literacy-fair
Detecting early Alzheimer's memory pattern	**Grober–Buschke, CVLT**	20–30 min	Cueing fails to improve recall
Frontal or subcortical deficit	**FAB, Trail-Making B, verbal fluency**	5–10 min	Subcortical screen
Global staging	**CDR, Reisberg GDS**	Interview	Combine cognition and function
Severe dementia	**SIB**	40 min	Below the MMSE floor

C. Latest Updates (2023–Present)

1. 2024 NIA-AA Revised Criteria define Alzheimer's disease **biologically** through **Core 1 biomarkers — plasma p-tau217, amyloid PET, or CSF p-tau181/Aβ42, t-tau/Aβ42 or Aβ42/40**. Cognitive tools now **stage the disease rather than diagnose it**.

2. Plasma p-tau217 is the practice-changing test. Models combining **plasma p-tau217, age and ApoE genotype** achieve **AUC about 0.94**. In the very elderly, **p-tau181, p-tau217 and GFAP discriminate probable Alzheimer's from normal cognition, while Aβ42, Aβ40 and the Aβ42/40 ratio do not** — so amyloid ratios should not be relied on in the oldest old.

3. Digital cognitive assessment. Mobile cognitive assessment systems show **diagnostic equivalence to MMSE and MoCA** in Alzheimer's screening. Future work is on **composite scores integrating digital tests, wearables (for example sleep fragmentation) and plasma p-tau217**, with cross-ethnic validation and regulatory frameworks still pending.

4. Mild behavioural impairment (MBI) — late-onset persistent neuropsychiatric symptoms — is now recognised as an early dementia-risk indicator that may **precede measurable cognitive decline**, and is being validated against plasma p-tau217.

5. Mandated inclusion in assessment frameworks. German S3 CGA guideline (2024): cognition and delirium is one of six core dimensions. **WHO ICOPE:** cognition is one of six intrinsic-capacity domains, with **Mini-Cog, MoCA, MMSE and GPCOG** as named step-2 tools. **Age-Friendly 4Ms:** cognition sits under **Mentation**. **ASCO 2023 Practical Geriatric Assessment** includes a cognitive domain for every cancer patient aged 65 and above.

6. Indian tools and data. LASI wave-1 found **impaired cognition in 9.3%** of those aged 60 and above. Because the MMSE penalises illiteracy heavily, Indian practice uses the **Hindi Mental State Examination (HMSE)**, **RUDAS**, **vernacular MoCA versions**, the **ICMR Neuro-Cognitive Tool Box**, and the **AIIMS Comprehensive Neuropsychological Battery in Hindi**.

D. Current Practice Update

Aspect	Pathy's 6th ed. (2022)	Current position (2023–2026)
What the tools do	Screen for and grade cognitive impairment	**Stage the disease** and **triage to biomarker testing**
Diagnosis of Alzheimer's	Clinical, supported by imaging and sometimes CSF	**Biological (2024 NIA-AA)** — Core 1 biomarkers
Blood tests	Not diagnostically useful	**Plasma p-tau217 in routine pathways**; GFAP adds value
Amyloid measures	CSF amyloid used in some centres	**Aβ42/40 unreliable in the oldest old**
Mode of testing	Paper-and-pencil, face to face	**Mobile and computerised testing equivalent to MMSE/MoCA**
Preclinical markers	Subjective cognitive decline, MCI	Adds **mild behavioural impairment**
Cross-cultural use	MMSE with an education caveat	**Literacy-fair instruments mandatory** in India — RUDAS, HMSE, vernacular MoCA

E. Exam One-Liners

- **Four groups of tools:** delirium instruments, brief screens, screening batteries, detailed batteries and staging scales.
- **CAM for delirium; Mini-Cog or AD8 to screen; MoCA or MMSE to assess; ACE-III to subtype; CDR or Reisberg GDS to stage.**
- **Mini-Cog = 3-word recall plus clock drawing**, and is relatively education-independent.
- **AD8 and IQCODE are informant-based** — they measure change from the person's own baseline.
- **MoCA detects MCI and executive dysfunction better than MMSE; add 1 point if education is 12 years or less.**
- **MMSE has a ceiling effect, is education-dependent, and tests executive function poorly.**
- **RUDAS, HMSE and vernacular MoCA are the literacy-fair choices for Indian patients.**
- **Grober–Buschke free and cued recall demonstrates the hippocampal amnestic pattern of Alzheimer's disease.**
- **ACE-R incorporates the MMSE and clock drawing.**
- **Exclude delirium and depression before interpreting any cognitive score.**
- **No cognitive screening test determines decision-making capacity** — executive tests correlate better than the MMSE, but capacity needs its own assessment.
- **2024 NIA-AA: plasma p-tau217, amyloid PET or CSF ratios define the disease; cognition defines the stage.**

14. Depression Rating Scales in the Elderly

A. 20 High-Yield Points

Which Scale, and When

1. **A fundamental distinction must open the answer:** - **Screening scales** (detect possible cases) — **GDS, CES-D, PHQ-2 and PHQ-9** - **Severity and monitoring scales** (measure change once diagnosed) — **MADRS (preferred), HDRS** - **Special-population scale** (severe cognitive impairment) — **Cornell Scale for Depression in Dementia (CSDD)**
2. **Diagnosis is clinical.** Formal diagnostic interviews such as the **Composite International Diagnostic Interview (CIDI)**, aimed at establishing a DSM diagnosis, are **very time-consuming and require extensive training** — so the diagnosis is usually made clinically, with or without a screening instrument.
3. **Screening in primary care is critical** — not only because depression is frequent, but because **suicidal ideation can be detected by screening**.
4. **The key cognitive threshold:** in **severe cognitive impairment (MMSE below 18)**, the GDS and CES-D become **less reliable**, and the **Cornell Scale for Depression in Dementia is advised** instead.
5. **Monitoring rule:** once depression is diagnosed, severity **must be evaluated with a depression scale before starting treatment and at least every three months during treatment**, and **at least every three months for every older person started on any antidepressant treatment — drug or non-drug**.
6. **MADRS is the preferred severity scale** (Montgomery and Asberg, 1979) because it was **designed to be sensitive to change**.
7. **The Hamilton Depression Rating Scale (HDRS, 1960) is widely used but has a specific geriatric flaw:** it contains **relatively many somatic items**, so **somatic comorbidity can cause over-reporting** of depression severity.
8. **This flaw has distorted the literature.** The frequently repeated claim that late-life depression presents more somatically rests on a **meta-analysis based on studies using the HDRS** — so it is unclear how much of the "somatic presentation" is really an artefact of comorbid physical disease inflating somatic items. **This is a first-class critical-appraisal point.**
9. **Always accompany a depression scale with a cognitive scale** — objectify cognition with the **MMSE (Folstein) or MoCA (Nasreddine)** whenever depression is assessed in an older person.

Standardised Scales Recommended in Primary Care

10. **Depression:** - **Screening tool: PHQ-2** - **Assessment tools: PHQ-9**, **GDS-15 and GDS-30**, **SLU AMSAD** (Saint Louis University AM SAD), **CSDD**
11. **The rest of the recommended primary-care set, often asked as a whole:** - **Delirium:** Confusion Assessment Method (**CAM**) - **Dementia screening: AD8**, **Mini-Cog** · **Dementia assessment: SLUMS**, MMSE, MoCA - **Alcohol: CAGE** (screen), then **SMAST-G** (Short Michigan Alcohol Screening Test – Geriatric version) - **Agitation in dementia: Cohen-Mansfield Agitation Inventory – short form**

Where the Scales Sit in Geriatric Assessment

12. In the CGA framework, the **mental health and affective dimension** is measured by the **Geriatric Depression Scale (Yesavage)** and the **Zung Self-Rating Depression Scale**. Both are **in wide use**, and **shorter screening versions are available without significant loss of accuracy**.
13. **A depression screen is one of only two mandatory screens** in any older person with functional disability — the other being **cognitive screening**.
14. **RCP/BGS endorsed scales for rehabilitation:** depression is measured by the **Geriatric Depression Scale — 30 items, with a 15-item short form**. The **Philadelphia Geriatric Center Morale Scale** measures *subjective morale*, which is **distinct from depression though with some overlap**.
15. **Disease-specific scales:** - **Dementia → Cornell Scale for Depression in Dementia** (Alexopoulos, 1988) - **Stroke → Stroke Aphasic Depression Questionnaire, Signs of Depression Scale, Visual Analogue Mood Scale** - **Primary care → PRIME-MD** (5 questions mapping to DSM categories)
16. **The GDS family** is available as **30-item, 15-item, 5-item and 2-item** versions. **The 2-item version is used for preliminary screening, followed by a longer version if positive.**
17. **PHQ-2 detail:** asks how often over the **last 2 weeks** the person has had **(1) little interest or pleasure in doing things** and **(2) feeling down, depressed or hopeless**. Responses: not at all / several days / more than half the days / nearly every day. **Score 0–6; 3 or more indicates depression.**
18. **Before attributing symptoms to depression, exclude physical illness.** Because of symptom overlap and the close association of depression with medical conditions, older patients presenting with apparent depression need careful physical evaluation — this also **builds trust with the patient and family**.
19. **Baseline investigations in a depressed older patient: complete blood count, ESR, serum electrolytes (sodium**

mEq/L), glucose and calcium (mg/dL), renal and liver function, thyroid function, vitamin D, vitamin B12 or methylmalonic acid, folate, syphilis serology, urinalysis.

20. **Risk factors to elicit alongside the score:** female sex, previous depression, chronic physical illness and **ADL limitations**, cognitive disorders, personality problems, **loss of partner**, lack or loss of social contacts, **subjective loneliness**, recent life events, genetic factors, and **drugs commonly used by older people** that can cause mood disorder.

B. The Scales in Detail

Geriatric Depression Scale (GDS) — Yesavage, Brink et al., 1983

Feature	Detail
Design	Purpose-built for older adults
Format	Simple **yes/no** answers — deliberately chosen for ease of use, including in mild cognitive impairment
Key innovation	**Somatic items were deliberately excluded**, avoiding the false positives that physical illness produces on general scales
Versions	GDS-30 (original) · **GDS-15 (1986, the workhorse)** · GDS-10 · GDS-5 · GDS-4 · GDS-2
Scoring GDS-15	1 point per depressive answer; **0–4 normal, 5–9 mild, 10 or more moderate to severe**
Scoring GDS-30	**0–9 normal, 10–19 mild, 20–30 severe**
Translations	Over **30 languages**; validated Hindi, Tamil, Bengali, Malayalam and Kannada versions exist

Diagnostic accuracy (meta-analysis, 53 studies, 17,018 participants): - **GDS-30:** sensitivity **82%**, specificity **76%**, AUC **0.85** - **GDS-15:** sensitivity **86%**, specificity **79%**, **AUC 0.90 — the best of the family** - **GDS-10:** sensitivity 87%, specificity 75%, AUC 0.83 - **GDS-4:** sensitivity 74%, specificity 71%

The shorter GDS-15 and GDS-10 outperform the original GDS-30. An earlier primary-care meta-analysis confirmed the **GDS-15 correctly identifies more patients than the GDS-30 (77.6% versus 71.2%)** and rated the GDS-15 as **"good" for screening** but **"poor" for case-finding** — it rules out, it does not rule in.

Limitation: accuracy **falls in cognitive impairment** — specificity decreases below MMSE 28, and below MMSE 18 the scale should be abandoned for the CSDD.

Comparison of All the Major Scales

Scale	Items	Type	Best use in geriatrics	Key limitation
GDS-30 / GDS-15	30 / 15	Self-report, **yes/no**	**First-line screening** in any older person	Unreliable if **MMSE below 18**
GDS-5 / GDS-4 / GDS-2	5 / 4 / 2	Self-report	Ultra-rapid screening in busy or low-resource settings	Lower specificity
PHQ-2 then PHQ-9	2 then 9	Self-report	**Two-step primary-care pathway**; PHQ-9 also grades severity and asks about suicide	Contains **somatic items**
CES-D	20	Self-report	Epidemiological and population studies	Not geriatric-specific
Zung SDS	20	Self-report	Listed in the CGA dimension table	Dated; somatic items
CSDD	19	**Two-part interview — informant plus patient**, observation-based	**Depression in dementia** — the *only* scale validated when MMSE is below 18	Needs an informant; takes about 30 minutes
MADRS	10	Clinician-rated	**Severity monitoring — the preferred scale**; sensitive to change	Requires a trained rater
HDRS / HAM-D	17 / 21	Clinician-rated	Historic trial standard	**Too many somatic items — over-scores physically ill elders**
BASDEC	19 cards	Deck of cards	**Deaf or hard-of-hearing** inpatients	Not widely available
PGC Morale Scale	17	Self-report	**Morale, not depression** (RCP/BGS endorsed)	Different construct

MADRS 10 items: apparent sadness, reported sadness, inner tension, reduced sleep, reduced appetite, concentration difficulties, lassitude, inability to feel, pessimistic thoughts, suicidal thoughts. Each 0–6; **total 0–60**. Cut-offs: **0–6 normal, 7–19 mild, 20–34 moderate, 35 or more severe.** Its geriatric advantage is that **only 2 of 10 items are somatic** (sleep, appetite) — far fewer than the HDRS.

C. Latest Updates (2023–Present)

1. USPSTF 2023 — the headline guideline change. Clinicians should **screen all adults, including those 65 and older, for major depressive disorder (Grade B)**, and screen adults aged 19–64 for anxiety disorders. Critically, the USPSTF **concluded there was insufficient evidence to recommend for or against screening for anxiety disorders**

in adults 65 years or older, or for **suicide risk in any adult**. The tools specifically examined were the **PHQ (any version), CES-D, EPDS for perinatal persons, and the GDS for older adults**. This replaces the 2016 depression and 2014 suicide-risk statements.

2. The paradox to quote: depression screening in the elderly is now a **Grade B recommendation**, but **suicide-risk screening remains Grade I (insufficient evidence)** — despite older men having the highest suicide rates of any group. The PHQ-9 includes a question assessing suicidal thoughts, so many patients still receive de facto suicide-risk screening.

3. GDS-15 remains the geriatric standard, with an updated cut-off debate. At the recommended **cut-off of 5 or more**, pooled **sensitivity 0.89 and specificity 0.77**. An updated 2000–2019 review found pooled sensitivity **0.80**, specificity **0.79**, **AUC 0.89** — and flagged that **diagnostic accuracy is lower in older adults with cognitive impairment**. A head-to-head study against structured clinical interview found **AUC 0.97 for GDS-15 versus 0.95 for PHQ-9**, with **GDS-15 of 6 or more giving sensitivity 94% and specificity 88%**, while the standard **PHQ-9 cut-off of 10 or more was too high for older people** (specificity 95% but sensitivity only 71%).

4. Practical implication: the conventional **GDS-15 cut-off of 5 or more misses very few cases of major depression but performs less well for minor depression**; some validation work in the very old supports **6 or more**. For the **PHQ-9, the standard adult cut-off of 10 may be too high in older adults** — consider a lower threshold.

5. Ultra-short versions are now meta-analytically validated. GDS-4 and GDS-5 were assessed across 23 studies (5,048 participants): at **cut-off 2**, **GDS-5 sensitivity 0.85, specificity 0.75**; **GDS-4 sensitivity 0.77, specificity 0.75**. These are the appropriate choices for **resource-limited and high-volume Indian settings**.

6. Screening is now embedded in structural frameworks: - **German S3 CGA guideline (2024): "affect" is one of the six mandatory CGA core dimensions**. - **WHO ICOPE: psychological well-being** is one of the six intrinsic-capacity domains screened, with **PHQ-9** as the step-2 tool. - **ASCO 2023 Practical Geriatric Assessment:** includes a **psychological and depression domain** for every cancer patient aged 65 and above. - **Age-Friendly 4Ms:** depression falls under **Mentation**, alongside dementia and delirium.

7. Indian context. LASI wave-1 found **major depressive disorder in 8.2%** of adults aged 60 and above. Depression is also the **critical multiplier of disability**: the combination of **hypertension plus arthritis plus high depressive symptoms** was associated with **greater ADL–IADL disability than any other multimorbidity combination**, with population attributable fractions of about **22–24%**. In practice, **screening for depression is itself a disability-prevention intervention**.

D. Current Practice Update

Aspect	Pathy's 6th ed. (2022)	Current position (2023–2026)
Who to screen	"Screening in primary care is critical"	**USPSTF 2023: universal depression screening, all adults including 65 and above, Grade B**
Suicide screening	"Suicidal ideation can be detected by screening"	**USPSTF 2023: Grade I — insufficient evidence** for standalone suicide-risk screening
Anxiety in the elderly	Covered as a clinical entity	**Insufficient evidence to screen for anxiety in those 65 and above**, unlike younger adults
Best GDS version	GDS-30 with a 15-item short form	**GDS-15 outperforms GDS-30** (AUC 0.90 versus 0.85); GDS-5 validated for rapid use
GDS cut-off	Not stated	**5 or more** conventional; **6 or more** may be better in the very old; a "good screening, poor case-finding" tool
PHQ-9 in the elderly	Listed as an assessment tool	**Standard cut-off of 10 or more may be too high in older adults** — sensitivity falls to about 71%
Cognitive threshold	**MMSE below 18 means use CSDD**	Unchanged and reaffirmed; GDS accuracy declines even below MMSE 28
Severity monitoring	**MADRS preferred; reassess at least 3-monthly**	Unchanged — remains best practice

E. Exam One-Liners

- **Screening is not severity.** GDS, PHQ and CES-D screen; **MADRS and HDRS measure severity and change**; **CSDD** is for dementia.
- **GDS equals Yesavage and Brink, 1983**; yes/no format; **somatic items deliberately omitted**.
- **GDS-15: 0–4 normal, 5–9 mild, 10 or more moderate to severe. GDS-30: 0–9 normal, 10–19 mild, 20–30 severe.**
- **GDS-15 beats GDS-30** — sensitivity 86%, specificity 79%, **AUC 0.90**.
- **GDS-15 is "good for screening, poor for case-finding"** — it rules out, it does not rule in.
- **MMSE below 18 means abandon the GDS and use the Cornell Scale for Depression in Dementia.**
- **CSDD is the only depression scale validated in dementia** and uses **both informant and patient interview**.
- **HDRS over-scores physically ill older people** because of its many somatic items — and this artefact may explain the "somatic presentation of late-life depression."

- **MADRS is preferred for monitoring** — 10 items, 0–60, sensitive to change, only 2 somatic items.
- **Reassess severity before treatment and at least every 3 months** on any antidepressant, drug or non-drug.
- **PHQ-2 of 3 or more out of 6 is positive**, then proceed to PHQ-9.
- **USPSTF 2023: screen everyone 65 and above for depression (Grade B); suicide-risk screening Grade I.**
- **PGC Morale Scale measures morale, not depression** — overlapping but distinct constructs.
- **Always pair a depression scale with MMSE or MoCA**, and exclude thyroid disease, B12 deficiency, anaemia and hypercalcaemia first.

15. Assessment of Decisional Capacity in an Older Person

A. 20 High-Yield Points

Concepts and Definitions

1. **Capacity and competence are not the same.** A **clinician evaluates a patient's capacity to make a decision**; **competence is a legal term** implying that a person **has been deemed competent or incompetent by a court of law**. Someone not competent to act on their own behalf **requires an agent to act for them**.
2. **Competence** refers to a person's ability to **act reasonably after understanding the nature of the situation being faced**.
3. **Capacity is decision-specific and time-specific.** A patient may have capacity for one decision and not another, and may regain capacity later.
4. **Capacity is presumed until proved otherwise.** A **diagnosis of dementia — even by type — is not a sufficient indicator of a person's ability to comprehend and express a meaningful preference**. Intellectual deficits are **spotty**: a person may get lost or forget things yet still make decisions **with appropriate prompting**.
5. **The ethical foundation is autonomy** — the capacity of each adult and cognitively competent individual to **process information and freely decide on a course of action that best suits their own values, preferences, goals and desires**. Respect for autonomy underpins **informed consent**.
6. **Preconditions of valid informed consent: competence to understand and decide**, and **voluntariness in deciding**. The **information element** is **disclosure of relevant information** about the condition and the proposed course of action.
7. **An unwise decision is not incapacity.** In a patient with capacity, autonomy means "*you may not do things to me without my permission*" — **even if a patient's decision to refuse treatment seems unwise, if they have capacity their refusal stands**, and the clinician's **duty of care does not diminish**; all other care continues without prejudice.
8. **Just as it is wrong to infantilise patients** by directing questions to others who answer faster, **it is equally wrong to prejudge their ability to participate in decisions about their own care**.

The Four Elements of Decisional Capacity

9. **Decisional capacity is predicated on four elements** — the single most examinable list: - **Understanding** — the ability to comprehend the disclosed information about the nature of the situation or medical problem, the procedures involved, and the risks and benefits of undergoing or not undergoing testing or treatment. - **Appreciation** — of the significance of the disclosed information and the potential risks and benefits **for one's own situation and condition**. - **Reasoning** — the ability to engage in a reasoning process about the risks and benefits. - **Choice** — being able to **express a choice** whether or not to undergo further testing or treatment.
10. **The four US legal standards for determining decisional capacity** (a parallel, equally examinable list): - The patient is **aware of the treatment options and expresses a treatment preference clearly and consistently**. - The patient can give a **conventionally understandable reason** for the decision that is **consistent with their past behaviour**. - The patient **remembers and can tell back the information** about diagnosis, prognosis and treatment options given by the practitioner. - The patient can **state what their decision means for their own state of health**.
11. **The sliding-scale principle (Figure 95.1):** the **standard of decisional capacity required rises with the risk of the intervention**. A **fully cognisant patient with good health literacy** can consent to high-risk options such as a **phase 1 clinical trial**; a **cognitively impaired patient lacking decisional capacity could not consent to experimental treatment** and, given reduced life expectancy in advanced dementia, would be unlikely to benefit from highly toxic therapy even if it is standard of care. **The area between is the domain of best clinical judgement.** Hence **"staging the ageing is as important as staging the cancer."**

Assessment in Practice

12. **Distinguish memory from understanding** — a person may fail a memory test yet understand the decision at hand.
13. **No cognitive screening test is appropriate to determine decision-making capacity.** Screening tools that focus on **executive function** — the **Clock Drawing Test** or the **Executive Interview** — are **more likely to be associated with decisional capacity** than the MMSE.
14. **Formal capacity instruments exist but are difficult for older adults**, because they require consideration of **hypothetical situations and complex abstract thinking**. Examples: **MacCAT-T (MacArthur Competence Assessment Tool for Treatment — the reference standard)**, **Aid to Capacity Evaluation (ACE)**, **Capacity to Consent to Treatment Instrument (CCTI)**, **Hopemont Capacity Assessment Interview**, **Understanding Treatment Disclosures**.
15. **A useful practical criterion** in routine informed consent is to **recheck with the patient after an interval** to be certain that they **understand the situation and consistently state the same preference**.
16. **Maximise capacity before declaring its absence.** Before deciding a person cannot decide: correct **sensory deficits**

(a deaf patient may need a hearing aid, many older people need glasses), **treat delirium, depression, pain and infection**, review sedating drugs, use **simple language, repetition and rephrasing**, choose a **quiet, distraction-free environment**, and involve a familiar person. Written material must be legible — a leaflet is useless if **the typeface is too small, the colour contrast is poor, or there is no white space**.

17. **Formal assessment of mental state may be necessary**, but **no specific score is associated with an inability to make decisions about one's personal healthcare** — this is an important negative statement to write in an examination.
18. **Where capacity is absent:** consult any **advance care directive** and the **surrogate or proxy decision-maker**. A proxy may be formally appointed through an **enduring power of attorney**, or the patient's wishes may be recorded in an **advance directive**; if none exists, the lead clinician must decide in the patient's **best interests**, or apply for **guardianship**.
19. **Hierarchy of treatment decision-makers** (Australian model, quoted in the textbook): **enduring guardian with authority → guardian with authority → adult son or daughter → parent → sibling → primary unpaid carer → other person with a close personal relationship and frequent contact**. The clinician goes to the first person who is **18 or older, has full legal capacity, and is reasonably available and willing**.
20. **Communication and documentation:** - **Relatives must not be given the impression that the decision is theirs to make** — they may suffer significant distress from the emotional burden. Their role is to provide **valuable information about what would be in the patient's best interests**. - Where families cannot bear to decide, the humane option is **"informed assent"** — explicitly offering them the choice to **defer to the provider's judgement**, documented clearly in the record. - **Anticipate decisions early in admission** so the patient can be involved as much as possible and **proxy decision-making is avoided**. - Clinicians must disclose: **the condition being treated; the nature of the proposed treatment or procedure; anticipated results; recognised alternatives; and recognised serious risks, complications and benefits, including of non-treatment.** - **Document the assessment, the reasoning and the outcome.**

B. Structured Approach to a Capacity Assessment

Step	Action
1. Define the decision	Capacity is **decision-specific** — state exactly which decision is in question
2. Presume capacity	Start from presumption; a diagnosis alone never establishes incapacity
3. Optimise the person	Treat delirium, pain, depression, infection; correct vision and hearing; review sedatives; choose the best time of day and a quiet setting
4. Disclose	Condition, proposed treatment, expected outcome, alternatives, serious risks, and the consequences of non-treatment — in **plain language**
5. Test the four elements	**Understanding** ("tell me in your own words") · **Appreciation** ("what does this mean for you?") · **Reasoning** ("how did you weigh those up?") · **Choice** ("so what would you like to do?")
6. Apply the sliding scale	Higher risk demands a higher standard of capacity
7. Recheck	Re-ask after an interval to confirm **consistency** of preference
8. Conclude and act	If capacity present, the decision stands even if unwise. If absent, consult advance directive, then surrogate, then best interests or guardianship
9. Document	Record the specific decision, the disclosure made, the four elements, the conclusion and the reasoning

C. The Indian Legal Framework

1. **Mental Healthcare Act (MHCA), 2017** is the governing statute across India and was rewritten after India ratified the **UNCRPD in 2007**. Capacity has a **pivotal role**.
2. **Section 4, MHCA 2017:** every person is **presumed to have the capacity to make mental healthcare and treatment decisions** if they can **understand the information, appreciate the foreseeable consequences of the decision, or communicate the decision by speech, gesture or any other means**.
3. **Memory loss, cognitive deficits, or even a formal diagnosis of dementia do not imply incapacity automatically** — the Act emphasises **establishing capacity**, and gives **preference to the choices and wishes of a person who has capacity**.
4. **The opinion of the assessor or a third party that a decision is "unwise" must not influence the capacity assessment.** Capacity is **specific to the task and to the time**.
5. **Advance directives — Section 5(3):** an advance directive is **invoked only when the person becomes incapacitated** and **remains effective until they regain capacity**. The Act does not clearly distinguish advance statements from advance directives.
6. **Nominated representative:** decision-making by a nominated representative is **restricted to those lacking capacity**

for mental healthcare decisions. **Supported decision-making** ranges from minimal support to complete support.

7. **Other relevant Indian law:** the **Maintenance and Welfare of Parents and Senior Citizens Act, 2007** (maintenance, protection, and transfer-of-property provisions), the **Rights of Persons with Disabilities Act, 2016** (limited guardianship), the **National Trust Act, 1999**, and **Elder Line 14567** for reporting abuse and neglect.
8. **Practical Indian caveat:** the **MacCAT-T, though the international reference standard, is not standardised for the Indian population**, and **family plays a central role in healthcare decision-making** in Indian and other Asian societies — creating real difficulties in operationalising individual autonomy and advance directives.

D. Latest Updates (2023–Present)

1. Indian evidence base is only now emerging. A 2025 descriptive cross-sectional study of mental capacity and advance directives in Indian persons with mental illness confirmed that **published Indian data remain very scarce**, that **the MacCAT-T is regarded as the gold standard but is not standardised for India**, and that the **central role of the family creates practical difficulty in determining advance directives** and enabling individual decision-making about future treatment.

2. Dementia and Indian law. A published analysis of the assessment of dementia under prevailing Indian laws confirms that under **Section 4 of MHCA 2017 a diagnosis of dementia does not itself establish incapacity**, and sets out the four components of the decision-making process as **understanding, appreciating, reasoning and communicating**. It also distinguishes **capacity** (clinical) from **competence** (legal — relating to valid contracts and financial and personal decision-making).

3. UK practice guidance (2024). NHS England guidance restates the functional test as the ability to **understand the relevant information, retain it long enough to make the decision, weigh up the arguments on each side, and communicate the decision** (by talking, signing or any other means). It provides a **practical checklist**: choose a quiet space free from distractions and noise, avoid jargon, **repeat and rephrase questions**, and **identify in advance what the "relevant information" for that specific decision is**. Essex Chambers publishes decision-specific guidance on what constitutes relevant information for different categories of decision.

4. High prevalence of impairment in institutional settings. Older adults evaluated in long-term care have **rates of capacity impairment between 44% and 69%** — an argument for routine, structured assessment rather than ad hoc judgement.

5. Convergence with capacity-based frameworks. The **German S3 CGA guideline (2024)** mandates cognition and delirium assessment as a core dimension; **WHO ICOPE** includes cognition and psychological well-being. Both feed directly into capacity evaluation, and the **Age-Friendly 4Ms** place **"What Matters"** — eliciting the person's own goals — as the first M, operationalising autonomy in routine care.

E. Current Practice Update

Aspect	Textbook position	Current position (2023–2026)
Legal framework	Generic Western frameworks; Australian guardianship hierarchy quoted	**India: MHCA 2017 governs**, with statutory presumption of capacity and nominated representatives
Effect of a dementia diagnosis	A diagnosis is not a sufficient indicator of incapacity	**Statutorily reinforced** — Section 4 MHCA: dementia does not imply incapacity
Instruments	Formal tools exist but are hard for older adults	**MacCAT-T remains the gold standard but is not standardised for Indian populations**
Cognitive tests	No screening test determines capacity; executive tests correlate better	Unchanged and reaffirmed
Role of family	Consult relatives for best interests, but do not transfer the decision	**In India, family-centred decision-making is normative**, creating tension with individual-autonomy models
Documentation	Recommended	Now expected to be **decision-specific**, recording the relevant information disclosed
Autonomy in routine care	Ethical principle	Operationalised as **"What Matters"** in the 4Ms framework

F. Exam One-Liners

- **Capacity is clinical and decision-specific; competence is legal and determined by a court.**
- **The four elements: Understanding, Appreciation, Reasoning, Choice.**
- **Capacity is presumed** — a diagnosis of dementia never establishes incapacity by itself.
- **An unwise decision is not incapacity.** If the patient has capacity, their refusal stands, and the duty of care does not diminish.
- **Sliding scale:** the higher the risk of the intervention, the higher the standard of capacity required.
- **"Staging the ageing is as important as staging the cancer."**

- **No cognitive screening test determines capacity**; **clock drawing and the Executive Interview** correlate better than the MMSE.
- **No specific MMSE score defines incapacity.**
- **MacCAT-T is the reference instrument** — but is not standardised for India.
- **Maximise capacity first:** treat delirium, depression and pain; correct hearing and vision; simplify language; choose a quiet room and the best time of day.
- **Recheck after an interval to confirm the preference is consistent.**
- **Section 4, MHCA 2017 (India):** presumption of capacity; understand, appreciate consequences, communicate by any means.
- **Section 5(3), MHCA 2017:** advance directive is invoked only on incapacity and lapses when capacity returns.
- **Never make the family feel the decision is theirs** — offer **"informed assent"** where they cannot bear to decide.
- **Anticipate decisions early in the admission** so that proxy decision-making is avoided.

Appendices

Appendix A — Four Frequently Confused Terms

1. GOHAI — Geriatric Oral Health Assessment Index

What it is: A self-report scale measuring **oral health–related quality of life** in older people. Developed by **Atchison and Dolan, 1990**.

Why it appears in CGA: It is the tool for the **oral health dimension**. Oral problems are a major hidden cause of the **anorexia of ageing**, weight loss and malnutrition, yet are almost never volunteered by the patient.

Feature	Detail
Items	**12 questions** (a 3-item short form also exists)
Recall period	Past 3 months
Three dimensions	**Physical function** (eating, speaking, swallowing) · **Psychosocial function** (worry about teeth, self-consciousness, avoiding social contact) · **Pain and discomfort** (medication use, sensitivity)
Scoring	Each item 1–5, giving a **total of 12–60**
Direction	**Higher score equals better oral health quality of life**
Interpretation	57 or more high · 51–56 moderate · **50 or less low**

Exam point: GOHAI is more sensitive than clinical dental examination alone, because it captures **functional and psychosocial impact** — a person with dentures may be dentally "treated" but still unable to eat.

2. EHSAS — Elderly Health Status Assessment and Screening Tool

What it is: A **brief, culturally adapted, multidimensional community screening tool developed and validated in India** to identify which rural older adults actually need a full CGA. Developed by **Kshatri and colleagues, ICMR–Regional Medical Research Centre, Bhubaneswar**, with the University of Edinburgh and Maastricht University; validation published in *Age and Ageing* (December 2025).

The problem it solves: CGA is the gold standard but impractical to deliver universally in resource-limited settings. India has almost **150 million people aged 60 and above, about 70% in rural areas** with limited access to specialist geriatric services, and produces only around **20 geriatricians annually**, while each community health worker serves a population of over 1,000.

Feature	Detail
Full form	**E**lderly **H**ealth **S**tatus **A**ssessment and **S**creening
Origin	**ICMR-RMRC Bhubaneswar, Odisha, India**
Structure	**11 items** spanning key geriatric domains, **plus a frailty item not included in the score**
Administered by	**Community health workers (ASHA and Anganwadi workers)**
Cut-off	**3 or more "Yes" responses** triggers referral for CGA
Psychometrics	Cronbach's alpha above 0.7 · test–retest Cohen's kappa above 0.79 · **sensitivity 76.3%, specificity 76.5%, negative predictive value 93.2%**
Criterion variable	Frailty status
Design basis	Aligned with **WHO ICOPE** domains

Exam point: EHSAS operationalises **"stepped assessment"** — brief multidimensional screening at community level, full CGA at facility level. Most multidomain geriatric screening tools are validated in high-income countries and require costly licensing. Its **high negative predictive value is the key design feature** — it is built to safely rule *out*, so scarce specialist time goes only to those who need it. This is the most quotable recent Indian contribution to CGA.

3. MPI — Multidimensional Prognostic Index (Italy)

What it is: A **CGA-derived prognostic index** that converts a standard comprehensive geriatric assessment into a single **mortality-risk score**. Developed by **Pilotto and colleagues, Italy (2008)**, and **specifically recommended by the Italian National CGA Guideline (2024)** to predict negative outcomes across all care settings.

Structure — eight domains, 63 items:

#	Domain	Instrument used
1	Basic ADL	**Katz Index**
2	Instrumental ADL	**Lawton IADL**
3	Cognition	**Short Portable Mental Status Questionnaire (SPMSQ)**
4	Nutrition	**Mini Nutritional Assessment (MNA)**
5	Pressure sore risk	**Exton-Smith Scale**
6	Comorbidity	**Cumulative Illness Rating Scale (CIRS)**
7	Medications	**Number of drugs**
8	Social support	**Co-habitation status**

Scoring: each domain scored **0 (no problem), 0.5 (minor problem), 1 (major problem)**, summed and divided by 8, giving a **continuous score from 0 to 1**.

Grade	Score	Risk
MPI-1	0.00–0.33	**Low risk**
MPI-2	0.34–0.66	**Moderate risk**
MPI-3	0.67–1.00	**Severe risk**

Why it matters: it answers the criticism that CGA generates a lot of data but no single actionable number. MPI is validated to predict **short- and long-term mortality, hospitalisation, length of stay, institutionalisation and readmission** across hospital, community, nursing home and outpatient settings, and is used to guide **treatment intensity decisions** — whether to anticoagulate, whether to offer surgery, whether to escalate oncology treatment.

Exam one-liner: "*CGA tells you what is wrong; the MPI tells you what it means for prognosis.*"

4. Ceiling Effect in the Barthel Index

Definition: A **ceiling effect** occurs when a large proportion of people score **at or near the maximum** on a scale, so the scale **cannot detect any further improvement** and **cannot discriminate between people who are actually different**.

Why the Barthel has one: - The Barthel Index measures only **basic ADLs** — feeding, bathing, grooming, dressing, bowels, bladder, toilet use, transfer, mobility, stairs. - **Anyone who can walk, wash and dress themselves scores 100 out of 100** — even if they cannot cook, shop, handle money, use public transport or manage their own medicines. - A **fully independent 70-year-old** and a **frail 85-year-old who can just about self-care but cannot leave the house** both score **100**. - The scale has therefore **hit its ceiling** — there is no score above 100 to record the difference, and no room to demonstrate improvement in someone already at 100.

Practical consequences:

Consequence	Explanation
Insensitive in ambulatory and community patients	Most community-dwelling elders score at or near maximum, so the scale cannot detect early decline
Cannot measure rehabilitation gains near discharge	A patient improving from 95 to full independence shows no measurable change
Underestimates disability	Misses the entire IADL layer, where **loss occurs first**
Skewed statistics	Scores cluster at one end, violating assumptions of many statistical tests and reducing power in trials

The solution: - Use the **Barthel for hospital, stroke and rehabilitation populations**, where scores are genuinely distributed across the range. - Use the **Lawton IADL scale, gait speed, SPPB or frailty measures** in community-dwelling or relatively well older people. - This is precisely the reason for the **hierarchy rule** in CGA: *do not measure basic ADLs in a robust or pre-frail person — measure frailty instead.*

The mirror image — floor effect: the opposite problem, where everyone clusters at the minimum. The **MMSE has a floor effect in advanced dementia** (many patients score 0 and cannot be distinguished), which is exactly why the **Severe Impairment Battery** was developed.

Exam one-liner: "*Barthel has a ceiling effect in ambulant patients and MMSE has a floor effect in severe dementia — both mean the instrument has run out of room to measure.*"

Appendix B — 3D-CAM and CAM-ICU

Both are **derivatives of the Confusion Assessment Method (CAM)** — the parent instrument — adapted for two different practical problems: "*I have three minutes*" and "*my patient is intubated*."

A. The Parent — CAM (Inouye, 1990)

The diagnostic algorithm — four features:

Feature	Content	How to test
1. Acute onset and fluctuating course	Change from baseline, waxing and waning	Informant or nurse report
2. Inattention	Cannot focus, sustain or shift attention	**Months of the year backwards**, days of the week backwards, **digit span 3 or 4 backwards**; or observe **inappropriate distraction or losing the thread of conversation**
3. Disorganised thinking	Rambling, illogical flow of ideas, incoherence	Orientation to time and place; observe **illogical flow of ideas**
4. Altered level of consciousness	Anything other than alert	Vigilant, lethargic, stuporous, comatose

Diagnosis requires Feature 1 AND Feature 2, PLUS either Feature 3 OR Feature 4.

Performance: sensitivity 94%, specificity 89%, with high inter-rater reliability.

Its limitation: the original CAM tells you *what* to assess but not *how*. Untrained raters have to improvise the mental status testing, so real-world sensitivity in routine practice falls far below 94%. Both 3D-CAM and CAM-ICU exist to fix this by **scripting the questions**.

B. 3D-CAM — the 3-Minute Diagnostic Interview for CAM

Purpose: to make the CAM **fast, scripted and reproducible on a general ward**, so any trained clinician or nurse gets the same answer.

Feature	Detail
Full name	**3-Minute Diagnostic Interview for CAM-defined Delirium**
Developed by	Marcantonio and colleagues, 2014
Duration	**About 3 minutes**
Structure	**20 items** — 10 patient-response cognitive and symptom items plus 10 interviewer observations, mapped onto the 4 CAM features
Setting	**General medical and surgical wards**; verbal, non-intubated patients
Key advantage	**Specific scripted questions** for every feature, removing improvisation

Validation performance (mean age 84):

Population	Sensitivity	Specificity
Patients WITH dementia	**96%**	**86%**
Patients WITHOUT dementia	**93%**	**96%**

Note the pattern: in dementia, sensitivity stays excellent but **specificity falls** — because chronic cognitive impairment mimics inattention and disorganised thinking. This is the classic difficulty of **delirium superimposed on dementia**.

Ultra-brief screening derived from 3D-CAM data:

Screen	Sensitivity	Specificity
"Months of the year backwards" alone — the single best item	**83%**	**69%**
Months backwards plus "What is the day of the week?" — best two items	**93%**	**64%**

These two-question screens are designed as a **rule-out**: negative means delirium is unlikely; positive means proceed to full 3D-CAM or CAM.

C. CAM-ICU — for the Non-Verbal or Intubated Patient

Purpose: to assess delirium in patients who **cannot speak** — intubated, tracheostomised, or too sedated for verbal testing. Developed by **Ely and colleagues, 2001**.

Prerequisite step: first assess arousal with the **Richmond Agitation–Sedation Scale (RASS)**. - **RASS −4 or −5** (deeply sedated or unarousable) means **stop, the patient is unassessable**; reassess later. - **RASS −3 to +4** means proceed. - **Any RASS other than 0** satisfies Feature 4.

The four features, adapted for a non-verbal patient:

Feature	CAM-ICU method
1. Acute change or fluctuating course	Change from baseline mental status, or fluctuation in RASS or sedation score over 24 hours
2. Inattention	**Attention Screening Examination** — auditory: "*Squeeze my hand every time you hear the letter A*", then read **"S-A-V-E-A-H-A-A-R-T"**. An error is squeezing on a non-A **or failing to squeeze on an A**. **More than 2 errors equals inattention.** A picture-based visual version is the alternative
3. Disorganised thinking	**Four yes/no questions** answered by hand squeeze (for example "*Will a stone float on water?*", "*Are there fish in the sea?*", "*Does one pound weigh more than two pounds?*", "*Can you use a hammer to pound a nail?*") **plus a two-part command** (hold up two fingers, then do the same with the other hand)
4. Altered level of consciousness	**RASS score other than 0**

Same algorithm: Features 1 plus 2, plus 3 or 4.

Performance: sensitivity approximately **80%**, specificity approximately **95%**. A positive CAM-ICU is trustworthy, but **hypoactive and mild delirium can be missed**, especially in routine bedside use.

D. Head-to-Head Comparison

	CAM	3D-CAM	CAM-ICU
Year	1990	2014	2001
Setting	Any	**General ward, outpatient**	**ICU, intubated or non-verbal**
Patient must speak?	Yes	Yes	**No**
Time	5–10 min	**3 min**	2–3 min
Structure	Algorithm only, unscripted	**20 scripted items**	Scripted, non-verbal responses
Attention test	Months and days backwards, digit span	Months backwards, digit span, scripted items	**"SAVEAHAART" letter-A hand squeeze**
Arousal measure	Clinical judgement	Clinical judgement	**RASS (mandatory first step)**
Sensitivity	94%	**93–96%**	About 80%
Specificity	89%	**86–96%**	**About 95%**
Main weakness	Rater-dependent; needs training	Specificity falls in dementia (86%)	Misses hypoactive and mild delirium

E. Latest Updates (2023–Present)

1. The 4AT is now the main competitor on general wards. It requires **no special training**, takes **under 2 minutes**, incorporates **AMT-4** and the **months backwards** test, and — uniquely — **scores unassessable or drowsy patients as positive** rather than excluding them. Pooled accuracy is broadly comparable to 3D-CAM (sensitivity about 80–90%, specificity about 80–90%), and it is the tool recommended in most current UK delirium pathways.

2. Ultra-brief two-item screening is now standard practice as the first step of a two-stage pathway: **UB-2 (months backwards plus day of the week) → if positive, 3D-CAM or 4AT → if positive, full clinical assessment**. This mirrors the screening and assessment split used elsewhere in CGA.

3. Delirium superimposed on dementia remains the unsolved problem. All CAM derivatives lose specificity when baseline cognitive impairment is present. Current work focuses on **informant-reported acute change** — the key discriminator — and on **fluctuation over time** rather than cross-sectional testing.

4. Electronic and automated delirium detection. Record-derived delirium prediction models and **automated chart-based CAM (CHART-DEL)** are used for retrospective case-finding and quality audit; wearable and actigraphy-based detection of sleep–wake fragmentation is under investigation.

5. Framework integration. Cognition and delirium is one of the **six mandatory core dimensions** of the German S3 CGA guideline (2024). Delirium sits under **Mentation** in the **Age-Friendly 4Ms**. The Italian CGA guideline (2024) found CGA **reduces delirium risk** across settings.

6. Prevention still outperforms detection. The **Hospital Elder Life Program (HELP)** multicomponent intervention remains the benchmark — targeting orientation, sleep, mobility, vision, hearing and hydration — and is the reason delirium is classed as a geriatric syndrome with shared, modifiable risk factors.

F. Exam One-Liners

- **CAM algorithm: Features 1 plus 2, plus 3 or 4** — acute and fluctuating, inattention, disorganised thinking, altered consciousness.
- **CAM: sensitivity 94%, specificity 89%.**

- **3D-CAM is the 3-minute scripted CAM for the general ward**; sensitivity **96%** with dementia, **93%** without.
- **Specificity of 3D-CAM falls to 86% in dementia** — the delirium-superimposed-on-dementia problem.
- **Best single delirium item: "months of the year backwards"** (sensitivity 83%, specificity 69%).
- **Best two items: months backwards plus day of the week** (sensitivity 93%, specificity 64%).
- **CAM-ICU is for the patient who cannot speak** — responses by hand squeeze.
- **CAM-ICU attention test: "SAVEAHAART" — squeeze on A; more than 2 errors equals inattention.**
- **CAM-ICU requires RASS first**; **RASS −4 or −5 means unassessable**, and any RASS other than 0 satisfies Feature 4.
- **CAM-ICU is highly specific (about 95%) but less sensitive (about 80%)** — it misses hypoactive delirium.
- **4AT needs no training and scores drowsy or unassessable patients as positive** — its main advantage over the CAM family.
- **Hypoactive delirium is the commonest and most missed subtype** — always screen, never wait for agitation.

MD GERIATRIC MEDICINE

Examination Revision Compilation

Elder Mistreatment & Alcohol Use in Older Adults

Primary sources: Pathy's Principles and Practice of Geriatric Medicine, 6th edition; Kane's Essentials of Clinical Geriatrics

Supplemented with: International and Indian guidelines and peer-reviewed evidence, 2023–2026

Format: High-yield points → Latest Updates → Current Practice Update → Exam One-Liners

Prepared for Dr. Sudarsan · 19 August 2026

Contents

1. **Elder abuse / mistreatment** — epidemiology, risk factors, types, forms, identification and management **[15 marks]**
2. **Role of the social worker in identifying elder mistreatment** **[15 marks]**
3. **Effects / ill effects of alcohol in older adults and how to address alcoholism** **[15 marks]**
4. **Alcohol de-addiction** **[15 marks]**

Q1. Elder Abuse / Mistreatment — Epidemiology, Risk Factors, Types, Identification and Management [15 marks]

A. Definition and Concept

1. Elder abuse is **any act or omission that endangers an older person's life, threatens their physical or psychological integrity, restricts their freedom, or compromises their personal development or financial security** (Council of Europe, 1987). The WHO Toronto Declaration adds the key element of a **relationship of expectation of trust**.
2. Two broad forms: **individual mistreatment** (act or omission by a third party against a person) and **institutional abuse**, which arises within a care facility and reflects **inadequate governance**.
3. **Neglect must be separated from abuse.** Neglect arises from forgetfulness, ignorance, or inattention to professional duty — **there is no intent to harm**. Abuse implies an act or omission that harms, whether intentional or not.
4. A related concept is **psychological control ("abuse of weakness")** — cognitive, affective and behavioural conditioning that removes the older person's capacity for discernment, so they make decisions they would not otherwise make. The perpetrator is usually a **close relative, neighbour or family member**.

B. Epidemiology

1. Meta-analysis (Yon et al.): **about 15.7% of community-dwelling older people worldwide experience abuse each year**; **psychological abuse (11.6%)** and **material/financial abuse (6.8%)** are commonest.
2. **Vulnerable people of any age are the usual targets** — children, people with disability, those with loss of autonomy, and **especially those with cognitive disorders**.
3. In long-term care: staff-to-resident physical abuse, financial abuse, sexual assault, and the under-recognised **resident-to-resident aggression (RRA)** — negative, aggressive, intrusive physical, sexual, verbal or material interactions between residents. RRA linked to **28 deaths in Australia (2000–2014)** and **101 fatal incidents in the US (2003–2016)**.
4. **Indian data (LASI Wave 1):** among elders reporting ill-treatment — **77.3% verbal/emotional, 26.5% economic exploitation, 23.7% physical, 52.6% neglect**. Highest State prevalence in Bihar (~12%) and Karnataka (~10%). Commonest perpetrators: **son, then daughter-in-law**.

C. Classification — Forms of Abuse (Council of Europe)

1. **Physical violence** — beating, wounding, restraint, mishandling, **sexual violence**, disregard for pain.

2. **Psychological violence** — inappropriate familiarity, **infantilisation**, verbal violence, deprivation of visits.
3. **Material and financial violence** — embezzlement, misappropriation of money or inheritance, **abusive power of attorney**.
4. **Medical violence** — lack of basic and appropriate care, withholding information about treatment.
5. **Drug abuse** — misuse of **sedatives and neuroleptics as chemical restraint**; physical restraint is a closely linked harm (~60% prevalence in German nursing homes in 2009).
6. **Violation of rights** — deprivation of identity, freedom of movement, civil rights, religious practice; **misuse of legal protection/guardianship**.

D. Risk Factors

1. **Victim factors:** poor health, **physical and cognitive impairment**, dementia, ADL dependency, social isolation, female sex, age 80+, financial dependence.
2. **Perpetrator / environment factors: shared living arrangement** with a relative or friend suspected of **alcohol or substance misuse, mental illness, or a history of violence**; caregiver burden and exhaustion; institutional understaffing and poor governance.
3. **Societal factor — ageism.** Ageism, stereotyping and social exclusion create the climate in which mistreatment is tolerated; **technopenia** (lack of access to useful technology — 53% of French elders aged 75+ use little or no internet) is itself argued to be a form of deprivation.

E. Identification and Diagnosis

1. **Screening for elder abuse is a core part of Comprehensive Geriatric Assessment** in frail, dependent patients, alongside functional and environmental/social assessment. **Ambulatory-care quality indicators require clinicians to identify older people at high safety risk, including elder abuse/neglect, and to develop a plan for assessment or referral.**
2. **Red flags: frequent emergency department visits** for injury or exacerbation of chronic illness; unexplained bruises, skin tears or fractures; **new-onset falls**; sudden withdrawal or behavioural change; delayed presentation; inconsistency between history and injury pattern.
3. **Interview the older person alone**, separately from the accompanying caregiver, in a **safe and private setting**. Corroborate with collateral history, examine the whole body, document injuries objectively, and assess **decision-making capacity**. Abuse is a **crime in only a few jurisdictions** (e.g. California); prosecution is difficult when the victim has dementia and cannot withstand cross-examination.

F. Management and Prevention

1. **Immediate step is safety** — treat injuries, assess risk of further harm, arrange separation or respite where danger persists. The **WHO ICOPE social-care pathway explicitly includes elder abuse** alongside daily activities, finances and loneliness, with a **parallel care pathway for the caregiver**.
2. **Support the caregiver.** Advise carers to seek help early rather than reach exhaustion — carer training associations, **support and respite care**, temporary cognitive-behavioural units. This is an important part of the preventive response.
3. **Institutional prevention:** team support organised by the service head, **analysis of professional practices**, planned ethical reflection, **ageing-simulator training** to build empathy, and **inclusion of residents in social-life / patient committees** as an exercise in health democracy.
4. **Legal and system-level response:** adult-safeguarding legislation (**UK Care Act 2014**; Australian Law Reform Commission recommendation for adult-safeguarding agencies), mandatory serious-incident reporting, coronial investigation of premature deaths. **India — Maintenance and Welfare of Parents and Senior Citizens Act 2007 (Amendment Bill 2019), Elder Line 14567, NALSA free legal aid, Integrated Programme for Senior Citizens.**
5. **Prevention is a duty of public authorities, associations and professionals**, guided by fundamental ethical principles; **medical and economic vulnerability are the situations that most often lead to maltreatment**.

Latest Updates (2023–Present)

1. **USPSTF 2025 (JAMA, July 2025) — the headline update.** The USPSTF recommends screening for intimate partner violence in women of reproductive age (**B recommendation**), but concludes that current evidence is **insufficient** to assess the balance of benefits and harms of screening for caregiver abuse and neglect of older or vulnerable adults — an **"I" statement**, unchanged in substance from 2018. It applies only to persons **without recognised signs and symptoms**; clinicians must still evaluate anyone with red flags.
2. **Screening tools perform poorly.** For the **Hwalek–Sengstock Elder Abuse Screening Test (H-S/EAST)**, sensitivity was only **46%** and specificity **73%** for detecting physical or verbal abuse — the key reason for the "insufficient evidence" verdict.
3. **Optimal delivery conditions emphasised.** The USPSTF highlighted concern about potential **harms** of screening and specified that optimal screening occurs in **safe and private settings**.
4. **Emergency department focus (Geriatric ED Guidelines 2.0, 2025–26).** Systematic reviews of ED-based screening tools for patients aged 60+ and of ED-initiated interventions (literature Dec 2018 – Dec 2024) concluded that the evidence for the impact of ED screening or intervention strategies is **still not well characterised**.

5. **Newer ED tools — ERASE.** A 2024–25 scoping review identified six nurse-administered ED screening tools; with **ERASE**, **3.9% screened positive** and **88% of assessors** agreed the starting and signalling questions helped map objective indicators. ED nurses are well positioned to identify abuse because of time spent observing patients.
6. **Indian data 2023–2026. HelpAge India 2023**: nearly **50%** of elders face emotional, physical or financial abuse but only about **15% report it**. **HelpAge India 2024**: awareness of the Maintenance and Welfare Act is only **9%**. **NCRB**: **18% rise** in abuse cases between 2023 and 2024; the 60+ population is projected to exceed **20%** of India's population by 2050.
7. **Indian community prevalence.** *Indian Journal of Community Medicine* 2024 (Mangaluru): prevalence **44.6%** (rural 50.7%, urban 38.6%), significantly associated with being unemployed, living in extended families, and staying with children.

Current Practice Update

Aspect	Textbook position	Current evidence / guideline (2023–2026)
Universal screening	Screening for abuse recommended as part of CGA in frail, dependent elders	**USPSTF 2025: "I" statement** — insufficient evidence for routine screening of asymptomatic older adults; case-finding on red flags remains mandatory
Screening tools	Tools referenced without accuracy data	**H-S/EAST sensitivity only 46%** — poor accuracy is now the stated reason for the I statement
Setting of detection	Ambulatory and long-term care emphasis	**ED is now the priority setting** (Geriatric ED Guidelines 2.0); **ERASE** and nurse-administered tools emerging
Prevalence figures	Yon meta-analysis, global ~15.7%	Indian community studies show **much higher rates (44–50%)** with only ~15% reporting
Legal framework	UK Care Act 2014; Australian adult-safeguarding proposals	**India: Maintenance & Welfare Act 2007 + 2019 Amendment Bill, Elder Line 14567** — but awareness only 9%
Harms of screening	Not addressed	USPSTF now explicitly cautions that screening must occur in **safe, private settings** to avoid precipitating retaliation

Exam One-Liners

- **Definition to quote:** "A single or repeated act, or lack of appropriate action, occurring within any relationship where there is an expectation of trust, which causes harm or distress to an older person" — WHO / Toronto Declaration.
- **Six Council of Europe forms:** physical, psychological, material/financial, medical, drug (chemical restraint), violation of rights.
- **Commonest type globally = psychological; commonest in India = neglect and verbal/emotional.**
- **Commonest perpetrator in India = son, then daughter-in-law.**
- **Neglect ≠ abuse** — neglect lacks intent to harm.
- **RRA = resident-to-resident aggression**, the most under-recognised form in long-term care.
- **USPSTF 2025 = "I" statement** for elder/caregiver abuse screening; **B recommendation** only for IPV in reproductive-age women.
- **Red flag triad:** repeated ED visits + unexplained bruises/fractures + delayed presentation.
- **Always interview the elder alone.**
- **Indian helpline = Elder Line 14567. World Elder Abuse Awareness Day = 15 June.**

Q2. Role of the Social Worker in Identifying Elder Mistreatment [15 marks]

A. Why the Social Worker Is Central

1. Elder mistreatment is a **biopsychosocial problem, not a purely medical one**. Its roots lie in family dynamics, dependency, finances and housing — domains of social work practice rather than clinical medicine.
2. The social worker is a **core member of the interdisciplinary geriatric team**. Once acute clinical problems are treated, the physician engages nursing, physiotherapy, occupational therapy and **social work** to restore the patient's highest level of function and quality of life.
3. **Comprehensive Geriatric Assessment mandates an environmental and social assessment**, focusing especially on the caregiver's support and the resources available to meet the patient's needs — the domain in which mistreatment is most often first suspected.
4. RCTs of geriatric assessment show that **social worker assessment and referral in the community** reduced mortality, reduced hospital use, reduced permanent nursing home placement, and delayed development of disability — evidence that the role produces hard outcomes.
5. Social workers can obtain **additional certified training in Gerontology**, giving formal expertise in ageing-specific safeguarding issues.

B. Identification — Specific Functions

1. **Comprehensive psychosocial / biopsychosocial assessment.** In institutional care done **within 1–2 weeks of admission** and repeated as needed, to determine past social history, **family relationships and social resources**, and to identify serious psychosocial signs warranting mental-health referral.
2. **Understanding family dynamics.** The social worker **assists other care team members in understanding the family dynamics** — the key to recognising coercion, dependency, and the "abuse of weakness" pattern.
3. **Detecting financial and material abuse.** Assessment of finances, pension, property and **abusive power of attorney** is a social work function; financial exploitation is otherwise invisible on clinical examination.
4. **Home and environment evaluation.** Assessing the fit between the older person and their environment (e.g. the **Housing Enabler**) exposes neglect, unsafe living conditions and confinement.
5. **Recognising caregiver strain as a precursor.** About **35% of caregivers of those aged 50+ report moderate-to-high financial strain**; the social worker identifies exhaustion and burden before it converts into neglect, using the **Zarit Burden Interview** and **AMA Caregiver Self-Assessment**.

6. **Screening within the WHO ICOPE pathway.** The ICOPE social care and support assessment covers **daily activities, living conditions, finances, loneliness, elder abuse, social engagement and participation**, with a parallel **care pathway for the caregiver** — the social worker's operational checklist.
7. **Interviewing skills.** Trained to interview the older person **alone, privately and non-judgementally**, obtain collateral history from family and neighbours, and document disclosures accurately.
8. **Assessing discharge safety.** Discharge planning is done with **social work, case management, nursing and physical therapy**, matching services to clinical needs, personal values, and **social and financial resources** — an unsafe discharge back to an abusive household is a preventable harm.

C. Intervention and Advocacy

1. **Advocacy role.** The hospice/palliative model defines the social worker as one who provides **advice and counselling to the patient and all family members during the crisis period, and acts as an advocate for the patient and family in making use of community resources**. The same role applies in mistreatment.
2. **Linking to community resources.** The classic example is identifying resources providing **friendly visiting for the socially isolated older adult** — isolation being both risk factor for and consequence of abuse.
3. **Caregiver support and respite.** Directs carers to **training associations (e.g. Alzheimer's associations), support and respite care, and temporary cognitive-behavioural units** — an important part of the preventive response.
4. **Legal and protective referral.** Referral to adult-safeguarding agencies under frameworks such as the **UK Care Act 2014** and the Australian Law Reform Commission recommendations. **India: referral to the Maintenance Tribunal under the Maintenance and Welfare of Parents and Senior Citizens Act 2007, Elder Line 14567, NALSA legal aid, and the medical social worker post sanctioned under NPHCE at Regional Geriatric Centres and District Hospital geriatric clinics.**
5. **Institutional prevention.** Supports **analysis of professional practices, team support for staff, ethical reflection**, and the running of **social-life committees in residential homes and patients' committees in hospitals** — an exercise in health democracy giving residents a voice.
6. **Combating ageism.** Social work education and awareness campaigns address ageist attitudes that legitimise mistreatment, including **ageing-simulator training** for institutional staff.
7. **Documentation and continuity.** Communicates relevant psychosocial data to the interdisciplinary team and ensures concerns raised at one contact are not lost across transitions of care.

Latest Updates (2023–Present)

1. **Formal recognition of the specialised social worker role (Journal of Elder Abuse & Neglect, 2023–24).** In hospital- and ED-based elder mistreatment response teams, the social worker's unique skillset allows them to establish close connections with community resources, collaborate with hospital stakeholders, support patients/families/caregivers, navigate legal and protective systems, and **balance patient safety against quality of life in disposition decisions**. Their defined role: **comprehensive biopsychosocial assessment, safe discharge planning, appropriate referral**. Underpins the **Vulnerable Elder Protection Team (VEPT, New York)** and **VESPA (Colorado)** models.
2. **ED social workers often lead — but are under-trained.** In EDs with a social worker on the clinical team, that social worker may be expected to **lead** the assessment for potential elder mistreatment and initiate interventions, yet many **lack training or experience** and are unclear about their responsibilities. A dedicated **ED Elder Mistreatment Assessment Tool for social workers** has been developed by modified Delphi consensus.
3. **Multidisciplinary teams (MDTs) as the standard response model.** MDTs provide regular, coordinated case review across practice areas; integrated case review is associated with greater interagency collaboration and more efficient delivery of assistance. MDTs bring together health, social service and justice professionals for complex cases of abuse, neglect and self-neglect.
4. **Social workers are the largest professional group in these teams.** National Elder Abuse MDT Survey (January 2026): the largest group of respondents identified as **Adult Protective Services (APS) / social workers**, followed by aging services, victim services, healthcare professionals, law enforcement, prosecutors and long-term care ombudsmen; **78%** integrate person-centred practices and **67%** use trauma-informed practices.
5. **Bridging the ED–APS gap.** ED providers focus on **acute medical needs** while APS focuses on **long-term psychosocial needs**; assigning **named liaison individuals** in both entities improves communication, and both sides value MDT participation.
6. **Engagement training for frontline workers.** The **"Opening the Door" (OTD)** training was developed with APS caseworkers to enhance engagement skills in suspected cases, since APS caseworkers are the primary responders and ideally positioned to improve detection. Capacity varies widely — **75%** of APS programmes in large counties had regular multidisciplinary case review versus only **28%** in small counties.

Current Practice Update

Aspect	Textbook position	Current practice (2023–2026)
Social worker's role	Generic team member for psychosocial assessment, resource linkage, discharge planning	**Specialised elder-mistreatment social worker** with a defined role on formal ED/ hospital response teams (VEPT, VESPA)
Setting	Community, outpatient, nursing home, hospice	**Emergency department is now the priority identification setting**; ED social worker often leads assessment
Tools	Housing Enabler, Zarit Burden Interview, psychosocial assessment forms	Dedicated **ED Elder Mistreatment Assessment Tool for social workers** (Delphi-derived); MDT case-selection algorithms
Model of response	Individual referral to safeguarding agency	**Multidisciplinary team (MDT) case review** as the standard, with trauma-informed and person-centred practice
Training	Optional gerontology certification	Explicit recognition of a **training gap**; OTD-type engagement training being rolled out
India	Not specified	**Medical social worker under NPHCE**, Elder Line 14567, Maintenance Tribunal referral — but Act awareness only ~9%

Exam One-Liners

- The social worker performs the **biopsychosocial assessment** — the missing dimension in a purely medical evaluation.
- **Three core functions:** comprehensive biopsychosocial assessment, safe discharge planning, appropriate referral.
- **Environmental and social assessment focusing on the caregiver's support** is a mandatory CGA component.
- Social worker assessment and referral in the community **reduces mortality, hospital use and nursing home placement** (RCT evidence).
- The social worker **helps the team understand family dynamics** and **acts as advocate for the patient**.
- **WHO ICOPE** social care pathway explicitly lists **elder abuse** alongside finances, loneliness and living conditions.
- **MDT case review** is the current gold-standard response model; **APS/social workers are its largest professional group**.

- **VEPT (New York) and VESPA (Colorado)** = named hospital-based elder mistreatment response teams.
- India: **medical social worker under NPHCE**; refer to **Maintenance Tribunal** and **Elder Line 14567**.
- Key gap to quote: **most ED and hospital social workers lack formal elder-mistreatment training.**

Q3. Effects / Ill Effects of Alcohol in Older Adults and How to Address Alcoholism [15 marks]

A. Definitions and Burden

1. **Alcohol use disorder (AUD) is the commonest form of substance use disorder in older adults.** Alcohol falls under **socially sanctioned psychoactive substances**, alongside medications (prescribed and non-prescribed) and illicit substances.
2. **One-year community prevalence of AUD: 2.75% in elderly men and 0.51% in elderly women.** Far higher in **primary care, where at-risk drinking is 5–15%**. **Older military veterans** have a particularly high prevalence.
3. Overall SUD prevalence is **underestimated**, because **DSM-5 criteria have limited applicability to this age group** — tolerance, work impairment and legal problems are poor markers in a retired, socially isolated person.
4. Two subgroups: **early-onset** (lifelong drinkers surviving into old age) and **late-onset** drinkers who begin heavy drinking in later life, often after bereavement, retirement or loss.

B. Why the Older Person Is More Vulnerable

1. **Reduced total body water and lean mass with increased fat** gives a **higher blood alcohol concentration for the same intake**; reduced first-pass metabolism and hepatic blood flow prolong exposure.
2. **The same amount of alcohol that caused no untoward social or physical effects in middle age now causes difficulty** — which is precisely why **older adults and their relatives are often in denial**.
3. **Even modest alcohol consumption produces excessive disability and poorer perceived health** in older adults with chronic physical and mental health disorders.
4. **The ageing brain is especially vulnerable** — those who begin drinking heavily late in life show cognitive impairment comparable to those who have drunk for decades.

C. Ill Effects — Systemic

1. **Neurocognitive. Alcohol-related cognitive decline** is a recognised neurocognitive disorder. Chronic heavy intake causes **brain atrophy and dementia**; there is a **doubling of the odds of later severe memory impairment** in those with a history of AUD. **Memory and executive function** are worst affected, with **prefrontal and frontal damage** on neuroimaging. Contributions of alcohol, **thiamine deficiency** and ageing are hard to separate.
2. **Persistence after abstinence.** A **lifetime history of alcohol dependence — even without current heavy use — causes sustained impairment** in memory, motor and attention/executive domains. **Word-list recall (free verbal recall)** is a sensitive discriminator.

3. **Even moderate-heavy drinking impairs brain networks.** On functional MRI, moderate-heavy older drinkers show **decreased central executive network connectivity during a working memory task**, with no difference in default mode network connectivity.
4. **Psychiatric.** Worsens depression and anxiety, causes insomnia, and **older adults with AUD face a greater risk of suicide**.
5. **Falls, injury and delirium.** Impairs gait and balance, precipitates falls and fractures, and is a common precipitant of **delirium** — both from intoxication and from withdrawal.
6. **Drug interactions.** Combining alcohol with **prescription and OTC medications, especially psychoactive agents**, is a major hazard; **cross-tolerance between alcohol and benzodiazepines** is clinically important and, if unrecognised, leads to dependence being overlooked. **15% of over-65s requiring inpatient alcohol detoxification are dependent on a second substance**, usually a hypnotic, anxiolytic or analgesic.
7. **Systemic disease.** Liver disease, pancreatitis, cardiomyopathy, arrhythmia, hypertension, malnutrition, anaemia, immunosuppression, and **malignancy** (alcohol is an **IARC Group 1 carcinogen**).
8. **Social consequences.** Self-neglect, financial loss, family and marital discord, and — importantly for the geriatrician — **being an abuser or victim in elder mistreatment**: a shared living arrangement with a relative suspected of **alcohol or substance misuse** is a recognised risk factor for elder abuse.

D. The Moderate-Drinking Paradox (Textbook Nuance)

1. Observational evidence suggests **moderate intake (1–3 standard drinks/day) may be associated with reduced risk of cognitive impairment**. In the **Framingham Offspring Cohort**, **late-life (not midlife) moderate consumers had larger hippocampal volume** and light consumers better episodic memory than abstainers. In the **MoVIES project**, minimal and moderate drinking were associated with less decline on MMSE and Trail Making Test.
2. **Interpret with great caution.** In the reanalysed **PAQUID** study (20-year follow-up, 3676 subjects), dementia risk was lower only in light drinkers (**HR 0.85**), non-significant in moderate drinkers (**HR 0.79**) and neutral in heavy drinkers (**HR 1.01**). Likely explanation: **light-to-moderate intake is merely a marker of a broader favourable psychosocial behaviour pattern**, not a protective agent. **Confounding dominates this literature — never advise an abstainer to start drinking.**

E. Addressing Alcoholism — Identification

1. **Screening is part of CGA.** The screening question is simply **"Do you drink alcohol?"**, followed by **CAGE questionnaire** (primary screening tool), **Michigan Alcohol Screening Test-9 / Short MAST-Geriatric version (SMAST-G)**.
2. **Case-finding, not self-presentation.** Older people present with **physical problems**, not with a drinking complaint. They may not realise they are ill or that help exists, they **under-report**, services staffed by young professionals feel intimidating, and **stigma** is

high because the disorder is seen as self-inflicted. **Physicians find it hard to discuss substance misuse with older people.**

3. **Approach.** A **non-judgemental and tactful approach is essential**, especially in ageing women. Home carers and the multiple professional contacts an older person has are an **underdeveloped opportunity for detection** and need better training.

F. Addressing Alcoholism — Management

1. **Brief interventions work.** Structured brief intervention by primary care physicians — **five minutes, five brief sessions** targeting at-risk drinking — is often fairly effective.
2. **Pharmacotherapy. There is some evidence supporting naltrexone.** There is **insufficient evidence to endorse disulfiram, acamprosate or topiramate** in older adults.
3. **Education on interactions.** Specific advice about combining alcohol with prescription and OTC drugs, **especially psychoactive agents, must be given and regularly reinforced**.
4. **Psychosocial approaches must be routine** — support groups, **Alcoholics Anonymous**, individual counselling and family intervention — alongside treatment of comorbid depression, which perpetuates the disorder.

Latest Updates (2023–Present)

1. **WHO 2023 — the "no safe level" statement.** In January 2023 the WHO stated in *The Lancet Public Health* that while cancer risk rises substantially with amount consumed, **there is no threshold below which alcohol is safe**, whatever the beverage type. **IARC classifies alcohol as a Group 1 carcinogen**; it is a major risk factor for oral, pharyngeal, laryngeal, oesophageal, liver, pancreatic, colorectal and breast cancer, accounting for about **741,300 new cancer cases (4%)** worldwide annually.
2. **US Surgeon General Advisory, January 2025.** Issued an advisory on the alcohol–cancer link, calling for **cancer warning labels** on alcoholic beverages and for health professionals to routinely discuss alcohol use with patients. Alcohol is now described as a leading cause of cancer **after tobacco and obesity**, accounting for about **5% (~97,000) of new US cancer cases**.
3. **But the evidence is contested — needed for a balanced answer.** The **2018 Global Burden of Disease** study concluded zero alcohol was safest, but its **2022 update** introduced an age-specific, risk-weighted model asserting that in populations with high baseline cardiovascular disease rates — **particularly older adults** — light-to-moderate consumption could carry net health benefits. In January 2025 the Surgeon General discouraged any intake while a simultaneous **NASEM (National Academies)** report emphasised **tailoring recommendations to individual risk**, finding no association between moderate consumption and colorectal, oral cavity, pharyngeal, oesophageal or laryngeal cancers. **Mendelian randomisation** studies, however, found no benefit and increased risk even at low doses.

4. **Public awareness is poor.** A 2025 *JAMA Oncology* analysis of the 2024 Health Information National Trends Survey (6,793 US adults) found only **37%** knew alcohol increases cancer risk, and about **9%** believed it had no effect or reduced risk.
5. **Treatment gap remains enormous.** An estimated **29 million** US residents had AUD in 2023, yet only about **2% received pharmacological treatment**. Alcohol-related deaths in the US have risen by **29% since 2020**, driven mainly by liver disease.
6. **Reduction, not only abstinence, is now an accepted goal.** Reducing drinking even without full abstinence improves health outcomes — a harm-reduction framing particularly suited to frail older patients in whom total abstinence may be unrealistic.

Current Practice Update

Aspect	Textbook position	Current evidence (2023–2026)
Safe level	Moderate intake (1–3 drinks/day) may reduce cognitive impairment risk	**WHO 2023 / Surgeon General 2025: no safe level for cancer risk**; GBD 2022 and NASEM 2025 dissent, allowing possible net benefit in older adults at high CV risk — **the field is genuinely contested**
Advising abstainers	Confounding limits interpretation	**Universal: never advise a non-drinker to start** — unchanged and uncontroversial
Cancer	Not emphasised as an alcohol harm	**Alcohol = IARC Group 1 carcinogen**, 3rd leading modifiable cancer cause; 7 cancer sites
Screening tool	CAGE, SMAST-G, MAST-9	**AUDIT-C** increasingly preferred; USPSTF supports unhealthy-alcohol-use screening in adults
Treatment goal	Abstinence-oriented	**Harm reduction / drinking reduction accepted as a valid endpoint**
Public messaging	Not addressed	Cancer warning labels proposed; **only 37% of the public know the alcohol–cancer link**

Exam One-Liners

- **AUD is the commonest substance use disorder in older adults.**
- Prevalence: **2.75% elderly men, 0.51% elderly women**; **5–15% at-risk drinking in primary care**.
- **DSM-5 criteria are poorly applicable** to older adults → under-diagnosis.

- **Same intake, higher blood level** — less body water, more fat, reduced first-pass metabolism.
- **Even modest intake causes excessive disability** in those with chronic disease.
- **Alcohol-related cognitive decline** = a named neurocognitive disorder; **doubling of odds of severe memory impairment**.
- **Memory and executive function** worst hit; **prefrontal/frontal atrophy**.
- **Late-onset drinkers are as impaired as lifelong drinkers** — the ageing brain is uniquely vulnerable.
- **Higher suicide risk** in older adults with AUD.
- **Screening = CAGE / SMAST-G**; ask "Do you drink alcohol?" in every CGA.
- **Brief intervention = 5 minutes × 5 sessions.**
- **Naltrexone has some evidence; disulfiram, acamprosate, topiramate — insufficient evidence in the elderly.**
- **Alcohol = IARC Group 1 carcinogen; WHO 2023: no safe level.**

Q4. Alcohol De-addiction [15 marks]

A. Principles

1. **Treatment of substance misuse is a multistage process integrating physical, psychological and social interventions.** These should run **concurrently, not consecutively**, and must be **acceptable to the individual and sensitive to the specific needs of the elderly**.
2. **Five sequential components:** (i) **identification of the case**, (ii) examination of the patient's **attitude and motivation**, (iii) **detoxification** where physical dependence exists, (iv) treatment of **perpetuating comorbidity** (especially depression), and (v) **psychological rehabilitation and social intervention**.
3. **Nihilism is the enemy.** Pessimistic attitudes held by professionals and carers are frequently shared by the patient — a **fatalistic resignation to a life of substance misuse**, particularly in long-term users, while **more recent-onset users express greater motivation for treatment**.

B. Step 1 — Case Identification

1. **Clinical observation allied to sensitive yet persistent enquiry**, supported by **routine use of standardised screening tools** (CAGE, SMAST-G, MAST-9) to focus clinical impression accurately.
2. Patients rarely present complaining of alcohol; they present with **associated physical problems** — falls, delirium, malnutrition, liver disease, poor diabetes control, or unexplained functional decline.

C. Step 2 — Motivation and Assessment

1. **Examine the patient's attitude to their drinking. Exploration of the risks and discussion of potential avenues for change** helps establish or reinforce **motivation to change**.
2. **Assess comorbidity and polysubstance use.** Always calculate the **total benzodiazepine or opioid dose using class-specific equivalence charts**, because **cross-tolerance exists within drug classes and also between alcohol and benzodiazepines** — widely exploited during detoxification, but causes dependence to be missed if overlooked.
3. **Assess the social matrix.** Housing, and **a social network consisting mainly of substance misusers**, perpetuates the problem and must be examined for opportunities to change.

D. Step 3 — Detoxification (Withdrawal Management)

1. **Drugs causing significant physical dependence necessitate a detoxification regimen.** In older adults, **safe withdrawal may take weeks to months, compared with days to weeks in younger adults** — the single most important age-specific point.
2. **The withdrawal period in the elderly is far more likely to be complicated by confusion (delirium)** than in younger adults. Withdrawal may also present with **psychotic episodes with hallucinations and delusions, nocturnal restlessness, paradoxical excitement, anxiety and increased suicide risk**.
3. **Setting.** Where severe dependency is suspected or rapid withdrawal is necessary, detoxification **is best conducted in an inpatient setting**. **Rapid detoxification risks breakthrough withdrawal symptoms and may be complicated by convulsions.**
4. **Supportive measures: parenteral thiamine before any glucose load** (to prevent Wernicke's encephalopathy), correction of electrolytes and magnesium, hydration, nutrition, and a quiet, well-lit, orientating environment with delirium precautions.
5. **Benzodiazepine-assisted withdrawal** uses the alcohol–benzodiazepine cross-tolerance principle; in older adults a **shorter-acting agent (e.g. lorazepam)** is preferred where hepatic impairment exists, with **symptom-triggered rather than fixed-dose regimens** to limit total exposure.

E. Step 4 — Pharmacotherapy for Relapse Prevention

1. **Naltrexone has some supporting evidence in older adults.** There is **insufficient evidence to endorse disulfiram, acamprosate or topiramate** in this age group. Disulfiram is particularly hazardous in the elderly because of cardiovascular comorbidity.
2. **Treat comorbid depression adequately**, since it perpetuates the disorder; do not treat anxiety in the recovering drinker with **benzodiazepines**, given cross-dependence risk.

F. Step 5 — Psychological Rehabilitation

1. **After detoxification, rehabilitation is necessary** to address the issues behind the substance use and to foster **future coping strategies**. Detoxification alone is not treatment.
2. **Outcomes equal those in younger patients.** Janik and Dunham compared over **3,000 people aged over 60** with younger entrants to alcohol treatment programmes: **at six months there were no differences between the groups** — a powerful argument against therapeutic nihilism.
3. **Age-specific programmes improve retention.** Kofoed et al. showed **greater retention in an age-specific outpatient group focused on socialisation and minimal confrontation**, compared with older patients in a mixed-age group — although at one-year follow-up the effect was lost.
4. **Low-confrontation models are superior in the elderly.** Kashner et al. found **elders in a confrontational programme had half the abstinence rate at one year** compared with a programme promoting **self-esteem, tolerance and peer relationships**.

Confrontation, traditionally regarded as fundamental to overcoming denial, is **inappropriate for older adults**.

5. **Effective components:** models encouraging development of **social networks with self-management skills**; **behavioural approaches including cue identification and avoidance**; and **variants of the AA 12-step model tailored to elders** (reported with varying success).
6. **Psychosocial approaches must be routine** — support groups, AA, individual counselling and **family intervention**.
7. **Address the psychosocial aetiology.** Four theories guide intervention: **social control theory** (weak social bonds), **social learning theory** (role models), **behavioural economics / behavioural choice theory** (lack of alternative rewards — educational, social, physical activities), and **stress and coping theory** (substance use as avoidance coping). **Providing alternative rewarding activities is therapeutic, not merely recreational.**
8. **Aftercare** may be delivered through **generic old-age psychiatry services or specialist drug services**, depending on which best meets the individual's needs. **Avoid drugs with dependence potential** in ongoing prescribing where practical.

Latest Updates (2023–Present)

1. **Standard pharmacotherapy — current consensus.** Three medications are FDA-approved for AUD — **acamprosate, naltrexone and disulfiram** — with **gabapentin and topiramate** as additional evidence-based options; naltrexone is also available as an **extended-release intramuscular** formulation. **Acamprosate and oral or XR naltrexone are the preferred pharmacologic treatments** for moderate or severe AUD, and head-to-head trials have not consistently shown either superior. **This diverges from the textbook's "insufficient evidence for acamprosate."**
2. **Adherence and pill burden matter.** Acamprosate requires **two tablets three times daily**, oral naltrexone is **once daily**, and XR naltrexone is given **every 28 days** — XR naltrexone is therefore attractive in older adults with polypharmacy or poor adherence.
3. **More options in Europe. Baclofen, nalmefene and sodium oxybate** are approved in some European regions; naltrexone and nalmefene are safe and effective particularly in patients not yet abstinent, and **targeted (as-needed) naltrexone** significantly reduces drinks per drinking day.
4. **GLP-1 receptor agonists — the major emerging development.** A **phase 2 double-blind RCT of once-weekly subcutaneous semaglutide** over 9 weeks in adults with AUD evaluated alcohol consumption and craving; semaglutide was generally well tolerated (decreased appetite, GI symptoms, headache), participants lost 5% body weight, and no serious adverse events occurred. Current guidance: consider semaglutide **only after failed trials of the FDA-approved medications**, with careful comorbidity screening. **Phase 3 trials are underway**; high cost may limit adoption.

5. **Behavioural treatment components standardised.** The most effective AUD interventions comprise **assessment, personalised feedback, motivational interviewing, goal setting, planning and review of homework, problem-solving skills, and relapse prevention and management**.
6. **Psychedelic-assisted therapy — investigational.** Hallucinogenic (**psilocybin**) and dissociative (**ketamine**) agents combined with psychotherapy may reduce heavy drinking days and increase abstinent days, but more research is needed.

Current Practice Update

Aspect	Textbook position	Current guideline / evidence (2023–2026)
Acamprosate	Insufficient evidence in older adults	**Now a first-line preferred agent** (with naltrexone) for moderate–severe AUD; caution in renal impairment — relevant to the elderly
Naltrexone	Some evidence; oral	**Preferred**; **XR intramuscular naltrexone every 28 days** solves the adherence problem in older patients
Second-line agents	Topiramate — insufficient evidence	**Gabapentin and topiramate now recognised evidence-based alternatives**
European options	Not covered	**Baclofen, nalmefene, sodium oxybate** approved in parts of Europe; **as-needed (targeted) naltrexone** for craving
Novel agents	None	**Semaglutide** — positive phase 2 RCT, phase 3 ongoing; **third-line after approved drugs fail**
Treatment goal	Abstinence	**Reduction in drinking is a legitimate outcome** (harm reduction)
Psychotherapy	AA, counselling, family intervention	Formalised as **motivational interviewing + CBT + relapse prevention**; low-confrontation age-specific approach still validated
Treatment gap	Not quantified	**Only ~2% of people with AUD receive pharmacotherapy** — the biggest practical failure

Exam One-Liners

- **De-addiction = a multistage process integrating physical, psychological and social interventions, run concurrently, not consecutively.**
- **Five steps:** identify → motivate → detoxify → treat comorbidity → rehabilitate.
- **Safe withdrawal takes weeks to months in the elderly vs days to weeks in the young.**
- **Elderly withdrawal is complicated by confusion/delirium** far more often; **rapid detoxification risks convulsions**.
- **Thiamine before glucose, always.**
- **Inpatient detoxification** if severe dependency or rapid withdrawal is required.
- **Cross-tolerance between alcohol and benzodiazepines** — exploited for detoxification, dangerous if ignored.
- **15% of over-65s in alcohol detoxification are dependent on a second drug** (hypnotic, anxiolytic or analgesic).
- **Confrontational programmes halve one-year abstinence rates in the elderly** — use **low-confrontation, self-esteem-promoting, age-specific** groups.
- **Outcomes at 6 months are no different from younger patients** — never be nihilistic.
- **Naltrexone (± XR) and acamprosate are now the preferred pharmacotherapy**; disulfiram is hazardous in the elderly.
- **Semaglutide = the emerging third-line option after approved drugs fail.**
- **Only ~2% of people with AUD receive pharmacotherapy** — the treatment gap is the headline statistic.

MD Geriatrics Exam Answers

MD Geriatric Medicine — High-Yield Exam Answers

Endocrine, Metabolic, Renal and Genitourinary Systems (Questions 1–17)

Structured 15-mark answers • Pathy's Principles and Practice of Geriatric Medicine (6th ed.) with Kane's Essentials of Clinical Geriatrics, supplemented by current international guidelines and Indian data

Q1. Investigation and Management of Diabetic Ketoacidosis

A. Definition and Basic Concept

1. **DKA** is an acute metabolic emergency defined by the triad of **hyperglycaemia, ketosis and metabolic acidosis**, caused by absolute or relative insulin deficiency with excess counter-regulatory hormones.
2. In older people DKA is less common than **HHS (hyperosmolar hyperglycaemic state)**, but many elderly patients present with a **"mixed" picture** — glucose 270–450 mg/dL, pH 7.2–7.3, without marked dehydration or coma.
3. Older subjects with hyperglycaemic comas have **higher mortality, longer hospital stay, more previously undiagnosed diabetes, more renal impairment**, and need larger insulin doses than the young.

B. Precipitating Factors in the Elderly

4. Commonest precipitants are **infection (chest, urine, foot), myocardial infarction, stroke, inadequate or omitted insulin, and inappropriate drug treatment**.
5. **Thiazide diuretics and corticosteroids** raise blood glucose and can precipitate DKA; thiazides and furosemide particularly precipitate HHS.
6. **Care-home residence, dementia, thirst impairment and dependence on carers** are important risk factors; COVID-19 and other acute illness can cause rapid decompensation.

C. Clinical Features

7. Classical polyuria, polydipsia and weight loss may be **absent or blunted**; presentation is often **non-specific — confusion, falls, drowsiness, incontinence, poor oral intake**.
8. Look for **Kussmaul breathing, ketotic breath, dehydration, hypotension, tachycardia** and abdominal pain; **hypothermia and absent fever** may mask underlying sepsis.

D. Investigations

9. **Capillary and venous blood glucose**; venous (not arterial) blood gas for **pH and bicarbonate**.
10. **Bedside quantitative beta-hydroxybutyrate (β-OHB)** is the preferred ketone test; urine ketones (nitroprusside) detect only acetoacetate and are less reliable.
11. **Serum electrolytes** — sodium (corrected for glucose), potassium, chloride, bicarbonate; **anion gap**; **blood urea and creatinine**.
12. **Effective serum osmolality** = 2 × Na (mEq/L) + glucose (mg/dL)/18 — to identify overlap with HHS.
13. **Search for precipitant**: full blood count, CRP, blood and urine cultures, chest X-ray, **ECG and troponin** (silent infarction is common), foot examination.
14. **Diagnostic criteria (current international consensus)**: glucose ≥200 mg/dL **or** known diabetes; **β-OHB ≥3.0 mmol/L** or urine ketones ≥2+; **pH <7.3 and/or bicarbonate <18 mEq/L**. Severity is graded by pH, bicarbonate and mental state.
15. **Euglycaemic DKA** (glucose <200 mg/dL) must be actively excluded in patients on **SGLT2 inhibitors**, in starvation, and in chronic liver disease.

E. Management

16. **Fluids first** — 0.9% saline or balanced crystalloid, typically 1 litre over the first hour, then titrated. In the elderly with **heart failure or CKD, give slower and monitor closely** for pulmonary oedema.
17. **Fixed-rate intravenous insulin infusion 0.1 units/kg/hour**, without a bolus in most patients; aim to fall β-OHB by 0.5 mmol/L/hour and glucose by 50–75 mg/dL/hour.
18. **Potassium**: withhold insulin if K <3.3 mEq/L until replaced; add 20–40 mEq/L to fluids when K is 3.3–5.2 mEq/L; withhold if K >5.2 mEq/L.

19. **Add 5–10% dextrose** when glucose falls to about 250 mg/dL, continuing insulin until ketosis clears. **Bicarbonate only if pH <7.0**; **phosphate only if <1.0 mg/dL** with cardiac or respiratory weakness.
20. **Resolution**: β-OHB <0.6 mmol/L with pH >7.3 or bicarbonate ≥18 mEq/L; then overlap intravenous insulin with **subcutaneous basal insulin by 1–2 hours** before stopping the infusion.
21. **Complications to prevent**: hypoglycaemia, hypokalaemia, fluid overload, aspiration, venous thromboembolism, and (rarely in old age) cerebral oedema.
22. **Prevention**: sick-day rules, ketone meters at home, structured education of patients and care-home staff, avoidance of insulin omission, and review of diabetogenic drugs.

Latest Updates (2023–Present)

- **ADA/EASD/AACE/JBDS/DTS Consensus Report on Hyperglycaemic Crises in Adults (Diabetes Care, 2024)** — the first major revision in 15 years. Key changes: glucose threshold lowered to **≥200 mg/dL or a known history of diabetes** (to capture euglycaemic DKA); **quantitative β-OHB incorporated into diagnosis, severity grading and resolution**; the **anion gap criterion removed**; **arterial sampling abandoned** in favour of venous gases.
- The same consensus **simplified treatment to three pillars — fluids, insulin and potassium**, and for the first time endorsed **subcutaneous rapid-acting insulin protocols in a non-critical-care ward for mild and uncomplicated moderate DKA**.
- The 2024 consensus provides the **first formal HHS resolution criteria** (normalisation of effective osmolality and recovery of mental status) and redefines HHS hyperosmolarity as **effective osmolality >300 mOsm/kg or calculated total osmolality >320 mOsm/kg**.
- **SGLT2 inhibitor–associated euglycaemic DKA** is now a recognised inpatient hazard; current guidance is to **withhold SGLT2 inhibitors during acute illness, fasting and around surgery**, which is directly relevant to older multimorbid patients.
- **ADA Standards of Care (2025 and 2026), Older Adults section** — reiterates that in frail and care-dependent older adults, treatment should be **simplified and de-intensified**, with the primary aim of **avoiding both hypoglycaemia and hyperglycaemic crisis** rather than achieving a tight HbA1c.
- Published commentary on the 2024 consensus has questioned the **omission of 0.45% saline**, since many patients have true free-water deficits once sodium is corrected for hyperglycaemia — a practical caution in the dehydrated older patient.

Current Practice Update

Aspect	Textbook Position	Current Evidence / Guideline
Ketone assessment	Urine ketones / clinical acidosis	**Quantitative bedside β-OHB** for diagnosis, severity and resolution
Glucose criterion	Marked hyperglycaemia expected	**≥200 mg/dL or known diabetes**; euglycaemic DKA formally recognised
Acid–base testing	Arterial blood gas, anion gap used	**Venous gas**; anion gap dropped from criteria
Insulin route	Intravenous infusion for all	**Subcutaneous rapid-acting insulin acceptable** in mild/uncomplicated moderate DKA on the ward
HHS resolution	Not formally defined	**Defined criteria** — normal effective osmolality plus recovered mental status
Drug precipitants	Thiazides, steroids emphasised	**SGLT2 inhibitors** added as an important modern precipitant

Exam One-Liners

- DKA in old age is often a **"mixed" DKA–HHS picture** with modest acidosis.
- **β-OHB ≥3.0 mmol/L** is the modern ketosis criterion; **anion gap is no longer required**.
- Insulin is withheld until **K >3.3 mEq/L**.
- Add **dextrose at glucose ~250 mg/dL**; treat the **ketosis, not the glucose**.
- **Overlap IV and subcutaneous insulin by 1–2 hours** to avoid rebound.
- Suspect **euglycaemic DKA** in any unwell patient on an SGLT2 inhibitor.

Q2. Newer Oral Antidiabetic Agents

A. Background and Rationale

1. Metformin remains **first-line after lifestyle measures** in older adults; it lowers glucose without hypoglycaemia, causes modest weight loss, reduces microvascular complications, and reduced cardiovascular events and mortality in obese patients in UKPDS.
2. Long-term metformin causes **vitamin B12 deficiency**; periodic measurement is advised. It must be avoided in severe renal impairment and withheld in dehydration, sepsis and before contrast or surgery.
3. Newer agents were developed because **sulphonylureas cause hypoglycaemia and weight gain** and **thiazolidinediones cause fluid retention, heart failure and fractures** — both particularly hazardous in the elderly.

B. DPP-4 Inhibitors (Gliptins)

4. **Sitagliptin, vildagliptin, linagliptin, saxagliptin, alogliptin, teneligliptin** inhibit degradation of GLP-1 and GIP, giving a **near-physiological, glucose-dependent** incretin effect.
5. HbA1c reduction **0.5–1.0%**; **weight neutral, no intrinsic hypoglycaemia, minimal adverse effects** — making them among the safest agents in frail older adults.
6. **Linagliptin needs no dose adjustment in renal failure** (CARMELINA showed cardiovascular and renal safety in high-risk patients); others require dose reduction.
7. As a class they are **cardiovascular-neutral**; **saxagliptin increased heart-failure hospitalisation** (SAVOR-TIMI 53) and is best avoided in cardiac patients.

C. SGLT2 Inhibitors (Gliflozins)

8. **Empagliflozin, dapagliflozin, canagliflozin, ertugliflozin** block proximal tubular glucose reabsorption, giving **insulin-independent glycosuria**.
9. HbA1c reduction **0.5–1.0%**, with **weight loss of 2–3 kg**, blood pressure reduction, and **no hypoglycaemia** when used alone.
10. **Empagliflozin and canagliflozin reduce MACE; empagliflozin reduces cardiovascular death, heart-failure hospitalisation and worsening nephropathy** — benefits now extended to patients without diabetes.
11. Cautions in the elderly: **volume depletion, postural hypotension and falls, genital mycotic infection, urinary infection, euglycaemic ketoacidosis, weight loss aggravating sarcopenia**, and (with canagliflozin) amputation risk.

D. GLP-1 Receptor Agonists (including the oral form)

12. **Oral semaglutide (Rybelsus)** is the first orally available GLP-1 receptor agonist; it must be taken fasting with ≤120 mL water and 30 minutes before food.
13. GLP-1 receptor agonists lower HbA1c by **1.0–2.0%**, cause **weight loss of 1.6–3 kg or more**, and **liraglutide, semaglutide and dulaglutide reduce cardiovascular events** in high-risk patients.
14. In older adults, **nausea, vomiting, anorexia, weight loss and sarcopenia** limit their use; a **43% discontinuation rate** was seen with exenatide in participants aged ≥65.

E. Prescribing Principles in the Older Patient

15. Choose by **renal and hepatic function, frailty, hypoglycaemia risk, cardiac and renal comorbidity, cost and swallowing ability** — not by HbA1c alone.
16. **Consider a glinide (repaglinide) if eating is irregular**, avoid SGLT2 inhibitors where weight loss, dehydration or foot disease predominate, and use **gliclazide** if a sulphonylurea is unavoidable.
17. **De-intensification (deprescribing)** is an active clinical duty in those who are frail, underweight, cognitively impaired or renally impaired — simplification or withdrawal is usually feasible without loss of control.
18. In India, cost, availability of generic **teneligliptin, vildagliptin and dapagliflozin**, and coverage under **AB-PMJAY** strongly influence choice; screen for **tuberculosis and genital/urinary infection** before starting SGLT2 inhibitors.

Latest Updates (2023–Present)

- **Tirzepatide**, a dual **GIP/GLP-1 receptor agonist**, gives greater HbA1c and weight reduction than semaglutide or insulin glargine in the **SURPASS** programme; a pooled post-hoc analysis of SURPASS-1 to -5 (2025) found **HbA1c reductions of 1.97–2.10% in participants aged ≥65 with BMI <30 kg/m²**, with no age-related loss of efficacy or safety.
- **ADA Standards of Care 2026 (Older Adults)** states that **GLP-1 receptor agonists and tirzepatide are highly effective with low hypoglycaemia risk and may be used at reduced eGFR, including during dialysis**.
- **High-dose oral semaglutide (25 mg and 50 mg)** has shown additional HbA1c and weight benefit over the 14 mg dose in the PIONEER PLUS and OASIS programmes, expanding oral options.

- **Small-molecule oral GLP-1 receptor agonists (e.g. orforglipron)** — not requiring fasting administration — have completed late-phase trials and represent the next oral class.
- **Finerenone**, a non-steroidal mineralocorticoid receptor antagonist, is now standard add-on therapy for **diabetic kidney disease with albuminuria** (FIDELIO-DKD/FIGARO-DKD), reducing kidney and cardiovascular events; potassium monitoring is essential in the elderly.
- Guidelines have moved decisively from a **glucose-centred to an organ-protection-centred** algorithm: **SGLT2 inhibitors for heart failure and CKD, GLP-1 receptor agonists for atherosclerotic disease and obesity**, chosen independently of HbA1c.
- **Beers 2023 criteria** continue to advise avoiding **long-acting sulphonylureas (glibenclamide/glyburide)** in older adults, and caution with sliding-scale insulin.

Current Practice Update

Aspect	Textbook Position	Current Evidence / Guideline
Sequencing after metformin	Free choice of "any second agent"	**Comorbidity-driven**: SGLT2i for HF/CKD, GLP-1 RA for ASCVD/obesity, regardless of HbA1c
Incretin classes	GLP-1 RA and DPP-4 inhibitors	**Dual GIP/GLP-1 (tirzepatide)** now the most potent agent, efficacy preserved in ≥65 years
Oral incretin therapy	Not available	**Oral semaglutide up to 50 mg**; small-molecule oral GLP-1 RAs in late-phase trials
Renal protection	ACE inhibitor / ARB	ACEi/ARB **plus SGLT2 inhibitor plus finerenone** as pillars
Older-adult strategy	Achieve individualised HbA1c	Explicit emphasis on **de-intensification and simplification** in frailty

Exam One-Liners

- **Metformin first, always check B12**.
- **DPP-4 inhibitors** — safest class in frailty; **linagliptin** needs no renal adjustment.
- **SGLT2 inhibitors** — cardio-renal protection, but dehydration, genital infection and euglycaemic DKA.
- **Saxagliptin and heart failure; canagliflozin and amputation** — two classic negative associations.
- **Tirzepatide** = dual GIP/GLP-1; most potent HbA1c and weight reduction.
- **Deprescribing is a prescribing decision** in the frail older diabetic.

Q3. Newer Insulin Analogues

A. Concept and Rationale

1. Insulin analogues are **recombinant insulins with altered amino-acid sequence or acylation** that change absorption kinetics, producing either **faster prandial action** or **flatter, longer basal profiles**.
2. The direction of insulin development has been to make insulin **"longer, faster and easier"**.
3. Human insulin exists as a **hexamer** in the vial and must dissociate to dimers and monomers before absorption — the step that analogues manipulate.

B. Rapid-Acting (Bolus) Analogues

4. **Insulin lispro, aspart and glulisine** have one or two amino-acid changes that weaken self-association; onset **5–15 minutes**, peak earlier and higher, duration **4–6 hours**.
5. They can be injected **immediately before or even after a meal** — a major advantage in older people with variable appetite, dysphagia or dementia.
6. Regular human insulin, by contrast, starts in 30 minutes and lasts 5–8 hours, causing late post-prandial hypoglycaemia.

C. Ultra-Rapid Formulations

7. **Faster aspart (Fiasp)** contains **niacinamide and L-arginine**, accelerating monomer formation and capillary permeation, with greater early glucose lowering.
8. **Lispro-aabc (LY900014/URLi)** contains **treprostinil** (local vasodilatation) and **citrate** (increased vascular permeability), speeding absorption.

9. **Inhaled insulin (Afrezza)** uses a dry powder on fumaryl diketopiperazine microparticles, with onset about **12 minutes** and offset by **90–180 minutes**; contraindicated in asthma and COPD.

D. Long-Acting (Basal) Analogues

10. **Insulin glargine (U100 and U300), detemir and degludec (U100/U200)** provide flat 24-hour basal cover with **less nocturnal hypoglycaemia than NPH**.
11. **Degludec** is a detemir-derived molecule with **B30 threonine deleted and a 16-carbon fatty diacid attached to B29 lysine via a glutamic acid spacer**, forming subcutaneous multihexamers; duration exceeds 42 hours and permits flexible timing.
12. **Glargine U300 is as effective as U100 in patients over 65 with less nocturnal hypoglycaemia** — hence long-acting analogues are the **preferred starting insulin in older adults**.

E. Combinations and Practical Use

13. **Co-formulations**: IDegAsp (degludec/aspart, Ryzodeg) — the only basal insulin that can be mixed with a rapid analogue in one injection; premixed analogues (Humalog Mix 25/50, NovoMix 30) suit twice-daily regimens.
14. **Fixed-ratio basal insulin + GLP-1 combinations — IDegLira (degludec/liraglutide)** and **iGlarLixi (glargine/lixisenatide)** — reduce injections, weight gain and hypoglycaemia.
15. In older adults with type 1 diabetes or those unable to manage basal-bolus regimens, **twice-daily premixed analogue** or **twice-daily long-acting analogue** is a reasonable simplification; HbA1c target at age ≥70 is about **7.0–7.5%**.
16. Insulin therapy carries a **higher hypoglycaemia risk than most oral agents** in older adults; **sliding-scale insulin should not be used** in hospital.
17. **Biosimilar glargine and aspart** have made analogue therapy affordable in India, an important consideration for pensioners and AB-PMJAY beneficiaries.

Latest Updates (2023–Present)

- **Once-weekly basal insulins** are the major advance. **Insulin icodec** (ONWARDS 1–6) and **insulin efsitora alfa** (QWINT 1–5) achieved **non-inferior HbA1c reduction versus daily glargine or degludec in type 2 diabetes**.
- **QWINT-1 (NEJM, 2025)** used a **fixed-dose escalation** and showed about **40% fewer clinically significant or severe hypoglycaemic events than glargine** in insulin-naïve type 2 diabetes; **QWINT-3 and QWINT-4 (Lancet, 2025)** confirmed non-inferiority in basal-experienced and basal-bolus patients.
- In **type 1 diabetes**, weekly insulins are less favourable — **QWINT-5** showed **higher rates of level 2/3 hypoglycaemia with efsitora versus degludec**, and ONWARDS 6 raised similar concerns for icodec.
- Practical implication for geriatrics: weekly insulin reduces injections from **365 to 52 per year**, potentially transforming care in **care homes, in patients dependent on visiting nurses or family carers, and in those with dementia** — but requires care with dose changes, since the effect of an error persists for days.
- **ADA Standards of Care 2026** emphasise **simplification of complex insulin regimens** in older adults, preferential use of **analogues over NPH and human regular insulin**, and pairing insulin therapy with **continuous glucose monitoring** to detect asymptomatic nocturnal hypoglycaemia.
- **Continuous glucose monitoring** is now recommended for older insulin-treated adults, with **time-in-range 70–180 mg/dL >50–70% and time-below-range <1%** as the geriatric targets, replacing HbA1c-only assessment.

Current Practice Update

Aspect	Textbook Position	Current Evidence / Guideline
Basal insulin frequency	Once or twice daily	**Once-weekly icodec and efsitora** non-inferior in type 2 diabetes
Weekly insulin in type 1 DM	Not applicable	**Higher hypoglycaemia** — not recommended as a routine option
Choice of basal insulin in elderly	Analogue preferred over NPH	Unchanged and **strengthened**; NPH and human regular discouraged
Monitoring	HbA1c and capillary glucose	**CGM with time-in-range metrics**, especially for hypoglycaemia detection
Regimen complexity	Individualise	Formal **simplification/de-intensification** protocols recommended

Exam One-Liners

- **Rapid analogues** — lispro, aspart, glulisine; onset 5–15 min, may be given after meals.
- **Faster aspart = niacinamide + arginine; lispro-aabc = treprostinil + citrate**.

- **Degludec** — ultra-long, multihexamer formation, flexible dosing, mixable with aspart.
- **Glargine U300 causes less nocturnal hypoglycaemia than U100 in those over 65**.
- **IDegLira and iGlarLixi** — fixed-ratio basal insulin plus GLP-1 agonist.
- **Weekly insulins** work in type 2 but not safely in type 1.

Q4. Diabetic Foot Syndrome

A. Definition and Burden

1. **Diabetic foot syndrome** is the combination of **infection, ulceration or destruction of tissues of the foot** associated with **neuropathy and/or peripheral artery disease** in a person with diabetes.
2. The majority of the elderly diabetic population is **at increased risk of foot ulceration**; foot ulcer independently predicts **amputation and mortality**.
3. **About 70% of lower-limb amputations are precipitated by foot ulceration**, and **three-year survival after major amputation is only about 50%** — worse than many cancers.
4. Increasing age and a higher level of amputation both increase **length of hospitalisation and cost**.

B. Risk Factors (Pathy's Table 86.3)

5. **Peripheral sensorimotor neuropathy** (the primary cause in the vast majority), **autonomic neuropathy**, **peripheral vascular disease**, **limited joint mobility**, **abnormal foot pressures and deformity**, **previous foot problems**, **visual loss**, and **alcohol excess**.
6. **Limited joint mobility and visual loss** are specifically geriatric — they impair the patient's ability to **inspect their own feet**.
7. In India, additional risks are **barefoot walking, ill-fitting footwear, delayed presentation, use of traditional remedies, and poor glycaemic control**.

C. Pathogenesis

8. **Neuropathy** causes loss of protective sensation, small-muscle wasting with clawing and high plantar pressures, and callus formation over metatarsal heads.
9. **Autonomic neuropathy** produces dry, fissured skin and arteriovenous shunting with a warm, well-perfused but vulnerable foot.
10. **Peripheral artery disease** in diabetes is typically **infra-popliteal and multi-segmental**, with medial calcification producing falsely high ankle pressures.

D. Clinical Features and Assessment

11. Neuropathic symptoms include **numbness, burning or lancinating pain, "pins and needles" and hyperaesthesia, typically worse at night**, with gait disturbance and falls.
12. **Diabetic ulcers form at weight-bearing sites** — metatarsal heads and heel — and are typically **deep, with undermined edges and surrounding hyperkeratosis, and painless**.
13. Assessment: **10 g Semmes-Weinstein monofilament, 128 Hz tuning fork, ankle reflexes, ankle-brachial index and toe pressures, pedal pulses, footwear inspection**, and **probe-to-bone test** for osteomyelitis.
14. **Charcot neuroarthropathy** presents as a hot, swollen, erythematous foot with preserved pulses; misdiagnosis as cellulitis or gout leads to permanent midfoot collapse (rocker-bottom foot).

E. Management

15. Management addresses the **underlying aetiology plus debridement, control of infection, appropriate dressings and offloading**, with **optimum glycaemic control** as the metabolic foundation.
16. **Offloading** is the single most important intervention for a neuropathic plantar ulcer; **revascularisation** is the key intervention in an ischaemic ulcer.
17. **Infection** is graded clinically; mild infections need oral Gram-positive cover, moderate/severe need broad-spectrum cover, urgent surgical drainage and admission.
18. **Multidisciplinary foot care team** — diabetologist, podiatrist, vascular surgeon, orthotist, microbiologist and specialist nurse — reduces amputation rates.
19. **Prevention**: annual risk stratification, daily foot inspection (with a mirror or by a relative if vision is poor), never walking barefoot, therapeutic footwear, professional nail and callus care, and prompt reporting of any lesion.
20. Careful caution: **corn plasters containing salicylic acid have caused ulceration** in diabetic patients with poor skin

integrity or neuropathy, and must be avoided.

Latest Updates (2023–Present)

- **IWGDF 2023 Guidelines** (practical, classification, prevention, offloading, infection with IDSA, PAD with ESVS/SVS, wound healing) are the current international standard.
- **Classification**: **SINBAD** (Site, Ischaemia, Neuropathy, Bacterial infection, Area, Depth) is now the **priority system for inter-professional communication and audit**; infection is graded by **IWGDF/IDSA** and ischaemia by **WIfI**. Ulcers are described as **neuropathic, neuro-ischaemic or ischaemic**.
- **Offloading**: a **non-removable knee-high device (total contact cast or rendered-irremovable walker) is first choice** for neuropathic plantar forefoot ulcers; a removable knee- or ankle-high device is second choice; **felted foam** is an alternative where nothing else is available.
- **Adjunctive wound therapies** (only after 4–6 weeks of failed optimal care): **sucrose-octasulfate impregnated dressing** in non-infected neuro-ischaemic ulcers without severe ischaemia; **autologous leucocyte/platelet/fibrin patch**; **placental membrane allografts**; **systemic hyperbaric oxygen** in ischaemic ulcers; **topical oxygen** in non-healing non-infected ulcers.
- **Strong negative recommendations**: do **not** use **topical phenytoin** or **herbal-impregnated dressings** for wound healing — directly relevant to Indian practice where such applications are common.
- **IWGDF/IDSA 2023 infection guideline** supports **shorter antibiotic courses**, including approximately **3 weeks rather than 6 weeks** for diabetic foot osteomyelitis when infected bone has been surgically resected, and diagnosis of soft-tissue infection on **clinical inflammatory signs** rather than swab culture.
- **India**: rising diabetic foot burden with LASI data showing high undiagnosed diabetes in older adults; **NPCDCS foot-care screening** and district-level podiatry services remain the main structural gap.

Current Practice Update

Aspect	Textbook Position	Current Evidence / Guideline
Ulcer classification	Wagner / University of Texas	**SINBAD** for communication and audit; **WIfI** for ischaemia; **IWGDF/IDSA** for infection
Offloading	Debridement, dressings, offloading (general)	**Non-removable knee-high device is first-line**, explicitly ranked above all alternatives
Advanced dressings	Wound Healing Society guidance	**Sucrose-octasulfate, leucocyte-platelet-fibrin patch, placental allografts** — but only after 4–6 weeks of failed standard care
Herbal/topical agents	Not addressed	**Strongly advised against** (phenytoin, herbal dressings)
Osteomyelitis antibiotics	Prolonged courses	**~3 weeks post-resection** may suffice; shorter courses favoured

Exam One-Liners

- **70% of amputations follow an ulcer; 3-year survival after amputation ≈ 50%**.
- **Neuropathy is the commonest single contributor**; PAD determines healing.
- **SINBAD** is the current first-choice ulcer classification.
- **Total contact cast** is the gold standard for the neuropathic plantar ulcer.
- A **hot swollen foot with good pulses = Charcot until proved otherwise**.
- **Never use corn plasters or herbal dressings** on a diabetic foot.

Q5. Testosterone Replacement Therapy in the Older Male

A. Physiological Background

1. After age 30, **free testosterone falls by about 1% per year**; in the European Male Ageing Study **total testosterone fell 0.4% and free testosterone 1.3% per year between ages 40 and 79**.
2. The fall in free testosterone is steeper because **SHBG rises with age**, binding more of the total hormone.
3. Ageing produces **reduced episodic and stimulated gonadotrophin secretion** together with **reduced Leydig-cell responsiveness to LH** — a mixed primary and secondary defect.

B. Definition and Diagnosis

4. **Hypogonadism** requires **both a subnormal serum testosterone and symptoms attributable to it** — a biochemical value alone is not a diagnosis.
5. Sample **between 07:00 and 11:00 hours, fasting, on two separate occasions**; measure **free or bioavailable testosterone** when SHBG is altered (obesity, ageing, liver disease, thyroid disease).
6. The **harmonised reference range for total testosterone is 264–916 ng/dL** in non-obese men aged 19–39; **age-adjusted reference ranges are not recommended**.
7. Follow-up tests: **LH and FSH** (to separate primary from secondary), **prolactin, iron studies, pituitary MRI** if secondary with very low testosterone, and a **karyotype** in young men with very small testes.

C. Indications and Contraindications

8. **Do not replace testosterone in asymptomatic older men with an age-related decline**; offer therapy on an **individualised basis after explicit discussion of risks and benefits** to symptomatic men with confirmed low levels.
9. **Absolute contraindications**: prostate carcinoma, breast cancer, prostate nodule or induration, unexplained PSA elevation, **haematocrit >50%**, unstable heart failure (NYHA III–IV), severe untreated sleep apnoea, and **severe LUTS with IPSS >19**.
10. Severe LUTS is a **relative contraindication that ceases to apply once the outflow obstruction is treated**.

D. Benefits and Expected Time Course

11. Testosterone increases **lean body mass, muscle strength, bone mineral density, erythropoiesis and libido**; in men it improves erectile strength, ejaculate volume and visuospatial cognition.
12. Time course: **libido, mood and energy improve within 3 months; fat mass, lean mass, insulin sensitivity and haemoglobin change at 3–6 months; BMD requires at least 1 year**.
13. The **Testosterone Trials (T Trials, 790 men aged >65)** showed **moderate benefit for sexual function, some benefit for mood and depressive symptoms, but no benefit for vitality or walking distance**.

E. Preparations, Targets and Monitoring

14. Available as **intramuscular esters (cypionate, enanthate — 2-weekly, often given weekly to reduce peaks and troughs), long-acting undecanoate, transdermal gels and patches, buccal, nasal gel and subcutaneous pellets**.
15. **Short-acting preparations are preferred initially**, so that therapy can be stopped quickly if adverse effects occur.
16. Target a **serum testosterone of 300–500 ng/dL in older men** — deliberately lower than the 500–600 ng/dL aimed for in the young — to minimise risk.
17. Check the level **within 3 months of starting**; for injections sample midway between doses, for transdermal gels about 6 hours after application.
18. Monitor **haematocrit, PSA and digital rectal examination at 3, 6 and 12 months, then annually**; the **major adverse effect is an excessive rise in haematocrit**. Other effects include gynaecomastia, fluid retention and transient worsening of sleep apnoea.
19. If symptoms do not improve, **stop treatment and investigate other causes**. **Selective androgen receptor modulators (SARMs)** are under development to avoid these adverse effects.

Latest Updates (2023–Present)

- **TRAVERSE trial (NEJM, 2023)** — 5,246 men aged 45–80 with hypogonadism and pre-existing or high cardiovascular risk. Testosterone gel was **non-inferior to placebo for major adverse cardiovascular events**, resolving a decade-old controversy.
- TRAVERSE nevertheless showed **significantly more atrial fibrillation (3.5% vs 2.4%), pulmonary embolism (0.9% vs 0.5%) and acute kidney injury (2.3% vs 1.5%)** in the testosterone arm — clinically important in older, comorbid men.
- **TRAVERSE Fracture subtrial (NEJM, 2024)** — testosterone **did not reduce, and numerically increased, clinical fractures** despite improving bone density in earlier studies. **Testosterone must not be prescribed for fracture prevention.**
- Testosterone **corrected anaemia** in hypogonadal men in a TRAVERSE substudy, and improved measures of glycaemia — potentially useful secondary benefits.
- The **European Expert Panel for Testosterone Research position statement (2025)** integrates TRAVERSE into practice, reaffirming that therapy is appropriate for **symptomatic, biochemically confirmed hypogonadism** with **structured haematocrit and PSA monitoring**, and is not a treatment for ageing.
- The **FDA labelling change (2025)** removed the broad cardiovascular warning while adding a **blood-pressure warning** for testosterone products.
- Emerging evidence links **weight loss (including with GLP-1 receptor agonists and bariatric surgery)** to

substantial rises in endogenous testosterone in obesity-related functional hypogonadism, supporting a **"treat the cause first"** approach.

Current Practice Update

Aspect	Textbook Position	Current Evidence / Guideline
Cardiovascular safety	Lack of long-term data; possible excess events in ≥65 in the first year	**TRAVERSE 2023: non-inferior for MACE** in high-risk men
Specific cardiac risks	Not quantified	**Increased atrial fibrillation, pulmonary embolism and AKI**
Bone/fracture	Increases BMD; fracture data unavailable	**TRAVERSE Fracture subtrial: no reduction, numerically more fractures**
Anaemia	Erythropoiesis noted as a benefit/risk	Formal evidence of **correction of unexplained anaemia**
Obesity-related low T	Comorbidity noted	**Weight loss first**; functional hypogonadism often reverses

Exam One-Liners

- **Symptoms + two low morning testosterone levels** = hypogonadism; a number alone is not.
- Reference range **264–916 ng/dL**; treat older men to **300–500 ng/dL**.
- **TRAVERSE: no excess MACE, but more AF, PE and AKI**.
- **Do not give testosterone to prevent fractures**.
- Monitor **haematocrit, PSA and DRE at 3, 6, 12 months then annually**.
- **IPSS >19, haematocrit >50%, prostate cancer** are contraindications.

Q6. Andropause

A. Terminology and Concept

1. Strictly, **"andropause" does not exist** — unlike the female menopause there is **no precipitous, universal loss of gonadal function** in men.
2. What occurs is a **gradual, variable decline in testosterone** with a continuously rising prevalence of low levels; this is termed **late-onset hypogonadism (LOH)**.
3. Even the term LOH is imperfect, because the decline is **continuous throughout adult life**, not confined to old age.
4. **LOH is defined as an age-associated clinical and biochemical syndrome** with symptoms and signs of hypogonadism plus serum testosterone below the young adult reference range.

B. Epidemiology

5. Although hypogonadal symptoms occur at least once in life in most men after age 50, the **prevalence of true LOH is only about 2–3%**.
6. Hypogonadism is **common but under-diagnosed and under-treated** in older men — only an estimated **5–35% of hypogonadal men receive treatment**.
7. Prevalence rises with **obesity, type 2 diabetes, metabolic syndrome, chronic illness, opioid use and glucocorticoid therapy** — i.e. it is largely **comorbidity-driven, not age-driven**.

C. Pathophysiology

8. A **combined primary and secondary defect**: reduced Leydig-cell responsiveness to LH (testicular) plus reduced episodic and stimulated gonadotrophin secretion (hypothalamo-pituitary).
9. **SHBG rises with age**, so free testosterone falls faster than total testosterone; measuring only total testosterone underestimates the deficit.
10. **Hypogonadotropic hypogonadism is frequent in type 2 diabetes and obesity**, mediated by insulin resistance, inflammation and aromatisation of androgen to oestrogen in adipose tissue — this form is often **reversible**.

D. Clinical Features

11. **Sexual symptoms are the most specific**: loss of morning erections, reduced sexual thoughts, and erectile dysfunction.

12. **Non-specific symptoms** overlap heavily with ageing and comorbidity: fatigue, low mood, poor concentration, reduced muscle mass and strength, increased visceral fat, decreased bone density, hot flushes, sweating and sleep disturbance.
13. Because of this overlap, **screening questionnaires (ADAM, AMS)** are sensitive but have **poor specificity** and are not recommended for case-finding.

E. Differential Diagnosis and Management

14. Exclude **depression, hypothyroidism, obstructive sleep apnoea, anaemia, chronic kidney or liver disease, malignancy, alcohol excess, opioids and glucocorticoids** before attributing symptoms to testosterone.
15. **Confirm biochemically** with two morning samples, plus LH/FSH, prolactin and (if indicated) pituitary imaging.
16. **First-line management is not hormonal**: weight reduction, exercise (especially resistance training), treatment of sleep apnoea and diabetes, and withdrawal of opioids or glucocorticoids where possible.
17. **Testosterone therapy is reserved for men with both low testosterone and clinically important symptoms**, after explicit discussion of risks and with the same monitoring and contraindications as any testosterone replacement.
18. Related age-related declines — **"adrenopause" (DHEA)** and **"somatopause" (GH/IGF-1)** — are similarly not indications for routine replacement; there is **no indication for testosterone treatment in the majority of adult men**.

Latest Updates (2023–Present)

- The concept of **functional versus organic hypogonadism** now dominates. **Functional hypogonadism** (obesity, diabetes, chronic illness, drugs) is potentially reversible and should be treated by correcting the cause; **organic hypogonadism** (structural hypothalamic, pituitary or testicular disease) requires replacement.
- **TRAVERSE (2023)** provided the reassurance that testosterone does not increase MACE in symptomatic hypogonadal men, but the **fracture subtrial (2024) and the excess of atrial fibrillation, pulmonary embolism and AKI** mean the risk–benefit conversation is now more nuanced, not less.
- The **European Expert Panel for Testosterone Research (2025)** and contemporary endocrine society positions maintain that **population screening for low testosterone in ageing men is not justified**.
- **Substantial weight loss** — through lifestyle, bariatric surgery or GLP-1 receptor agonists — reliably raises endogenous testosterone in obesity-related functional hypogonadism, and is now the recommended first intervention.
- Increasing concern about **direct-to-consumer "men's health" and anti-ageing clinics** prescribing testosterone without diagnostic confirmation; this is an important prescribing-safety and medico-legal issue in urban Indian practice as well.
- **Testosterone is being reframed as a quality-of-life drug**, not a disease-modifying or longevity therapy.

Current Practice Update

Aspect	Textbook Position	Current Evidence / Guideline
The term "andropause"	Rejected — no true male menopause	Unchanged; **LOH**, and increasingly **functional vs organic hypogonadism**
Cause of low testosterone in old age	Ageing plus comorbidity	Emphasis firmly on **comorbidity and obesity as the dominant reversible drivers**
First-line treatment	Testosterone if symptomatic and low	**Weight loss and treatment of comorbidity first**; testosterone if this fails
Cardiovascular safety	Uncertain, possible excess events	**TRAVERSE: MACE non-inferior**, but AF/PE/AKI signals
Screening	Not recommended	Reaffirmed — **no population screening**

Exam One-Liners

- **Andropause does not exist** — use **late-onset hypogonadism**.
- **Prevalence of true LOH ≈ 2–3%** despite symptoms being near-universal.
- **Sexual symptoms are the most specific**; ADAM and AMS lack specificity.
- **Free testosterone falls faster than total** because **SHBG rises**.
- **Functional hypogonadism is reversible** — treat obesity and diabetes first.
- **No indication for testosterone in most adult men.**

Q7. Alpha Receptor Antagonists

A. Classification

1. **Non-selective (α1 + α2)**: **phenoxybenzamine** (irreversible) and **phentolamine** (reversible) — used in phaeochromocytoma.
2. **Selective α1 (non-subtype-selective)**: **prazosin** (short-acting), **terazosin, doxazosin** (long-acting), **alfuzosin** (functionally uroselective).
3. **α1A-subtype selective ("uroselective")**: **tamsulosin, silodosin**; **naftopidil** is relatively α1D-selective.
4. **Selective α2 antagonists**: **yohimbine, idazoxan** — of historical/limited use.

B. Pharmacological Basis

5. The **bladder neck, prostatic stroma and prostatic urethra are rich in α1A receptors**; blockade relaxes smooth muscle, lowers tonic luminal pressure in the prostatic fossa and improves urine outflow.
6. **Vascular smooth muscle carries α1B receptors**; blocking these causes **vasodilatation, postural hypotension and reflex tachycardia** — the basis of most adverse effects.
7. **α1D receptors** in the bladder body and spinal cord may mediate storage symptoms such as urgency and nocturia.
8. Uroselective agents (tamsulosin, silodosin) act preferentially on α1A and therefore cause **less hypotension**, broadening the population of elderly men who can be treated safely.

C. Clinical Uses

9. **Lower urinary tract symptoms due to BPH** — the principal use, and an **excellent first-line option for men with bothersome symptoms who want treatment**. Alfuzosin, doxazosin, tamsulosin, terazosin and silodosin are all appropriate.
10. Alpha blockers give **rapid symptom relief within days**, but **do not alter the natural history of the disease** and **do not prevent long-term bladder outflow obstruction**.
11. **Combination with a 5α-reductase inhibitor** is appropriate when the prostate exceeds about 40 mL. In the **MTOPS study**, combination therapy reduced acute urinary retention by **66%**, versus **39% for alpha blockade alone and 34% for finasteride alone**.
12. After **6–9 months of successful combination therapy the alpha blocker may be withdrawn**, restarting it if symptoms recur.
13. Other uses: **phaeochromocytoma** (phenoxybenzamine pre-operatively), **resistant hypertension** (as add-on only), **medical expulsive therapy for distal ureteric stones**, **prazosin for PTSD-related nightmares**, and **scorpion envenomation (prazosin)** — relevant in Indian practice.
14. Alpha blockers have **not been shown to reduce symptoms of chronic prostatitis/chronic pelvic pain syndrome**, and increased dizziness and postural hypotension in trials.

D. Adverse Effects — Particular Relevance in the Elderly

15. **First-dose and postural hypotension, dizziness, syncope, asthenia, headache and rhinitis** occur in about **5–9%**; these led to **falls, fractures and institutionalisation** in older men in observational studies.
16. Early agents were designed as antihypertensives, so **orthostasis was a significant problem**; normotensive men also complained of asthenia and fatigue. Dose titration at bedtime reduces this.
17. **Intraoperative floppy iris syndrome (IFIS)** — iris billowing, prolapse and progressive miosis during cataract surgery. **Tamsulosin carries by far the highest risk**; stopping the drug does not reliably prevent it, so the **ophthalmologist must be informed before surgery**.
18. **Retrograde or absent ejaculation** is common, especially with **silodosin and tamsulosin** — a frequently under-discussed quality-of-life issue.
19. **Interactions**: additive hypotension with **PDE5 inhibitors, nitrates, diuretics and other antihypertensives**; caution in patients with **autonomic failure or Parkinson's disease**.
20. **Beers criteria advise against using peripheral α1 blockers as routine antihypertensives in older adults** because of orthostatic hypotension; in **ALLHAT the doxazosin arm was terminated early** for excess heart failure.

Latest Updates (2023–Present)

- **AUA LUTS/BPH Guideline (2023 amendment; comprehensive three-part guideline 2026)** retains alpha blockers as **first-line medical therapy**, but places far greater emphasis on **shared decision-making about sexual side-effects** and on **minimally invasive surgical therapies (MIST)** — prostatic urethral lift, water vapour thermal therapy, Aquablation, prostatic artery embolisation — for men who wish to avoid drug side-effects or preserve ejaculatory function.
- **Network meta-analysis and FAERS pharmacovigilance data (2022–2025)** confirm the IFIS hierarchy: **tamsulosin has a markedly higher risk than terazosin, alfuzosin or doxazosin**, with silodosin showing less mesopic pupil

constriction. Documentation of alpha blocker use is now a **standard pre-cataract-surgery checklist item**.

- **Observational cohort studies (2023–2025)** have reported associations between tamsulosin and **dementia, Parkinson's disease and depression** compared with other alpha blockers. These are **hypothesis-generating and confounded by indication**; no causal link is established, and guidelines have not changed prescribing.
- Conversely, some pharmaco-epidemiological work has suggested **terazosin and doxazosin may be neuroprotective** via PGK1-mediated enhancement of glycolysis — again unproven.
- **Medical expulsive therapy**: current urological guidance restricts alpha blockers to **distal ureteric stones larger than about 5 mm**, following the negative SUSPEND trial.
- Deprescribing literature increasingly identifies alpha blockers as **high-value targets for review in older fallers**, alongside sedatives and antihypertensives.

Current Practice Update

Aspect	Textbook Position	Current Evidence / Guideline
Place in BPH therapy	First-line for bothersome LUTS	Unchanged, but **MIST offered earlier** for men prioritising sexual function
IFIS	Recognised association	**Risk hierarchy quantified**; tamsulosin far highest; mandatory pre-operative disclosure
Neuropsychiatric safety	Not addressed	Observational **dementia/Parkinson signals with tamsulosin — unproven, no change in practice**
Use as antihypertensive	Historical indication	**Avoid as routine antihypertensive** in older adults (Beers, ALLHAT)
Ureteric stones	Adjunct use	Restricted to **distal stones >5 mm**
Chronic pelvic pain	Sometimes used	**Not recommended** — no benefit, more adverse effects

Exam One-Liners

- **α1A = prostate, α1B = vasculature, α1D = bladder body** — the basis of uroselectivity.
- **Tamsulosin and silodosin** are uroselective; **prazosin, terazosin, doxazosin** are not.
- **MTOPS: combination therapy reduced acute retention by 66%**.
- **Tamsulosin is the classic cause of intraoperative floppy iris syndrome**.
- **Silodosin causes the most retrograde ejaculation**.
- **Do not use alpha blockers as first-line antihypertensives in older adults** (Beers/ALLHAT).

Q8. Hyponatremia

A. Definition and Significance

1. **Hyponatraemia is a serum sodium below 135 mEq/L** and is the commonest electrolyte disorder in hospitalised patients, associated with increased morbidity, mortality, length of stay and cost.
2. Severity: **mild 130–134 mEq/L, moderate 125–129 mEq/L, profound/severe below 125 mEq/L**; by duration, **acute (<48 hours)** or **chronic**.
3. **Older patients are more susceptible and may show cognitive impairment even with mild hyponatraemia** — a fall in sodium, especially if rapid, can cause cerebral oedema, lethargy, seizures, coma and death.

B. Why the Elderly Are Predisposed

4. **Age-related tubular changes**: reduced sodium reabsorption in the thick ascending limb producing "senile sodium leakage", medullary hypotonicity, and inability to concentrate urine maximally.
5. **Reduced plasma renin and aldosterone** with blunted responses, plus **elevated natriuretic peptides**, enhance urinary sodium loss.
6. **Exaggerated vasopressin response to a hyperosmolar stimulus** with impaired baroreceptor-mediated suppression — older people over-secrete ADH.
7. Reduced renal blood flow and reduced ability to **excrete a water load**; salt-restricted diets and poor appetite compound the problem.

C. Causes in Older People

8. **Drugs are the leading cause** — **thiazide diuretics** (classic), SSRIs, SNRIs, tricyclics, carbamazepine and oxcarbazepine, antipsychotics, PPIs, NSAIDs, desmopressin, chlorpropamide, vincristine, ifosfamide, cyclophosphamide and opioids.
9. **SIADH** — from CNS disease (stroke, haemorrhage, infection, metastases), pulmonary disease (pneumonia, tuberculosis, tumour), and **small-cell carcinoma of the lung** as a paraneoplastic syndrome.
10. **Hypovolaemic**: vomiting, diarrhoea, diuretics, third-space losses, cerebral salt wasting, mineralocorticoid deficiency.
11. **Hypervolaemic**: heart failure, cirrhosis, nephrotic syndrome, advanced CKD.
12. **Endocrine**: hypothyroidism, glucocorticoid deficiency and **abrupt withdrawal of glucocorticoid therapy**.
13. **Low solute intake**: "tea-and-toast" diet and beer potomania — very relevant to socially isolated older Indians on rice-and-tea diets.
14. **Pseudohyponatraemia** (hyperlipidaemia, paraproteinaemia) and **translocational hyponatraemia** (hyperglycaemia, mannitol) must be excluded first.

D. Investigation

15. **Step 1: serum osmolality** — exclude pseudo- and translocational hyponatraemia. **Step 2: urine osmolality** — <100 mOsm/kg suggests primary polydipsia or low solute intake. **Step 3: urine sodium** — <20 mEq/L suggests hypovolaemia or oedematous states; >30–40 mEq/L suggests SIADH, diuretics or adrenal insufficiency. **Step 4: clinical volume status.**
16. **Diagnostic criteria for SIADH**: sodium <135 mEq/L, **plasma osmolality <280 mOsm/kg, urine osmolality inappropriately high (>500 mOsm/kg), urine sodium >20 mEq/L**, clinical euvolaemia, and normal renal, adrenal and thyroid function.
17. Additional tests: **thyroid function, 9 a.m. cortisol or short Synacthen test, blood urea and uric acid** (low uric acid supports SIADH), chest imaging and CT head if a cause is not evident.

E. Management

18. **Severe symptoms (seizures, coma, respiratory arrest): 3% hypertonic saline 100–150 mL as an intravenous bolus**, repeated up to three times as needed, aiming for a **rapid initial rise of 4–6 mEq/L** to relieve cerebral oedema.
19. **Correction limits are critical: no more than 8–10 mEq/L in 24 hours and 18 mEq/L in 48 hours**, and no more than **8 mEq/L/24 h in high-risk patients** (alcoholism, malnutrition, hypokalaemia, liver disease, sodium <105 mEq/L) — to prevent **osmotic demyelination syndrome**.
20. **Chronic asymptomatic hyponatraemia**: treat the cause, **stop the offending drug**, and use **fluid restriction (typically 500–1000 mL/day)** with adequate salt and protein intake.
21. **Second-line agents**: **oral urea 15–60 g/day** (effective, safe, cheap), **loop diuretic with oral salt**, **demeclocycline**, and **tolvaptan** in euvolaemic or hypervolaemic SIADH with high ADH activity.
22. If **over-correction occurs, re-lower the sodium** with 5% dextrose with or without **desmopressin**; monitor sodium 2–4 hourly during active correction.
23. Long-term consequences even of mild chronic hyponatraemia include **gait instability, attention deficits, falls, osteoporosis and fractures** — an important geriatric syndrome, not a laboratory curiosity.

Latest Updates (2023–Present)

- **"Hyponatraemia — Treatment Standard 2024" (Nephrology Dialysis Transplantation)** is the current reference standard. It endorses **3% saline as a 100–150 mL bolus or continuous infusion**, with the choice determined by symptom severity and persistence.
- The same standard states that **rapid intermittent bolus administration is preferred** for symptomatic hyponatraemia, based on the **SALSA randomised trial**, which found bolus and continuous infusion equally effective and safe, with **bolus therapy requiring less therapeutic re-lowering** and being simpler to administer (no calculation needed).
- **Fixed-dose bolus regimens can over- or under-correct**, particularly in **low-body-weight and low-BMI patients** — a group that includes many frail older Indian patients; weight-based adjustment and frequent monitoring are advised.
- **Oral urea** has re-emerged as a highly effective and safe second-line therapy for SIADH, now favoured over demeclocycline and, in many settings, over vaptans on grounds of cost and safety.
- **Peripheral administration of 3% saline** through a large-bore cannula is now accepted as safe in most settings, removing the delay caused by waiting for central access.
- **SGLT2 inhibitors (empagliflozin) have shown benefit in SIADH** in randomised work, by inducing osmotic diuresis — a promising but not yet standard option.
- Current guidance emphasises **gradual correction with clinical evaluation rather than rapid normalisation to the laboratory reference range**, and readiness to give hypotonic fluid or desmopressin if correction runs ahead.

Current Practice Update

Aspect	Textbook Position	Current Evidence / Guideline
Method of hypertonic saline	Infusion with furosemide and saline replacement	**Rapid intermittent 100–150 mL boluses of 3% saline preferred** (SALSA)
Correction limit	Not exceeding ~20 mEq/L over 48 hours	**≤8–10 mEq/L/24 h and ≤18 mEq/L/48 h**; ≤8 if high ODS risk
Second-line therapy	Demeclocycline for resistant SIADH	**Oral urea** favoured; demeclocycline largely superseded
Vaptans	Not established	Defined role in **euvolaemic/hypervolaemic high-ADH states**, with protocolised monitoring
Over-correction	Avoid	**Active re-lowering** with 5% dextrose ± desmopressin is standard rescue
Mild chronic hyponatraemia	Often observed	Treated because of **falls, fractures, gait and cognitive effects**

Exam One-Liners

- **Sodium <135 mEq/L**; profound below **125 mEq/L**.
- **Thiazides and SSRIs** are the commonest drug causes in the elderly.
- **SIADH: euvolaemic, urine osmolality >500 mOsm/kg, urine sodium >20 mEq/L, low uric acid**.
- **3% saline 100–150 mL bolus** for seizures or coma; aim for a **4–6 mEq/L rise**.
- **Never exceed 8–10 mEq/L in 24 hours** — osmotic demyelination.
- **Oral urea** is the modern second-line agent for chronic SIADH.

Q9. Hyperkalemia

A. Definition and Ageing Physiology

1. **Hyperkalaemia is a serum potassium above 5.0–5.5 mEq/L**; it is life-threatening above **6.5 mEq/L** or with any ECG change.
2. **Total body potassium is lower in the old** because of reduced muscle mass and poor dietary intake, so a normal or high serum level may coexist with total-body depletion.
3. **Renal potassium excretion is significantly reduced in ageing**, explained by **reduced serum aldosterone and reduced tubular responsiveness to aldosterone** (hyporeninaemic hypoaldosteronism), plus increased activity of the H+/K+-ATPase pump in the collecting duct.
4. Reduced GFR with age means that any additional potassium load is poorly handled — the elderly kidney has **little reserve**.

B. Causes

5. **Drugs are the dominant cause**: ACE inhibitors, ARBs, **mineralocorticoid receptor antagonists (spironolactone, eplerenone)**, potassium-sparing diuretics, **NSAIDs, trimethoprim/co-trimoxazole, heparin, beta-blockers, digoxin toxicity, calcineurin inhibitors**, and potassium supplements.
6. **Renal**: acute kidney injury, chronic kidney disease (especially stage 4–5), obstructive uropathy, and type 4 renal tubular acidosis (classically in diabetic nephropathy).
7. **Transcellular shift**: metabolic acidosis, insulin deficiency and hyperglycaemia, beta-blockade, rhabdomyolysis, tumour lysis syndrome, massive haemolysis, and suxamethonium.
8. **Endocrine**: adrenal insufficiency, isolated hypoaldosteronism.
9. **Dietary/iatrogenic**: **potassium-based "low-sodium" salt substitutes** (increasingly used in India for hypertension), coconut water, banana, citrus, stored blood transfusion.
10. **Pseudohyperkalaemia** — haemolysed sample, prolonged tourniquet, fist clenching, marked leucocytosis or thrombocytosis — must be excluded before treatment.

C. Clinical Features and ECG

11. Symptoms are **non-specific**: muscle weakness, flaccid paralysis, paraesthesiae, palpitations, and sometimes cardiac arrest as the first manifestation.
12. **Progressive ECG sequence**: **tall peaked T waves → flattened P waves with PR prolongation → widening QRS →**

sine wave pattern → asystole or ventricular fibrillation.

13. The ECG is **insensitive** — a normal ECG does not exclude dangerous hyperkalaemia, and the rate of rise matters as much as the absolute value.

D. Emergency Management (the "5-step" approach)

14. **1. Protect the myocardium**: **10 mL of 10% calcium gluconate intravenously over 2–5 minutes**, repeated if ECG changes persist. Onset is immediate but lasts only 30–60 minutes; use calcium chloride via a central line if severe. Use with great caution in digoxin toxicity.
15. **2. Shift potassium into cells**: **10 units of soluble insulin in 25 g of dextrose (50 mL of 50% or 125 mL of 20%)** intravenously over 15–30 minutes, plus **nebulised salbutamol 10–20 mg**. **Sodium bicarbonate** only if there is significant metabolic acidosis.
16. **3. Remove potassium from the body**: **loop diuretic with volume repletion**, **potassium binders**, and **haemodialysis** for severe or refractory hyperkalaemia, oliguric AKI or ESRD.
17. **4. Stop the source**: discontinue causative drugs, potassium supplements and salt substitutes; restrict dietary potassium.
18. **5. Monitor**: continuous ECG, and repeat potassium at 1, 2, 4 and 6 hours — **rebound hyperkalaemia is common** once the insulin effect wanes.
19. **Hypoglycaemia after insulin–dextrose is the commonest iatrogenic complication**, occurring in up to a fifth of patients — particularly in the elderly, in CKD, and in those not eating; monitor capillary glucose hourly for at least 6 hours.

E. Chronic Management

20. Long-term management aims to **allow continuation of prognostically important RAAS inhibitors** rather than stopping them: dietary counselling, correction of metabolic acidosis with **sodium bicarbonate**, use of loop or thiazide diuretics, and **potassium binders**.
21. **Sodium polystyrene sulfonate** is slow, unpalatable and carries a warning for **serious gastrointestinal necrosis**, especially with sorbitol — its role is now limited.

Latest Updates (2023–Present)

- **New potassium binders** have transformed chronic management. **Patiromer** (exchanges potassium for calcium in the distal colon) and **sodium zirconium cyclosilicate (SZC)** (exchanges sodium and hydrogen for potassium, onset within 1 hour) both give rapid and sustained potassium reduction with good tolerability.
- **KDIGO 2024 CKD guideline** and **ESC heart failure and ESH hypertension guidelines** recommend potassium binders specifically **to enable optimisation and continuation of RAAS inhibitor and MRA therapy** rather than dose reduction or withdrawal.
- The **DIAMOND trial** showed patiromer enabled more patients with heart failure to remain on target-dose RAAS inhibition; **AMBER** showed the same for spironolactone in resistant hypertension with CKD; SZC has similar data for spironolactone optimisation in heart failure (JACC, 2025).
- Consensus recommendations (2023–2025) stress a **proactive, long-term-condition model**: check potassium before starting a RAAS inhibitor and regularly thereafter, and **treat the hyperkalaemia before reducing the RAAS inhibitor**, because RAAS inhibitor withdrawal itself worsens cardiovascular and renal outcomes and mortality.
- **Lower-dose insulin regimens (0.1 units/kg, maximum 10 units)** are increasingly used in place of a fixed 10-unit dose to reduce hypoglycaemia in elderly patients and those with CKD, with comparable potassium lowering.
- Emergency-department use of oral potassium binders is being studied as a way to **avoid admission** in stable hyperkalaemic patients; real-world data (2025) suggest more patients can be discharged directly.
- **Public health caution**: potassium-enriched salt substitutes reduce stroke at population level, but pose a **real hyperkalaemia hazard in older adults with CKD, diabetes or on RAAS inhibitors** — patients must be asked specifically about them.

Current Practice Update

Aspect	Textbook Position	Current Evidence / Guideline
Chronic binder therapy	Sodium polystyrene sulfonate	**Patiromer and SZC** preferred; SPS carries GI necrosis warning
RAAS inhibitors after hyperkalaemia	Reduce or stop	**Treat the potassium and continue the RAAS inhibitor** (KDIGO 2024, ESC)
Insulin dose for shift	10 units with dextrose	**0.1 units/kg (max 10 units)** to reduce hypoglycaemia in elderly/CKD
Post-treatment monitoring	Repeat potassium	**Mandatory hourly capillary glucose for ≥6 hours** — hypoglycaemia is common
Salt substitutes	Not addressed	Recognised **modern cause** of hyperkalaemia in older adults

Exam One-Liners

- **Ageing kidney = hyporeninaemic hypoaldosteronism** → poor potassium excretion.
- **Calcium gluconate stabilises the myocardium but does not lower potassium**.
- **Insulin–dextrose plus nebulised salbutamol** shift; **diuretics, binders and dialysis** remove.
- **A normal ECG does not exclude dangerous hyperkalaemia.**
- **Patiromer and SZC allow RAAS inhibitors to be continued** — treat the potassium, not the prescription.
- Always ask about **"low-sodium" salt substitutes**.

Q10. Management of Recurrent Urinary Tract Infection in the Elderly

A. Definition

1. **Recurrent UTI is ≥2 culture-proven symptomatic episodes in 6 months or ≥3 in 12 months.**
2. Distinguish **relapse** (same organism within 2 weeks — suggests an unresolved focus such as prostatitis, stone or abscess) from **reinfection** (different organism or later — suggests host susceptibility).
3. UTI is **the commonest infection in the elderly** and is especially frequent in debilitated and institutionalised individuals.

B. Why Recurrence Occurs in Older People

4. **Obstructive uropathy** is the strongest pathogenetic association — **benign prostatic hyperplasia in men**, and pelvic organ prolapse or cystocele in women.
5. **Non-obstructive predisposing factors**: **vaginal and urethral atrophy from oestrogen deficiency**, **"puddling" of urine from bed rest and immobility**, and **bladder catheterisation**.
6. **Incomplete bladder emptying with a raised post-void residual**, detrusor underactivity, constipation and faecal incontinence.
7. **Diabetes, immunosenescence, functional dependence, cognitive impairment and previous antibiotic exposure** with resistant flora.
8. In men, **chronic bacterial prostatitis** must be excluded — it classically presents as recurrent UTI with the same organism in the absence of catheterisation.

C. Evaluation

9. **Confirm the diagnosis**: symptomatic episodes with a **positive urine culture** on each occasion — do not diagnose recurrent UTI on urinalysis or on asymptomatic positive cultures.
10. **Review all previous cultures and sensitivities** to guide empirical therapy and to detect ESBL or carbapenem-resistant organisms.
11. **Post-void residual by bladder ultrasound**; **ultrasound of the kidneys, ureters and bladder** for stones, hydronephrosis and prostatic size.
12. **Cystoscopy and upper-tract imaging** if there is **visible or persistent microscopic haematuria, suspected malignancy, obstruction, stone, or persistent infection with a urea-splitting organism**.
13. Assess and treat the **whole geriatric context**: hydration, mobility, toileting access, constipation, incontinence, atrophic vaginitis, glycaemic control and nutrition.

D. Non-Antibiotic Prevention (First Line)

14. **Fluid intake**: increase to at least **1.5 litres per day** where intake is low — a simple measure with randomised

evidence.

15. **Topical vaginal oestrogen** in postmenopausal women restores lactobacilli and vaginal pH and significantly reduces recurrence; **oral oestrogen is not effective and is not recommended**.
16. **Behavioural and hygiene measures**: complete bladder emptying, double voiding, post-coital voiding, avoiding spermicides, treating constipation, and timed toileting in the cognitively impaired.
17. **Methenamine hippurate 1 g twice daily** — converted to formaldehyde in acidic urine; a urinary antiseptic that **does not select for resistance**.
18. **Cranberry products and D-mannose** may be offered, with a clear explanation of modest and inconsistent evidence; **probiotics** have weak supporting data.
19. **Remove or avoid catheters** — this is often the single most effective intervention in the institutionalised patient.

E. Antibiotic Strategies and Stewardship

20. **Antibiotic prophylaxis** is reserved for those failing non-antibiotic measures: **continuous low-dose nightly** (nitrofurantoin, co-trimoxazole, cefalexin), **post-coital single dose**, or **patient-initiated self-start therapy** in cognitively intact women.
21. Prophylaxis should be **time-limited (typically 3–6 months) and reviewed**, with awareness that **benefit is lost once it stops**. **Nitrofurantoin should be avoided in eGFR <45 mL/min** and with long-term use because of pulmonary and hepatic toxicity.
22. **Do not treat asymptomatic bacteriuria**, and **do not perform test-of-cure cultures** in asymptomatic patients — both drive resistance and *Clostridioides difficile* infection without benefit.

Latest Updates (2023–Present)

- **AUA/CUA/SUFU Recurrent Uncomplicated UTI Guideline — 2025 amendment** significantly expands **non-antibiotic prophylaxis**: clinicians may now offer **cranberry, methenamine hippurate, and increased water intake (where intake is below 1.5 L/day)**. It states that **D-mannose alone may not be effective**, and that clinicians should **not perform post-treatment test-of-cure urinalysis or culture in asymptomatic patients**.
- **NICE NG112 (updated December 2024)** now recommends **methenamine hippurate as an alternative to daily antibiotic prophylaxis** in women who have failed behavioural measures, vaginal oestrogen and single-dose prophylaxis.
- The **ALTAR trial (BMJ, 2022)** established **non-inferiority of methenamine hippurate to daily antibiotic prophylaxis** — the pivotal study behind this shift.
- **ImpresU trial (Clinical Microbiology and Infection, 2025)** — the first randomised trial specifically in **women aged ≥70** — found methenamine reduced antibiotic treatments by about **25% during 6 months of treatment**, but with a **rebound increase in infections after stopping**, indicating suppression rather than cure and the need to plan duration and follow-up.
- **Sublingual bacterial vaccines (MV140/Uromune)** have randomised evidence for reducing recurrence over 9–12 months and are available in several countries; **long-term data in frail older adults are still lacking**.
- **Gepotidacin**, a first-in-class triazaacenaphthylene, was approved in 2025 for uncomplicated UTI and is active against many resistant uropathogens — an important future option given Indian resistance rates.
- Emerging emphasis on the **"continuum of UTI"** concept, recognising that many older adults sit between asymptomatic bacteriuria and clear-cut infection, and require observation rather than reflex antibiotics.
- **Indian context**: very high rates of **ESBL-producing *E. coli* and fluoroquinolone resistance** mean that **culture-directed therapy is mandatory**, and **fosfomycin and nitrofurantoin** retain the best oral activity for lower tract disease.

Current Practice Update

Aspect	Textbook Position	Current Evidence / Guideline
First-line prophylaxis	Antibiotic prophylaxis after evaluation	**Non-antibiotic measures first** — fluids, vaginal oestrogen, methenamine, cranberry
Methenamine hippurate	Minor role	**Non-inferior to antibiotic prophylaxis** (ALTAR); recommended by NICE 2024 and AUA 2025
Evidence in the very old	Extrapolated	**ImpresU (2025)** provides direct evidence in women ≥70, with rebound after cessation
D-mannose	Promising adjunct	**May not be effective alone** (AUA 2025)
Oestrogen	Vaginal or systemic considered	**Vaginal only**; oral oestrogen not recommended
Test of cure	Often performed	**Should not be performed** in asymptomatic patients

Exam One-Liners

- **≥2 in 6 months or ≥3 in 12 months**, each culture-proven and symptomatic.
- **Relapse = same organism <2 weeks** — look for prostate, stone or abscess.
- **Vaginal (not oral) oestrogen** is a cornerstone in postmenopausal women.
- **Methenamine hippurate is non-inferior to antibiotic prophylaxis** (ALTAR) and does not select resistance.
- **ImpresU: 25% fewer antibiotic courses in women ≥70, but rebound on stopping.**
- **Never treat asymptomatic bacteriuria; never do a test of cure.**

Q11. Asymptomatic Bacteriuria — Definition, Treatment and Prognosis

A. Definition

1. **Asymptomatic bacteriuria (ASB) is significant bacteriuria in the absence of signs or symptoms referable to the urinary tract.**
2. **Microbiological criteria**: in **women**, ≥10^5 CFU/mL of the same organism in **two consecutive** clean-catch voided specimens; in **men**, ≥10^5 CFU/mL in **one** clean-catch specimen; in **either sex**, ≥10^2 CFU/mL from a single catheterised specimen.
3. **Pyuria is not part of the definition** and does **not** convert ASB into infection — most bacteriuric older adults have pyuria.
4. ASB must be distinguished from UTI, which requires **localising genitourinary symptoms or systemic features of infection** in addition to bacteriuria.

B. Epidemiology

5. **Bacteriuria in older persons is common and usually asymptomatic**, and its prevalence **increases with advancing age**.
6. Approximate prevalence: **community-dwelling women over 70 — 10–20%; older men — up to 10%; long-term care facility women — 25–50%; men — 15–40%; patients with a long-term indwelling catheter — essentially 100%**.
7. Predisposing factors are the same as for UTI: **vaginal and urethral atrophy, urinary puddling from bed rest, bladder catheterisation, obstructive uropathy and diabetes**.
8. Prevalence of ASB **roughly parallels the prevalence of urinary incontinence**, so incontinent geriatric patients commonly have significant bacteriuria.
9. Common organisms are **E. coli, Klebsiella, Proteus, Enterococcus** and, in catheterised patients, **polymicrobial and frequently changing flora**.

C. Treatment — When Not To, and When To

10. **In the absence of obstructive uropathy, there is no evidence to support routine antimicrobial therapy for ASB in older persons.**
11. **Do not screen for or treat ASB** in: healthy non-pregnant women, older men and women in the community, **residents of long-term care facilities**, patients with diabetes, patients with spinal cord injury, and patients with indwelling catheters.
12. **Do not treat ASB in an older patient with delirium or a fall but no fever, no haemodynamic instability and no**

localising urinary symptoms — assess for other causes and observe carefully.

13. **Treat ASB only in**: **pregnancy**, and **before urological procedures likely to cause mucosal bleeding** (e.g. TURP, ureteroscopy with stone manipulation), where treatment is given shortly before the procedure.
14. Renal transplant recipients should **not** be routinely treated for ASB beyond the **first month** after transplantation.
15. One exception in the incontinence pathway: in an **incontinent patient with new or worsening incontinence**, bacteriuria may be treated once as part of the initial assessment; however, **eradicating bacteriuria does not affect chronic stable incontinence** in nursing home residents.
16. In patients with an indwelling catheter, **do not perform surveillance cultures** to guide management, because all chronically catheterised patients are bacteriuric, often polymicrobially, and the organisms change frequently.

D. Prognosis

17. **The prognosis of ASB is benign.** Studies suggesting increased mortality did not adjust for age and comorbidity; when adjusted, the excess mortality is explained by the **fatal underlying diseases associated with bacteriuria**, not by the bacteriuria itself.
18. ASB **does not cause progressive renal damage, hypertension or chronic kidney disease** in the non-obstructed urinary tract.
19. **Treatment does not improve survival, does not reduce symptomatic infection and does not improve continence**; earlier studies in hospitalised and institutionalised patients showed treatment was ineffective because of the **very high rate of recurrence**.
20. **Harms of treating ASB are real**: antimicrobial adverse effects, drug interactions, ***Clostridioides difficile*** **infection**, selection of multidrug-resistant organisms, and cost. There is also evidence that **stable bacteriuria may be protective against symptomatic reinfection**, so eradication can be counterproductive.

Latest Updates (2023–Present)

- The **IDSA 2019 Guideline on Asymptomatic Bacteriuria** remains the operative standard and is unchanged in principle; it explicitly recommends **against screening or treating ASB in older residents of long-term care facilities** and in functionally impaired older adults.
- IDSA specifically addresses the geriatric dilemma: in older patients with **functional or cognitive impairment and bacteriuria with delirium but without fever or haemodynamic instability**, the recommendation is **assessment for other causes and careful observation rather than antimicrobial treatment**. Observational data show **no association between bacteriuria and mental status change** after adjustment, and no difference in IL-6 between bacteriuric residents with and without non-specific symptoms.
- **Diagnostic stewardship** is now the central strategy: reduce **unnecessary urine testing**. Tools include the **Loeb minimum criteria** for ordering urine cultures in nursing home residents, the **McGeer surveillance criteria**, and the **AHRQ decision aid** for suspected UTI in long-term care (separate pathways for catheterised and non-catheterised residents).
- A urinalysis **negative for both nitrites and leucocyte esterase essentially rules out acute cystitis** — a useful rule-out, never a rule-in.
- The **"continuum of UTI" framework (2023–2024)** proposes moving beyond the rigid ASB/UTI dichotomy, acknowledging that older and catheterised patients often present without localising symptoms, and supporting **watchful waiting with re-assessment** as a legitimate management category.
- The **Inappropriate Diagnosis of UTI (ID-UTI) measure** is now used as a quality metric, and stewardship programmes using it have reduced ASB treatment even in small critical-access hospitals.
- Despite two decades of guidance, **35–93% of nursing home residents with ASB still receive inappropriate antibiotics** — closing this gap is a defined geriatric quality-improvement priority.

Current Practice Update

Aspect	Textbook Position	Current Evidence / Guideline
Screening in the elderly	Not supported	**Explicitly recommended against** (IDSA), including long-term care
Delirium or falls with bacteriuria	Bacteriuria may be treated in new incontinence	**Do not treat** on delirium/falls alone without fever or instability — observe and seek other causes
Catheterised patients	No surveillance cultures	Unchanged and reinforced
Strategy	Avoid unnecessary treatment	**Avoid unnecessary testing** — diagnostic stewardship (Loeb, McGeer, AHRQ tools)
Conceptual model	ASB versus UTI	**"Continuum of UTI"** with watchful waiting as a defined option
Renal transplant	Not specified	**No treatment beyond the first month** post-transplant

Exam One-Liners

- **Two consecutive positive cultures in women, one in men, ≥10^2 CFU/mL from a catheter specimen.**
- **Pyuria does not make bacteriuria an infection.**
- **Treat only in pregnancy and before mucosa-breaching urological procedures.**
- **Delirium plus bacteriuria without fever is not a UTI** — look elsewhere.
- **Prognosis is benign; excess mortality is due to comorbidity, not bacteriuria.**
- **The best way to avoid treating ASB is not to send the urine culture** — diagnostic stewardship.

Q12. Urinary Tract Infection in the Elderly

A. Epidemiology and Definitions

1. **UTI is the most common infection in the elderly**, and is especially frequent in **debilitated and institutionalised individuals**; it is the commonest source of bacteraemia in older adults.
2. **Uncomplicated UTI** occurs in a structurally and functionally normal tract; **complicated UTI** occurs with obstruction, stones, catheters, neurogenic bladder, renal impairment, immunosuppression or male sex — **most UTI in older people is complicated**.
3. **Catheter-associated UTI (CA-UTI)** and **urosepsis** are the two forms with the greatest mortality.

B. Predisposing Factors

4. **Obstructive uropathy** is the key mechanism — **prostatic hypertrophy in men**, prolapse and cystocele in women, stones and strictures in both.
5. **Non-obstructive factors**: **vaginal and urethral atrophy, puddling of urine from bed rest, bladder catheterisation**, incomplete emptying with a raised post-void residual, and faecal incontinence.
6. **Host factors**: diabetes, immunosenescence with reduced cell-mediated and humoral immunity, malnutrition, dementia and functional dependence.
7. Phagocytic function and the complement system appear **unaltered** in ageing; the defect is largely mechanical and mucosal rather than immunological.

C. Microbiology

8. **E. coli remains the commonest organism**, but the **frequency of Gram-negative bacilli other than E. coli increases with age** — Proteus, Klebsiella, Pseudomonas, Serratia — along with **Enterococcus** and, in catheterised patients, polymicrobial flora.
9. **Proteus mirabilis** is urease-producing and causes **alkaline urine, struvite stones and catheter encrustation**.
10. If **Staphylococcus aureus** is grown from urine, **take blood cultures** — it usually reflects bacteraemic seeding of the kidney rather than ascending infection.
11. In India, **ESBL-producing Enterobacterales are widespread**, with high resistance to fluoroquinolones and third-generation cephalosporins, and rising carbapenem resistance in hospital-acquired infection.

D. Clinical Presentation

12. **Classical dysuria, frequency and urgency may be absent**; presentation is often atypical — **confusion or delirium, functional decline, falls, anorexia, incontinence, or unexplained fever**.

13. **Fever may be blunted or absent**; conversely, in the very old a **febrile response is more likely to indicate bacterial rather than viral illness**. **Absence of leucocytosis does not exclude bacterial infection.**
14. **Acute unexplained functional deterioration should alert the physician to a potential acute infectious process.**
15. Nevertheless, **non-specific symptoms alone should not be attributed to UTI** unless other causes have been excluded — this is the central diagnostic discipline in geriatric practice.
16. **Acute pyelonephritis** presents with fever, rigors, loin pain and vomiting; in the elderly it may present simply as collapse or sepsis.

E. Diagnosis and Management

17. **Send a properly collected specimen**: clean-catch midstream, or a fresh condom-catheter specimen in incontinent men after cleaning; catheterised patients should have the sample taken from a **freshly changed catheter**, never from the drainage bag.
18. **Dipstick negative for both nitrites and leucocyte esterase makes cystitis very unlikely**; a positive dipstick alone does not establish infection in an older patient.
19. **Treatment principles**: choose according to **the organism isolated and local resistance patterns**; empirical cover should account for the higher frequency of Gram-negative organisms, and **renal function must be considered and monitored**, particularly with aminoglycosides.
20. **Typical regimens**: **nitrofurantoin 100 mg twice daily for 5–7 days** (avoid if eGFR <45 mL/min) or **fosfomycin 3 g single dose** for lower UTI in women; **7 days for men**; **7–14 days for pyelonephritis** with an aminoglycoside or third-generation cephalosporin initially, stepped down by culture; **carbapenem or a beta-lactam/beta-lactamase inhibitor** for ESBL infection.
21. **Fluoroquinolones should be avoided in older adults where possible** — tendon rupture, aortic aneurysm, QT prolongation, delirium, hypoglycaemia and *C. difficile*.
22. **Supportive care**: hydration, review of drugs, treatment of constipation, catheter removal, delirium prevention, and early mobilisation. Investigate for **obstruction, stone or abscess** if fever persists beyond 72 hours.
23. **Prevention**: adequate fluid intake, vaginal oestrogen, avoidance of unnecessary catheterisation, prompt catheter removal, bladder emptying strategies, and **not treating asymptomatic bacteriuria**.

Latest Updates (2023–Present)

- **EAU Urological Infections Guideline (2023–2025)** and **IDSA guidance** now foreground **antimicrobial stewardship**: culture-directed narrow-spectrum therapy, **shortest effective duration**, and avoidance of fluoroquinolones as first-line agents.
- **Diagnostic stewardship** tools (**Loeb minimum criteria, McGeer criteria, AHRQ decision aid**) are recommended in long-term care to decide **when to send urine**, not merely when to treat.
- The **"continuum of UTI" concept (2023–2024)** formally accommodates the older patient with bacteriuria and non-localising symptoms, encouraging **short-term observation and reassessment** in stable patients.
- **New antimicrobials for resistant uropathogens**: **gepotidacin** (approved 2025 for uncomplicated UTI, a novel oral class), and for complicated UTI **ceftazidime-avibactam, ceftolozane-tazobactam, meropenem-vaborbactam, imipenem-relebactam, plazomicin, cefiderocol** and **cefepime-taniborbactam** — most relevant to Indian tertiary practice with high ESBL and carbapenemase burden.
- **Nitrofurantoin** has been re-evaluated as **safe down to an eGFR of about 30–45 mL/min for short courses**, widening its use in older women, though long-term prophylaxis remains restricted.
- Randomised evidence continues to support **shorter courses**: 5 days of nitrofurantoin for cystitis, 7 days for febrile UTI in men where source control is achieved.
- **Indian data** consistently show **>50% fluoroquinolone resistance and 40–60% ESBL rates** in community *E. coli* isolates, making **empirical fluoroquinolone or cephalosporin use inappropriate**; nitrofurantoin and fosfomycin remain the reliable oral options.

Current Practice Update

Aspect	Textbook Position	Current Evidence / Guideline
Empirical therapy	Third-generation cephalosporin and/or aminoglycoside	**Culture-directed, narrow-spectrum**; fluoroquinolones and cephalosporins unreliable in India
Nitrofurantoin in renal impairment	Avoided in renal impairment	**Acceptable for short courses down to eGFR ~30–45 mL/min**
Duration	Conventional courses	**Shorter courses** — 5 days cystitis, 7 days in men, 7–14 days pyelonephritis
Non-specific symptoms	Atypical presentation is typical	Balanced by **diagnostic stewardship** — do not over-attribute delirium/falls to UTI
Resistant organisms	Not addressed	New agents — **gepotidacin**, and beta-lactam/beta-lactamase inhibitor combinations
Deciding to test	Send urine if UTI suspected	**Loeb/McGeer/AHRQ criteria** determine when to send urine at all

Exam One-Liners

- **UTI is the commonest infection in the elderly**; **obstructive uropathy** is the key mechanism.
- **E. coli commonest**, but **non-E. coli Gram-negatives rise with age**.
- **A urine growing *S. aureus* demands blood cultures.**
- **Negative nitrite plus negative leucocyte esterase virtually excludes cystitis.**
- **Avoid fluoroquinolones** in older adults; **nitrofurantoin and fosfomycin** are the workhorses.
- **Atypical presentation is the rule — but do not blame every delirium on the urine.**

Q13. Behavioural Intervention in Urinary Incontinence

A. Concept and Place in Management

1. **Behavioural interventions are the recommended first-line treatment for geriatric urinary incontinence**, because they are **non-invasive, free of drug side-effects and non-specific** — patients with stress and urgency incontinence respond equally well.
2. They are the **only treatment modality applicable to every type** of incontinence, and can be combined with drugs or surgery.
3. The essential classification is into **patient-dependent** procedures (requiring adequate function, learning capability and motivation, aiming to **restore normal voiding and continence**) and **caregiver-dependent** procedures (for functionally disabled patients, aiming to **keep the patient and environment dry**).
4. All patient-dependent procedures involve **continuous self-monitoring with a bladder record**; caregiver-dependent procedures use a staff-completed bladder and bowel record.

B. Non-Specific Supportive Measures

5. **Education**, modification of **fluid and caffeine intake** (aim for **1.5–2 litres of non-caffeinated fluid per day**), and review of medications, discontinuing diuretics where possible.
6. Environmental manipulation — **accessible toilets, commodes, urinals, adequate lighting, clothing that is easy to remove, walking aids** — plus appropriate garments and pads.
7. Address **reversible contributors**: constipation and straining, obesity, chronic cough, atrophic vaginitis (**long-term vaginal oestrogen** where irritative symptoms predominate), delirium, and restricted mobility.

C. Patient-Dependent Interventions

8. **Pelvic floor muscle (Kegel) exercises** are the essential component and produce **the greatest benefit** in treating urinary incontinence. They consist of repetitive contraction and relaxation of the pelvic floor muscles.
9. Technique: teach by asking the patient to interrupt voiding, or by squeezing the examiner’s fingers on vaginal examination — **without a Valsalva manoeuvre, which is the opposite of the intended effect**.
10. A full exercise is a **10-second squeeze and a 10-second relaxation**, built up gradually, practised up to **40 times a day**, and — crucially — **used in real life at moments that provoke leakage** (the “knack”: contracting before coughing, standing or hearing running water).
11. **Women over 75 usually require biofeedback** (vaginal or rectal pressure or EMG, with or without abdominal EMG) to identify the correct muscles; **older men also respond** to pelvic muscle and bladder training for overactive bladder

and urgency incontinence.

12. **Bladder training** combines education, bladder records, pelvic muscle exercises and **urge-suppression strategies**; it is **highly effective in motivated, functional community-dwelling patients**.
13. **Bladder retraining** progressively lengthens or shortens the inter-voiding interval, with intermittent catheterisation where there is persistent retention — used chiefly **after removal of an indwelling catheter** or after an overdistension injury.
14. A bladder retraining protocol after catheter removal: remove the catheter (clamping first is unnecessary), treat any infection, then toilet the patient **on waking, 2-hourly by day and evening, before bed and 4-hourly at night**, with monitoring of continent voids, incontinent episodes, fluid intake and post-void residual. If difficulty persists beyond **1–2 weeks**, seek reversible causes and consider urological referral.
15. **Trigger and emptying techniques** — running tap water, stroking the inner thigh, suprapubic tapping to trigger voiding; **bending forward, suprapubic pressure and double voiding** to complete emptying.

D. Caregiver-Dependent Interventions

16. **Scheduled (timed) toileting** — routine toileting at fixed intervals, usually **2-hourly during the day and 4-hourly at night**; usable in cognitive or physical impairment; the goal is to **prevent wetting episodes**.
17. **Habit training** — a **variable** toileting schedule individualised to the patient's own voiding pattern as shown by the bladder record.
18. **Prompted voiding** — the best studied of these procedures: contact the resident approximately **2-hourly by day, focus attention on voiding by asking whether they are wet or dry, check and give feedback, offer toileting, give social reinforcement for continence and for appropriate requests, and offer fluids routinely**.
19. **Up to 25–40% of nursing home residents respond well to prompted voiding during the day**, and responders can be identified by a **3-day trial**, which improves cost-effectiveness. Night-time toileting should be individualised against **sleep disruption and fall risk**.
20. Success of caregiver-dependent programmes depends **more on the knowledge and motivation of the staff or caregiver** than on the patient's physical or mental status — so **staff training, supervision and quality-improvement methods are integral** to the intervention.

Latest Updates (2023–Present)

- **Cochrane review (2018) and subsequent updates** confirm that women with stress urinary incontinence undertaking pelvic floor muscle training are about **eight times more likely to report cure or improvement** than untreated or placebo groups; **motivation and compliance are the key determinants of success**.
- **AUA/SUFU Non-Neurogenic Overactive Bladder Guideline (2024)** and **NICE NG123** both place **behavioural therapy as first-line for all patients**, with drugs as add-on rather than replacement; **combination of behavioural therapy with pharmacotherapy is superior to either alone**.
- Anticholinergic burden is now a major driver of practice: given the association between **long-term anticholinergic exposure and dementia**, guidelines recommend **beta-3 agonists (mirabegron, vibegron) over antimuscarinics in older adults**, and behavioural therapy as the way to minimise or avoid drug therapy altogether. Baseline renal function and blood pressure should be checked before a beta-3 agonist.
- **Functional Incidental Training (FIT)** — combining prompted voiding with strengthening, walking and transfer practice during routine toileting — improves both continence and mobility in nursing home residents, and is now the preferred model in long-term care.
- **Digital and app-supported pelvic floor muscle training**, telehealth-delivered programmes and **home biofeedback devices** have randomised evidence of efficacy and are increasingly used, though uptake in older cohorts depends on digital literacy and carer support.
- Growing evidence supports **weight reduction** as an effective behavioural intervention for stress incontinence in overweight women, and **treatment of constipation** as a modifiable factor for all types.
- **Indian context**: continence care is largely nurse- and family-delivered; **NPHCE-supported geriatric clinics** and **ASHA/ANM-based training** in toileting programmes are the most scalable route, with pads and garments a significant out-of-pocket cost.

Current Practice Update

Aspect	Textbook Position	Current Evidence / Guideline
Place in therapy	First-line, non-invasive	Unchanged and strengthened; **combination with drug therapy superior to either alone**
Drug therapy alongside	Antimuscarinics for urgency	**Beta-3 agonists preferred** in older adults due to anticholinergic-dementia concerns
Delivery of PFMT	Clinic teaching plus biofeedback	**Digital, app-based and telehealth PFMT** with randomised support
Nursing home programmes	Prompted voiding	**Functional Incidental Training** — continence plus mobility gains
Lifestyle	Fluids, caffeine, constipation	**Weight reduction** added as an evidence-based intervention

Exam One-Liners

- **Patient-dependent = restore continence; caregiver-dependent = keep the patient dry.**
- **Pelvic floor exercises give the greatest single benefit**; teach without Valsalva.
- **10-second squeeze, 10-second relax, up to 40 a day, plus "the knack".**
- **Prompted voiding: 2-hourly, social reinforcement, 25–40% respond, identified by a 3-day trial.**
- **Success depends on caregiver motivation more than on patient function.**
- **Behavioural therapy + drug is better than either alone**; prefer **beta-3 agonists** in the elderly.

Q14. Continuous Bladder Drainage / Long-Term Catheterization

A. Definitions and Types

1. **Long-term (chronic) catheterisation** is an indwelling catheter in place for **30 days or more**; short-term is under 30 days.
2. Three basic types of catheter are used in incontinence management: **external (condom) catheters, intermittent straight catheterisation, and chronic indwelling catheterisation** (urethral or suprapubic).
3. **Catheters should be avoided in the management of incontinence unless specific indications are present**, and should never be used for convenience, because they foster dependency.

B. Indications for a Chronic Indwelling Catheter

4. **Urinary retention** that is causing **persistent overflow incontinence, symptomatic infection or renal dysfunction**, and which **cannot be corrected surgically or medically** and **cannot be practically managed by intermittent catheterisation**.
5. **Skin wounds, pressure injuries or irritation being contaminated by incontinent urine.**
6. **Care of terminally ill or severely impaired patients** for whom bed and clothing changes are uncomfortable or disruptive.
7. **Patient preference**, where toileting or changing causes excessive discomfort.
8. Short-term additional indications include **accurate measurement of urine output in acute illness** and **perioperative use for defined procedures** — but the catheter must be removed as soon as the indication ends.

C. Key Principles of Catheter Care

9. **Maintain a sterile, closed gravity-drainage system and never break the closed system**; keep the bag below bladder level and off the floor.
10. **Secure the catheter to the upper thigh or lower abdomen** to prevent perineal contamination and urethral traction injury from catheter movement.
11. **Avoid frequent vigorous cleaning of the catheter entry site — washing with soap and water once a day is sufficient**; use clean technique and hand hygiene when emptying or changing the drainage system.
12. **Do not routinely irrigate.** If bypassing occurs in the absence of obstruction, consider **bladder spasm**, treatable with a bladder relaxant.
13. If **obstruction occurs frequently, increase fluid intake**; catheter blockage is usually due to **encrustation with struvite from urease-producing *Proteus mirabilis***, and the catheter change interval should be planned ahead of the predictable blockage time.
14. **Do not use prophylactic or suppressive urinary antiseptics or antimicrobials routinely.**

15. **Do not perform surveillance cultures** to guide management, because all chronically catheterised patients have bacteriuria, often polymicrobial, and the organisms change frequently.
16. **Do not treat infection unless the patient develops symptoms**; symptoms may be non-specific, so **other sources of infection must be carefully excluded first**.
17. If a patient develops **frequent symptomatic infections, arrange genitourinary evaluation** to exclude stones, periurethral or prostatic abscess and chronic pyelonephritis.

D. Complications

18. **Chronic bacteriuria is universal**; further complications include **catheter-associated UTI and urosepsis, bladder and renal stones, periurethral and prostatic abscess, epididymo-orchitis, urethral trauma, erosion and false passage, catheter bypassing and blockage, haematuria, and — with long-term use — squamous cell carcinoma of the bladder**.
19. **Nursing home residents, especially men, managed by chronic catheterisation are at relatively high risk of symptomatic infection.** Indwelling catheterisation remains **overused in acute hospitals**.
20. **Non-infectious harms** matter as much: pain, restricted mobility and rehabilitation, falls over tubing, loss of dignity, and being a “one-point restraint”.

E. Practical Alternatives and Removal

21. **Intermittent catheterisation** (self- or carer-performed, 2–4 times daily, keeping bladder volume under about 400 mL) is preferred where feasible; the catheter should be **clean but need not be sterile at home**. In nursing homes, anatomical abnormalities and resistant institutional flora may make repeated straight catheterisation impractical.
22. **External (condom) catheters** should be used **only for intractable incontinence in men without retention who are extremely physically dependent**; they still increase the risk of symptomatic infection and skin damage.
23. **Suprapubic catheterisation** avoids urethral erosion, is more comfortable, permits sexual activity, and is easier to change — preferred when long-term drainage is genuinely unavoidable.
24. **Plan removal actively**: use a **trial without catheter (TWOC)** with a bladder retraining protocol; **most indwelling catheters in acute hospitals and many in long-term care can be removed** this way.

Latest Updates (2023–Present)

- **“Strategies to Prevent Catheter-Associated Urinary Tract Infections in Acute-Care Hospitals: 2022 Update”** (SHEA with IDSA, APIC, AHA and The Joint Commission, published 2023) is the current compendium standard.
- It introduces the **“Disrupting the Life Cycle of the Indwelling Urethral Catheter”** model, which identifies **alternatives before insertion, safe insertion and maintenance, and prompts for timely removal** as the three intervention points.
- **Non-catheter strategies** are now formally listed as the first step: **prompt toileting, urinals, bedside commodes, incontinence garments**, and **intermittent straight catheterisation**.
- The update adds **urine-culture stewardship** as an essential practice — reducing inappropriate urine testing is now part of CAUTI prevention, not separate from it.
- Performance measures have been broadened from CAUTI rates alone to include **catheter harm** — trauma, obstruction and non-infectious complications.
- The update explicitly discusses the **limitations of external urinary catheters**, including non-catheter-associated UTIs arising from alternative collection devices, and skin injury — cautioning against simply substituting one device for another.
- **Unresolved issues** flagged include the **standard of care for routine replacement of catheters in place beyond 30 days**, reflecting continuing uncertainty about optimal change intervals.
- **Antimicrobial-impregnated and silver-alloy catheters have not shown clinically important benefit** in large trials and are not recommended routinely; **catheter valves** may improve bladder capacity and dignity in selected patients.

Current Practice Update

Aspect	Textbook Position	Current Evidence / Guideline
Framing of prevention	Correct indications and good catheter care	**"Disrupting the life cycle"** — alternatives, insertion, maintenance, removal
Testing	Do not perform surveillance cultures	Broadened to **urine-culture stewardship** as an essential practice
Outcome measured	CAUTI rate	**CAUTI plus catheter harm** (trauma, obstruction)
External catheters	Acceptable alternative in dependent men	**Limitations emphasised** — device-associated UTI and skin injury
Coated catheters	Not addressed	**No routine benefit** from antimicrobial or silver-alloy catheters
Replacement interval	Change when blocked	**Explicitly unresolved** beyond 30 days; individualise to blockage pattern

Exam One-Liners

- **Long-term = 30 days or more.**
- Indications: **uncorrectable retention, wound contamination, terminal care, patient preference.**
- **Closed drainage, secured catheter, no routine irrigation, no prophylactic antibiotics, no surveillance cultures.**
- **All chronically catheterised patients are bacteriuric — treat only symptoms.**
- **Proteus mirabilis** causes encrustation and blockage.
- **The best catheter care is not having a catheter** — offer toileting, commodes and intermittent catheterisation first.

Q15. Preliminary Assessment of an Older Male with Lower Urinary Tract Symptoms, and When to Refer to a Urologist

A. Background

1. **Benign prostatic hyperplasia is inexorably linked with ageing** and is a major contributing factor in the development of **lower urinary tract symptoms (LUTS)**; the prevalence of BPH-related symptoms may be as high as **50%** in multinational surveys.
2. LUTS are **not synonymous with BPH** — they may arise from the bladder (overactivity or underactivity), prostate, urethra, or from systemic disease and drugs.
3. **Voiding (obstructive) symptoms**: hesitancy, weak stream, straining, intermittency, terminal dribbling, incomplete emptying. **Storage (irritative) symptoms**: frequency, urgency, nocturia, urgency incontinence. **Post-micturition**: post-void dribbling.

B. History

4. Use the **International Prostate Symptom Score (IPSS) / AUA Symptom Index** — seven items scored 0–5: **0–7 mild, 8–19 moderate, 20–35 severe** — plus the **disease-specific quality-of-life ("bother") question**.
5. The score quantifies symptoms over time; **bother and quality of life, not the score alone, drive treatment decisions**.
6. A **3-day frequency-volume chart (bladder diary)** distinguishes polyuria, nocturnal polyuria and reduced functional bladder capacity — critical in the older man with nocturia.
7. Exclude other causes by history: **type 2 diabetes** (nocturia with polyuria >3 litres), **neurological disease** (neurogenic bladder), **gross haematuria or bladder pain** (tumour or calculi), **urethral trauma, urethritis or instrumentation** (stricture), **family history of BPH or prostate cancer**, and **sexual dysfunction-related LUTS**.
8. **Drug history is essential** — anticholinergics impair bladder function, sympathomimetics increase outflow resistance, diuretics increase frequency, opioids cause retention, and calcium-channel blockers worsen emptying.
9. Assess **fluid, caffeine and alcohol intake, and evening fluid timing**.

C. Examination

10. **Digital rectal examination** to assess prostate size and consistency and to detect **nodules or induration suggesting carcinoma**; also assess **rectal sphincter tone**, and exclude faecal impaction.
11. **Abdominal examination for a palpable bladder** (chronic retention), and examination of the **external genitalia** for meatal stenosis and phimosis.

12. **Focused neurological examination**, including perineal sensation and sacral reflexes.
13. **A general health and cognitive status evaluation is useful for choosing the best treatment, especially if a surgical procedure may be required** — frailty, cognition and life expectancy determine what is appropriate. **Dementia and other neurological conditions can themselves affect the urinary condition.**

D. Investigations

14. **Urinalysis** in all patients, to detect **infection and haematuria**; **urine cytology** is helpful when irritative symptoms or haematuria predominate, especially in smokers, to exclude bladder tumour.
15. **Serum creatinine is not routinely indicated** in the initial evaluation of LUTS attributable to BPH, since baseline renal impairment is no commoner in men with BPH than in age-matched peers — but measure it if there is chronic retention, hydronephrosis, or suspicion of obstructive nephropathy.
16. **PSA remains controversial but is still recommended after counselling**, and is chiefly appropriate where **life expectancy exceeds 10 years** and a diagnosis of cancer would change management. Remember: **PSA is not cancer-specific and rises in BPH, prostatitis, instrumentation and retention**, and **about 25% of men with prostate cancer have a PSA of 4.0 ng/mL or less**.
17. **Post-void residual urine** by bladder ultrasound and **uroflowmetry** are optional but useful; **a poor maximum flow rate, high post-void residual and greater symptom severity predict clinical progression**.
18. **Do not perform routine upper-tract imaging, cystoscopy or urodynamics** at the preliminary assessment.

E. When to Refer to a Urologist

19. **Refer urgently** for: **visible (gross) haematuria or persistent microscopic haematuria** without infection; **abnormal DRE (nodule or induration)** or **unexplained PSA elevation**; and **suspected malignancy**.
20. **Refer for**: **acute or chronic urinary retention with a palpable bladder**, **renal impairment or hydronephrosis attributable to obstruction**, **bladder or urethral stones**, **suspected urethral stricture** (previous trauma, urethritis or instrumentation), and **recurrent urinary tract infection or persistent sterile pyuria**.
21. **Refer for**: **failure of adequate medical therapy**, **severe bothersome symptoms in a man fit for and wishing to consider intervention**, **suspected neurogenic bladder**, and **any patient in whom surgery is being contemplated** (complex urodynamic testing is essential before surgical intervention and to define the cause of retention).
22. **Manage in primary care / general geriatric clinic**: mild symptoms (IPSS <7) with **watchful waiting and lifestyle measures** — fluid restriction before bedtime, avoiding caffeine and spicy food, monitoring diuretics, timed voiding and bladder retraining, pelvic floor exercises, and treating constipation. Patients with mild symptoms but **severe bother should undergo further assessment**.

Latest Updates (2023–Present)

- The **AUA LUTS/BPH Guideline (2023 amendment, expanded into a three-part guideline in 2026)** is the current reference. Initial workup remains **history, AUA-SI/IPSS and urinalysis**, with explicit elicitation of **patient goals** and discussion of side-effects.
- A major new emphasis is **pre-operative assessment of prostate size and shape** (ultrasound, cystoscopy or existing cross-sectional imaging) because **modality selection now depends on volume** — for example, water vapour thermal therapy is offered where prostate volume is **under 80 g**, with counselling on retreatment rates.
- **Minimally invasive surgical therapies (MIST) — prostatic urethral lift, water vapour thermal therapy, Aquablation, prostatic artery embolisation, temporary implantable nitinol device** — are now firmly in the guideline and are particularly attractive in **older, frailer men and those wishing to preserve ejaculatory function**. Enucleation techniques (**HoLEP, ThuLEP**) are size-independent options for very large glands.
- Guidelines now explicitly require **counselling about sexual side-effects** of medical and surgical therapy, following evidence of poor pre-treatment counselling in practice.
- **Deprescribing is part of the assessment**: reviewing **anticholinergics, opioids, diuretics and sympathomimetics** frequently improves symptoms without any new prescription.
- **Nocturia** is increasingly managed as a distinct multifactorial syndrome — sleep apnoea, heart failure, oedema, diabetes, drugs and nocturnal polyuria — rather than as a prostate symptom; desmopressin carries a **significant hyponatraemia risk in the elderly and requires sodium monitoring**.
- **PSA screening**: contemporary guidance supports **shared decision-making in men with >10 years' life expectancy** and generally **against PSA testing above age 70 or in significant frailty**, aligning with geriatric principles of avoiding diagnoses that cannot benefit the patient.

Current Practice Update

Aspect	Textbook Position	Current Evidence / Guideline
Core initial workup	History, IPSS, examination, urinalysis	Unchanged, plus explicit **elicitation of patient goals**
Prostate size assessment	Not routine at first visit	**Recommended before procedural therapy** — determines modality choice
Treatment options	Watchful waiting, drugs, MIST, surgery	Greatly expanded **MIST menu**; enucleation for large glands
Sexual side-effects	Mentioned	**Mandatory counselling** in guideline statements
Nocturia	Part of the symptom score	Managed as a **separate multifactorial syndrome**
PSA	Recommended with counselling	**Shared decision-making**; generally avoided above 70 or in frailty

Exam One-Liners

- **IPSS: 0–7 mild, 8–19 moderate, 20–35 severe** — plus the bother question.
- **A 3-day bladder diary distinguishes nocturnal polyuria from a prostate problem.**
- **Urinalysis in everyone; creatinine not routine; PSA only after counselling.**
- **Red flags for referral: haematuria, abnormal DRE, retention, renal impairment, stones, stricture, recurrent UTI, failed medical therapy.**
- **25% of prostate cancers have a PSA ≤4.0 ng/mL.**
- **Review the drug chart before writing a new prescription.**

Q16. Peritoneal Dialysis

A. Principle

1. **Peritoneal dialysis (PD)** uses the **peritoneum as a semipermeable membrane**: solutes are removed by **diffusion** down a concentration gradient into the dialysate, and water by **osmosis (ultrafiltration)** driven by an osmotic agent.
2. The effective exchange surface is the **peritoneal capillary endothelium**, described by the **three-pore model** — large pores, small pores and aquaporin-1 water channels (responsible for sodium sieving).
3. Access is by a **Tenckhoff catheter** placed surgically, laparoscopically or percutaneously, ideally **2 weeks before starting** to allow the exit site to heal.

B. Solutions and Modalities

4. **Glucose-based solutions (1.5%, 2.5%, 4.25%)** are standard; **icodextrin** (a glucose polymer) gives sustained ultrafiltration over a long dwell and is ideal for the overnight or day-long dwell; **amino-acid solutions** address protein loss.
5. **Low-glucose-degradation-product, neutral-pH, bicarbonate-buffered solutions** are more biocompatible and better preserve the membrane and residual renal function.
6. **CAPD (continuous ambulatory PD)** — typically **four exchanges of 2 litres daily**, performed manually by the patient or carer.
7. **APD (automated PD)** by a cycler at night — including **CCPD (continuous cycling PD)**, **NIPD (nightly intermittent PD)** and **TPD (tidal PD)**. **Nightly PD can be an alternative for more vulnerable elderly patients, including nursing home residents.**
8. **Peritoneal equilibration test (PET)** classifies membrane transport as **high, high-average, low-average or low**, guiding dwell time: high transporters need **short dwells (APD)**, low transporters need **long dwells (CAPD)**.
9. **Adequacy** is assessed by symptoms, volume status and nutrition, with a **weekly Kt/V urea of at least 1.7** as the conventional minimum; **preservation of residual renal function is a priority**.

C. Advantages, Particularly in Older Patients

10. **PD is a satisfactory alternative for geriatric ESRD patients, and most studies confirm that elderly patients on PD and HD have similar survival.**
11. **PD is better than HD for patients with residual diuresis, severe hypotension, complicated or short-lived vascular access, intradialytic arrhythmias, angina, or cardiovascular instability.**
12. Additional advantages: **home-based therapy** with independence and no travel, **gentle continuous solute and fluid**

removal without haemodynamic swings, **better preservation of residual renal function**, **no vascular access or systemic anticoagulation**, a **more liberal diet and fluid allowance**, and better preservation of anaemia control.

13. In India, PD avoids **repeated travel to a dialysis centre**, is feasible in rural areas, and is supported by dedicated government guidelines — important where transport and caregiver time are major barriers.

D. Problems and Complications in Older Patients

14. **The most frequent practical problems in older patients on PD are dialysate leakage, hernia formation, backache, difficulty in learning the technique, social isolation, family burn-out, and worsening of peripheral vascular disease or malnutrition.**
15. **Peritonitis** is the major infective complication — cloudy effluent with abdominal pain, effluent white cell count >100/µL with >50% neutrophils; treat with **intraperitoneal antibiotics** covering Gram-positive and Gram-negative organisms; recurrent, refractory or fungal peritonitis requires **catheter removal**.
16. **Exit-site and tunnel infection**, catheter malfunction and migration, constipation-related drainage failure.
17. **Metabolic**: glucose absorption causing hyperglycaemia, weight gain and dyslipidaemia; **protein loss of 5–15 g per day** in the effluent.
18. **Encapsulating peritoneal sclerosis** — a rare but devastating complication of long-duration PD.
19. **Nutrition**: malnutrition is frequently present; provide **more than 1.2 g/kg body weight of protein daily for patients on PD** (versus more than 1 g/kg for HD). Older patients on dialysis develop **more serious bone disease** than the young because of osteopenia, unbalanced diet, reduced physical activity and lack of sun exposure.
20. **Relative contraindications**: extensive abdominal adhesions or previous major abdominal surgery, active inflammatory bowel disease or diverticulitis, large irreparable hernia, stomas, severe respiratory disease, and — critically in geriatrics — **inability of the patient or carer to perform exchanges safely**, which is the commonest practical barrier.

Latest Updates (2023–Present)

- **Assisted peritoneal dialysis (aPD)** is now the key strategy for extending PD to frail older adults: a trained nurse, care assistant or family carer performs or supervises exchanges. It substantially widens eligibility and has become standard practice in several European systems and in Indian PD programmes.
- **ISPD guidance on "high-quality, goal-directed PD prescription"** has replaced rigid small-solute clearance targets with a **person-centred approach** prioritising **symptom control, volume status, quality of life and preservation of residual kidney function** over achieving a numerical Kt/V — a philosophy that fits geriatric practice precisely.
- **Incremental PD** — starting with fewer or smaller exchanges while residual renal function persists, then escalating — reduces treatment burden, glucose exposure and cost, and is particularly suited to older patients.
- **Urgent-start PD** (beginning within about 2 weeks of catheter insertion with low-volume supine exchanges) allows PD to be used for unplanned dialysis starts, avoiding a temporary central venous catheter and its infective risk.
- **ISPD peritonitis guidelines (2022)** emphasise **prevention** (exit-site antibiotic prophylaxis, prophylaxis before colonoscopy and dental procedures, treatment of constipation), **avoidance of prolonged empirical vancomycin and aminoglycosides**, and centre-level peritonitis rate targets.
- **Remote patient monitoring of APD cyclers** enables early detection of non-adherence, drainage problems and volume issues — valuable for older patients living alone or at a distance from the unit.
- **India**: the **Pradhan Mantri National Dialysis Programme (PMNDP)**, launched in 2016 under the National Health Mission, now includes a **dedicated peritoneal dialysis component** with operational guidelines issued to states, alongside free haemodialysis for below-poverty-line patients across all States/UTs and over 750 districts. The **PMNDP portal** supports a renal registry and "one nation–one dialysis" portability. PD is explicitly promoted as a **home-based option** for patients unable to travel, with **assisted PD by a family carer** recognised in the national guidance.

Current Practice Update

Aspect	Textbook Position	Current Evidence / Guideline
Adequacy target	Weekly Kt/V thresholds	**Goal-directed, person-centred prescription** (ISPD) — symptoms and volume over numbers
Eligibility of the frail elderly	Learning difficulty is a barrier	**Assisted PD** by nurse or family carer overcomes this
Starting prescription	Full four-exchange CAPD	**Incremental PD** while residual function persists
Unplanned start	Requires temporary HD catheter	**Urgent-start PD** now feasible and preferred
Solutions	Glucose-based, icodextrin	**Low-GDP, neutral-pH biocompatible solutions** to preserve membrane and residual function
Monitoring	Clinic review	**Remote monitoring of cyclers**
India	Not addressed	**PMNDP peritoneal dialysis component** with state operational guidelines

Exam One-Liners

- **Diffusion removes solute; osmosis (ultrafiltration) removes water.**
- **PET defines transport status: high transporters → short dwells (APD); low transporters → long dwells (CAPD).**
- **PD and HD give similar survival in the elderly.**
- **PD is preferred with residual diuresis, poor vascular access, hypotension, angina or arrhythmias.**
- **Protein requirement: >1.2 g/kg/day on PD, >1 g/kg/day on HD.**
- **Assisted PD and incremental PD are the two ideas that make PD a geriatric therapy.**

Q17. Role of Renal Replacement Therapy in the Elderly

A. Background

1. **Chronic kidney disease is predominantly a disease of older adults** — its incidence rises steadily with age and it is at least **10 times commoner at 75 and over than at 15–45**.
2. The commonest causes of end-stage renal disease in old age are **hypertension, nephrosclerosis, diabetes mellitus and obstructive uropathy**; in up to **one-third of cases no specific cause is identifiable**.
3. **Age itself does not constitute a contraindication to dialysis or transplantation.**
4. When **GFR falls below about 10 mL/min** with uraemic features, replacement therapy becomes necessary — but the modern approach is to start on **symptoms and clinical state rather than on an eGFR threshold alone**.

B. The Four Modalities

5. **Haemodialysis (HD)** — in-centre, satellite or home; requires vascular access and haemodynamic tolerance.
6. **Peritoneal dialysis (PD)** — home-based, continuous, gentler; **elderly patients on PD and HD have similar survival**.
7. **Kidney transplantation** — the best modality for survival and quality of life in selected older patients.
8. **Conservative kidney management (CKM)** — active, planned, non-dialytic care focused on symptom control, quality of life and advance care planning; **not "no treatment"**.

C. Haemodialysis in the Elderly

9. **Practical problems during HD in the aged**: **difficulty creating an arteriovenous fistula, permanent catheter or graft infection, gastrointestinal bleeding, intradialytic hypotension, headaches, vomiting, cramps, angina, arrhythmias and cardiovascular instability.**
10. **Older patients are more vulnerable to dialysis-related complications** — haemodynamic instability, bleeding and disequilibrium syndrome.
11. **Malnutrition is frequent**; provide **more than 1 g/kg body weight of protein daily on HD**. Bone disease is more severe than in the young.
12. **Very elderly patients on haemodialysis have a poor life expectancy: median survival is 15.6 months from age 80–84 and 11.6 months from age 85–89.**

13. **The leading causes of death in the oldest dialysis patients are elective withdrawal (38%), cardiovascular events (24%) and infection (22%)** — withdrawal being the single commonest, which makes **advance care planning essential from the outset**.

D. Predictors, Transplantation and Decision-Making

14. **Predictors of poor outcome on dialysis in older adults are: poor nutritional status, dementia, gait disorders, impaired activities of daily living and instrumental ADLs, and late referral to nephrology services.**
15. **Comprehensive geriatric assessment — not chronological age — should drive the decision**; frailty, cognition, functional status and social support predict outcome better than any laboratory value.
16. **Transplantation**: with improved patient selection, newer immunosuppressants and lower corticosteroid doses, **one-year patient and graft survival of 85% and 75%** is achievable; for patients over 60–65 the **five-year functional graft survival is 55–60%**.
17. Older recipients have **fewer rejection episodes**; **graft loss is mainly due to death with a functioning graft**, and the commonest causes of death are **cardiovascular disease and infection related to peaks of immunosuppression**.
18. **Donor age is the greatest adverse factor in cadaveric transplantation**; kidneys from older donors are "marginal" but can be used, singly or as a pair, in a subgroup of older recipients.
19. **Drug dosing must be individualised** — bioavailability, volume of distribution, renal excretion and half-life all change with age; use **low, slowly increasing doses and avoid polypharmacy**.

E. Conservative Kidney Management and Shared Decision-Making

20. **CKM is a legitimate, active pathway** comprising **symptom control (pruritus, nausea, restless legs, breathlessness, pain), anaemia and acidosis management, blood-pressure and volume control, dietary support, advance care planning and palliative care linkage**.
21. **The survival advantage of dialysis is attenuated in the oldest and frailest patients**; survival is broadly similar regardless of modality among those **aged 80 and over with high comorbidity, particularly ischaemic heart disease**, while dialysis imposes far greater treatment burden.
22. Use structured aids for prognostication and communication — the **"surprise question"** ("would I be surprised if this patient died within a year?"), validated mortality prediction models, and frailty measures — and frame the discussion around **quality of life, hospital-free days and patient goals**, not survival alone.
23. **Withdrawal from dialysis** should be discussed openly and revisited; it is a recognised, ethical decision and the commonest immediate cause of death in the very old on dialysis.

Latest Updates (2023–Present)

- **KDIGO 2024 CKD Guideline** formally states that **conservative kidney care is a reasonable option** for patients who do not wish to pursue kidney replacement therapy, embedding CKM into mainstream nephrology rather than treating it as a failure of care.
- A **target trial emulation study (Annals of Internal Medicine, 2024)** in older adults with kidney failure compared starting dialysis with continuing medical management. Starting dialysis conferred a **modest survival gain but fewer days at home** — the concept of **"home time"** as a patient-centred outcome is now central to the conversation.
- Contemporary cohort data confirm the age gradient: five-year survival from first nephrology attendance falls from about **84% under 50 years to 40% at 65–79 and 22% at 80 and over**; and a substantial minority of patients who opt for dialysis **die before ever reaching it**.
- Recent European cohort work (2026) reports median survival from the treatment decision of about **53 months for dialysis, 32 months for CKM in those considered suitable for dialysis, and 18 months for CKM in those less suitable** — with the difference narrowing markedly above age 80.
- **Assisted PD, incremental dialysis and "palliative" or symptom-directed dialysis prescriptions** are increasingly offered as middle paths between full conventional dialysis and pure conservative care.
- **European Renal Best Practice** guidance on older patients with advanced CKD, and new **prediction models comparing dialysis with conservative care** (presented at ASN Kidney Week 2025), give clinicians quantitative tools for shared decision-making.
- **Frailty assessment is now a formal part of dialysis and transplant decision-making**, with a growing literature on frailty's impact on transplant outcomes and on pre-habilitation before starting dialysis.
- **India**: the **Pradhan Mantri National Dialysis Programme** provides **free haemodialysis to below-poverty-line patients in district hospitals across all 36 States/UTs and 751 districts**, in PPP, in-house and hybrid models, with a **peritoneal dialysis component** and a national **PMNDP portal/renal registry**. Roughly **2 lakh Indians develop end-stage kidney failure every year**; access, transport, catastrophic out-of-pocket expenditure and very low deceased-donor transplantation rates remain the dominant limitations for older Indians. Coverage under **AB-PMJAY**

and state schemes has improved but not eliminated these barriers.

Current Practice Update

Aspect	Textbook Position	Current Evidence / Guideline
Timing of initiation	eGFR below ~10 mL/min	**Symptom- and clinically-based**, not eGFR-triggered
Conservative care	Not a formal option	**KDIGO 2024**: conservative kidney care is a reasonable, recognised pathway
Outcome measures	Survival	**Survival plus home time, symptom burden and quality of life**
Basis of the decision	Comorbidity and functional status	**Formal frailty assessment and validated prediction models**
Modality options	HD, PD, transplant	Plus **assisted PD, incremental dialysis, symptom-directed dialysis**
Advance care planning	Withdrawal noted as common	**Structured ACP from the outset**, with the surprise question as a trigger
Indian access	Not addressed	**PMNDP** free dialysis in all districts, with a PD component and national registry

Exam One-Liners

- **Age alone is never a contraindication to dialysis or transplantation.**
- **Median HD survival: 15.6 months at 80–84 and 11.6 months at 85–89.**
- **Elective withdrawal causes 38% of deaths** in the oldest dialysis patients.
- **Poor prognostic markers: malnutrition, dementia, gait disorder, ADL/IADL dependence, late referral.**
- **Transplant: 1-year patient/graft survival 85%/75%; 5-year functional graft survival 55–60% over age 60.**
- **The survival benefit of dialysis all but disappears above 80 with high comorbidity** — offer conservative kidney management as a genuine choice.

End of compilation — Questions 1 to 17.

Bioethics & Medico-Legal Geriatric Medicine

High-Yield Examination Answers

MD Geriatric Medicine — University Examination Preparation

End-of-Life Decision Making · Consent · Capacity · Communication

Primary textbook sources
• Pathy’s Principles and Practice of Geriatric Medicine, 6th edition
• Essentials of Clinical Geriatrics, 7th edition

Supplemented with
• Current evidence and guidelines, 2023–2026
• Indian legal framework — Supreme Court jurisprudence, BNS 2023, NMC/IMC Regulations, Mental Healthcare Act 2017, DPDP Act 2023
• Indian clinical and epidemiological context throughout

Compiled for revision — 5 questions · Textbook content and current updates clearly separated

Contents

1	**Living Will (Advance Medical Directive)** Procedure for writing one in India and how it is executed	**15 marks**
2	**Euthanasia and Physician-Assisted Suicide** Definitions, classification, ethical arguments, Indian legal position	**15 marks**
3	**Informed Consent** Components, disclosure, exceptions, *Samira Kohli* standard	**15 marks**
4	**Decisional Capacity** Factors affecting it and how to assess it	**15 marks**
5	**Communication and Breaking Bad News** Barriers in old age, SPIKES, collusion in the Indian setting	**15 marks**
★	**Rapid Revision Grid** Key dates, cases, statutes, protocols, mnemonics and numbers	**Appendix**

How to use this compilation

Each answer is arranged as **numbered textbook-sourced points** → **Current Practice Update** (two-column comparison of the textbook position against current evidence) → **Latest Updates 2023–present** with inline citations → **Examiner's Tip** panel carrying the verbatim mark-earning lines. Where a topic recurs across past papers, an additional **Extra Points to Remember** panel is included. Read the Examiner's Tip panels and the Rapid Revision Grid on the night before the paper.

Q1 Living Will (Advance Medical Directive) — the procedure to write one in India and how it is executed. 15 MARKS

Source: *Essentials of Clinical Geriatrics*, 7th ed., Ch. 17 — Ethical Issues in the Care of Older Persons (advance care planning, advance directives, living will, proxy directive); *Pathy's* 6th ed. — advance directives in palliative and end-of-life care. India-specific procedure from *Common Cause (A Registered Society) v. Union of India*, (2018) 5 SCC 1, as modified by the Supreme Court order dated **24 January 2023**.

A. Concept and Definitions

1. An **advance directive (AD)** is a legal document by which a person states what treatment they want if they later become **unable to express their wishes**.
2. A **living will** is one type of AD. It states care preferences **specifically for a terminal state at the end of life**.
3. The living will covers a **limited set of treatments** — CPR, intubation, artificial nutrition and hydration, dialysis and intravenous therapy.
4. Its main **limitation** is that it addresses only defined situations (terminal illness, vegetative state) and cannot anticipate grey areas, such as whether an antibiotic is life-support or comfort care.
5. The other form of AD is the **proxy directive** (durable power of attorney for health care), which appoints a person to decide. In geriatrics a **combined instructional plus proxy directive** is the most useful.
6. **Advance care planning** is the *process* of eliciting values and preferences; the living will is only the *document* that results. It should be discussed **in stable health, not during an acute admission**.

B. Legal Basis in India

7. In *Common Cause* (2018) a five-judge Constitution Bench held that the **right to die with dignity is a fundamental right under Article 21**, and gave legal recognition to living wills and to withdrawal of life support.
8. The 2018 procedure proved **unworkable** (magistrate countersignature, boards requiring 20 years' experience, District Collector's involvement), so uptake was negligible.
9. On an application by the **Indian Society of Critical Care Medicine**, the Supreme Court **simplified the procedure on 24 January 2023**. This order is the operative law today.

C. Procedure to WRITE a Living Will (2023 Order)

10. **Who may execute:** any **adult of sound mind**, capable of communicating and of understanding the consequences, acting **voluntarily and without coercion or inducement**.
11. **Form:** it must be **in writing** and must state in **unambiguous terms** the circumstances in which treatment may be **withheld or withdrawn**.
12. It must name **one or more guardians or close relatives** who will give consent for withdrawal when the executor loses capacity (the 2023 order permits **more than one** nominee).
13. **Signing:** the executor signs in the presence of **two attesting witnesses**, preferably independent.
14. **Attestation:** the document must be attested before a **Notary or a Gazetted Officer**. The requirement of **countersignature by a Judicial Magistrate First Class (JMFC) has been removed**.
15. The witnesses and the notary/gazetted officer must **record their satisfaction** that it was executed voluntarily and with full understanding.
16. **Distribution of copies:** to the **nominated guardian(s)**, to the **family physician**, and to family members.
17. **Registration:** a copy must be handed to the **custodian** — a competent official nominated by the **local government** (municipal corporation, municipality or panchayat), who maintains the registry.
18. The executor may also incorporate the directive into their **digital health records**, which greatly improves retrievability in an emergency.
19. **Revocation:** the living will may be **revoked or revised in writing at any time** while the person retains capacity; revised copies must go to all the same recipients.

D. How the Living Will is EXECUTED (Given Effect)

20. **Trigger conditions:** the executor is **terminally ill**, on **prolonged treatment**, with **no hope of recovery or cure**, and has **lost decision-making capacity**.
21. The **treating physician verifies authenticity** of the directive — from the digital health records if available, otherwise

from the custodian.

22. The hospital constitutes a **Primary Medical Board (PMB)**: the treating physician plus **two subject experts**, each with **≥5 years' post-qualification experience**; opinion within **48 hours**.
23. If the PMB agrees, the hospital constitutes a **Secondary Medical Board (SMB)**: a **different set of clinicians (≥5 years' experience)**, including **one nominated by the district Chief Medical Officer**; also within **48 hours**.
24. When **both boards concur**, the hospital **conveys (informs, not seeks approval from) the jurisdictional JMFC** along with the directive or surrogate consent, and implementation may proceed.
25. **If there is no living will:** the same two-board process applies, with **written informed consent of the next of kin / next friend / guardian** acting as surrogate.
26. **In case of non-concurrence or refusal**, the nominee, family member, treating doctor or hospital may move the **High Court under Article 226**; the Chief Justice constitutes a **Division Bench**, which may appoint an **independent three-doctor committee**.
27. **Key distinction: withholding/withdrawing life-sustaining treatment is lawful**; **active euthanasia remains illegal** and punishable in India.

E. Current Practice Update

Textbook position (*Essentials* 7e / *Pathy's* 6e)	Current Indian position (2023–2026)
ADs governed by **statute** — Patient Self-Determination Act 1991 and state laws	India has **no statute**; the operative law is **Supreme Court guidelines under Article 142** (*Common Cause* 2018, modified 2023)
Living will "witnessed, better notarised"; verbal ADs valid in some states	Only a **written** AMD attested before **two witnesses + notary/gazetted officer** is valid; **verbal directives are not recognised**
Document kept by patient, proxy or institution	Additional mandatory **deposit with a local-government custodian**; optional entry into **digital health records**
Futility decided by treating clinician ± **ethics committee**	Decided by **two statutory medical boards (PMB and SMB)** with **48-hour timelines** and **JMFC intimation**
POLST/MOLST as portable medical orders	**No Indian equivalent**; retrieval depends on custodian registries and state digital portals
Artificial nutrition/hydration listed among refusable treatments	**Judicially settled** — *Harish Rana* (2026) held **CANH is medical treatment** and may be withdrawn
A **single** proxy or agent named	The **2023 order permits multiple guardians / close relatives** as surrogates
Directive executed abroad generally portable across states	A living will **validly executed overseas is not legally valid in India** if it does not follow the SC procedure

Latest Updates (2023–Present)

- **Supreme Court order, 24 January 2023** (*Common Cause*, MA 1699/2019 in WP(C) 215/2005): removed JMFC countersignature and District Collector involvement, allowed **notary/gazetted officer attestation**, permitted **multiple nominees**, fixed **48-hour timelines** for both boards, reduced board experience to **5 years**, and made the JMFC's role **notification rather than approval**.
- **MoHFW/DGHS Draft Guidelines for Withdrawal of Life Support in Terminally Ill Patients (2024):** defined **terminal illness** as an irreversible/incurable condition with inevitable death; listed qualifying situations including **brainstem death** and **severe traumatic brain injury with no recovery beyond 72 hours**; gave prescribed formats for surrogate consent and PMB/SMB reports. The **IMA objected** and the guidelines are **not yet notified as final**.
- **Karnataka Government Orders, January–February 2025:** first state to **fully operationalise** the directives — clinicians already empanelled for **brain-death certification under THOTA 1994 (amended 2011)** are deemed nominated to the SMB; **AMD custodians** are Joint Commissioners of BBMP zones (urban) and Taluk Panchayat Executive Officers (rural).
- **Goa (2024):** first state to operationalise AMD registration, with a High Court judge executing the first living will before a gazetted officer.
- **Maharashtra (2024–2026):** BMC **ward medical officers designated custodians** after PIL in the Bombay High Court; a **digital portal (MahaULB)** now allows citizens to upload directives, with municipal commissioners as custodians.
- *Indian Journal of Medical Research, 2026* (Vallath et al.): a **10-step institutional implementation pathway** for withholding/withdrawing life-sustaining treatment with report templates; documented that a **US-executed living will was not legally valid in India**, so the no-AMD pathway had to be used.
- *Indian Journal of Critical Care Medicine, 2025* (Damani et al.): a **12-step advance care planning pathway** adapted to Indian practice.
- ***Harish Rana v. Union of India*, 2026 SCC OnLine SC 358 / 2026 INSC 222 (11 March 2026)** — the landmark update:
  - First case in which the *Common Cause* guidelines were **applied in full measure** by the Supreme Court, in a man in **persistent vegetative state for 13 years**.
  - Held that **clinically assisted nutrition and hydration (CANH) is medical treatment**, not basic care, and may be withdrawn — including when given at home.
  - Laid down the **"best interest" test**: a **balance-sheet exercise** weighing benefit against burden, indignity and futility, incorporating **substituted judgment**, with a rebuttable **presumption in favour of preserving life**.
  - Replaced "passive euthanasia" with **"withholding/withdrawing of medical treatment"**; **"euthanasia" now means active euthanasia only**, which remains impermissible.
  - Recognised a **30-day reconsideration (cooling-off) period**, waivable where all stakeholders are unanimous.
  - Directed **all High Courts** to instruct JMFCs to receive hospital intimations, and the Union and States to ensure every **district CMO maintains a standing panel** for SMB nomination.
  - Clarified the pathway for **home-care patients** and **urged Parliament to enact comprehensive end-of-life care legislation**.

Examiner's Tip

Skeleton: Definition (AD vs living will vs proxy) → Article 21 and *Common Cause* 2018 → why the 2023 modification was needed → **execution: adult of sound mind, written, unambiguous, nominee(s), two witnesses, notary/gazetted officer, copies to nominee + family physician + custodian, optional DHR, revocable → effectuation: trigger → verification → PMB (48 h) → SMB with CMO nominee (48 h) → JMFC intimation → implement** → conflict pathway via High Court under Article 226 → *Harish Rana* 2026 → active euthanasia remains illegal.

"After the Supreme Court's order of **24 January 2023**, a valid living will in India requires only that it be **in writing**, signed before **two attesting witnesses** and attested by a **notary or gazetted officer**, with copies to the **nominated guardian, the family physician and the local-government custodian** — the earlier requirement of countersignature by a Judicial Magistrate First Class stands **removed**."

"Execution now follows a **two-board pathway** — a Primary Medical Board and an independent Secondary Medical Board containing a **CMO nominee**, each reporting within **48 hours** — after which the hospital merely **informs** the jurisdictional JMFC; and since *Harish Rana* (2026), **clinically assisted nutrition and hydration is recognised as medical treatment** that may lawfully be withdrawn in the patient's best interests."

Extra Points to Remember — recurring topic

- **Four numbers to carry into the hall: 2** witnesses · **2** medical boards · **5** years' experience · **48** hours per board. Add **30** days for the *Harish Rana* reconsideration period.
- **Three landmarks in sequence:** *Gian Kaur* 1996 (no right to die) → *Aruna Shanbaug* 2011 (passive euthanasia with High Court approval) → *Common Cause* 2018 (Article 21 right to die with dignity, living wills) → modified 2023 → *Harish Rana* 2026 (first application).
- **The commonest error in answers:** writing that the JMFC must *approve*. Since 2023 the JMFC is only **informed**.
- **Distinguish clearly** between the **living will pathway** (directive verified → boards) and the **no-directive pathway** (surrogate written consent → the same boards). Both use PMB and SMB.
- **Why uptake is still poor in India:** low public awareness, custodians not notified in most states, no national registry, and no statutory backing — hence the Supreme Court's call to Parliament.

Q2 Euthanasia and physician-assisted suicide. 15 MARKS

Source: *Essentials of Clinical Geriatrics*, 7th ed., Ch. 17 (Physician-Assisted Suicide; the seven legal myths of end-of-life care; medical futility) and Ch. 18 (Palliative Care); *Pathy's* 6th ed. — end-of-life decision-making. Indian legal position from *Gian Kaur* (1996), *Aruna Shanbaug* (2011), *Common Cause* (2018, modified 2023), *Harish Rana* (2026), with the Bharatiya Nyaya Sanhita, 2023.

A. Definitions and Classification

1. **Euthanasia** is the act of a physician **deliberately administering a lethal substance or carrying out an intervention to cause the death** of a patient who has decision-making capacity, at that patient's own voluntary request.
2. **Physician-assisted suicide (PAS)** is when a physician, at the voluntary request of a capable patient, **provides or prescribes the means** by which the patient ends their own life.
3. The crucial difference is **who performs the final act** — in euthanasia the **doctor** is the final agent; in PAS the **patient** is the final agent.
4. **By consent: voluntary** (competent patient's own request), **non-voluntary** (patient cannot consent, e.g. vegetative state, advanced dementia), and **involuntary** (against the person's wishes — this is homicide and is never acceptable).
5. **By nature of the act: active** (a positive act causing death) and **passive** (withholding or withdrawing life-sustaining treatment so that death follows from the underlying disease).
6. The term **"passive euthanasia" is now discouraged**. The preferred term is **withholding or withdrawing life-sustaining treatment (WH/WD-LST)**, because the intention is to stop a futile burden, not to cause death.
7. **What is NOT euthanasia:** withdrawing futile treatment, honouring a valid refusal of treatment, a **DNAR order**, **palliative sedation**, and **voluntarily stopping eating and drinking (VSED)**.
8. The **doctrine of double effect** permits giving adequate opioids or sedatives to relieve suffering even if death may be **foreseen but not intended** — this is lawful and ethical practice, not euthanasia.

B. Textbook Content (US Position)

9. In the United States, PAS is **legalised state by state**; **Oregon was the first, in 1997**, under its Death with Dignity Act.
10. The textbook notes that after legalisation, **relatively few terminally ill patients actually requested** physician assistance.
11. **Even where PAS is legal, physicians are not required to participate** — conscientious objection is protected.
12. The textbook lists **seven legal myths** about end-of-life care (ACP/ASIM committee, *JAMA* 2000). Two are examination-relevant: that a doctor giving **high-dose analgesia that hastens death will be prosecuted**, and that **nothing lawful can be offered** when suffering is overwhelming — both are false.
13. Decisions are guided by **medical futility**, which is **quantitative** (death is highly likely and further effort will not succeed) and **qualitative** (the quality of any surviving life). Clinicians must avoid imposing their own quality-of-life judgements.

C. Position in India

14. **Active euthanasia and PAS remain illegal in India.** Only withholding/withdrawing life-sustaining treatment under the Supreme Court's guidelines is lawful.
15. Active euthanasia amounts to **murder under Section 101 BNS 2023**. If the patient is **above 18 and has consented**, **Exception 5** reduces it to **culpable homicide not amounting to murder (Section 105 BNS)** — this **mitigates the charge but does not make the act legal**.
16. **PAS amounts to abetment of suicide under Section 108 BNS 2023** (up to 10 years' imprisonment and fine); abetment involving a child or a person of unsound mind falls under **Section 107 BNS**.
17. **Attempted suicide is effectively decriminalised** — **Section 115 of the Mental Healthcare Act, 2017** presumes severe stress and bars punishment.
18. **Legal milestones:** *Gian Kaur* (1996) — the right to life does **not** include a right to die; *Aruna Shanbaug* (2011) — passive euthanasia permitted with High Court approval; *Common Cause* (2018, modified 24 January 2023) — **right to die with dignity under Article 21**, legalising WH/WD-LST and living wills; *Harish Rana* (2026) — first Supreme Court–sanctioned withdrawal, with **active euthanasia expressly held impermissible**.

D. Ethical Arguments

19. **For:** respect for **autonomy** and self-determination; relief of intractable suffering (**beneficence and mercy**); preservation of **dignity**; logical consistency with the accepted right to refuse life-sustaining treatment; regulation is safer than covert, unregulated practice.
20. **Against:** violates **non-maleficence** and the Hippocratic tradition; the **slippery slope** with progressive widening of indications; danger to **vulnerable older people** (loneliness, poverty, elder abuse, feeling a burden); **prognostic uncertainty**; **depression is treatable and impairs capacity**; erosion of trust in the doctor–patient relationship; conflict with **distributive justice** where palliative care is not universally available.

E. Geriatric Relevance

21. Older adults are the **predominant group** requesting assisted death, and the commonest reasons given are **loss of autonomy, loss of dignity and inability to enjoy activities — not uncontrolled pain**.
22. A request to die is often a **cry for help**. Always screen for **untreated pain, depression, delirium, existential distress, caregiver strain and the feeling of being a burden**.
23. Specific geriatric dilemmas include **capacity assessment in dementia**, the validity of **advance euthanasia directives** in dementia, and the **"completed life" or "tiredness of life"** debate in the very old.
24. The correct clinical response is to **explore the request rather than react to it** — optimise symptom control, refer early to **palliative care or hospice**, treat depression, and address social and spiritual suffering.

F. Current Practice Update

Textbook position (*Essentials* 7e / *Pathy's* 6e)	**Current position, 2023–2026**
PAS legal in a few US states; **Oregon 1997** the first	Now legal in **~12 US jurisdictions**, with **New York and Illinois added since 2024**; Oregon **removed its residency requirement in 2023**, causing a step increase in prescriptions
"**Very few** terminally ill patients actually request PAS"	Uptake is now **substantial and rising** — Oregon reported **400 deaths in 2025**, and in Canada **MAiD accounted for 5.1% of all deaths in 2024**
Framed entirely in the **US legal context**	In **India**, PAS = **abetment of suicide (BNS s.108)** and active euthanasia = **murder (BNS s.101; Exception 5 → s.105)** — the BNS replaced the IPC from **1 July 2024**
"Passive euthanasia" used loosely for withdrawal of treatment	The Supreme Court in *Harish Rana* (2026) formally **retired the term**, replacing it with **"withholding or withdrawing medical treatment"**; "euthanasia" now means **active euthanasia only**
Eligibility framed around **terminal illness**	Marked international **drift beyond terminal illness** — Canada's **Track 2** (death not reasonably foreseeable), psychiatric illness and dementia in the Benelux countries, and France's new law with **no prognostic time limit**
Artificial nutrition/hydration debated as basic care versus treatment	Judicially settled in India — *Harish Rana* (2026) held **CANH is medical treatment** and may be withdrawn
"Physicians are not required to participate"	Reaffirmed and strengthened — the **WMA (2019)** holds that no physician should be forced to participate **or even to make a referral**

Latest Updates (2023–Present)

Professional and ethical positions

- **WMA Declaration on Euthanasia and Physician-Assisted Suicide (2019)** and the **WMA Declaration of Venice on End of Life Medical Care**: the WMA is **firmly opposed** to both practices, and states that **no physician should be forced to participate or to refer**. This remains its standing position.
- **EAPC White Paper** and **IAHPC Position Statement**: withholding or withdrawing futile, burdensome treatment is **not euthanasia**, because the intention is not to hasten death; **non-voluntary and involuntary euthanasia must never be permitted**.

India

- **Bharatiya Nyaya Sanhita, 2023** (in force 1 July 2024) renumbered the offences — **murder s.101** (punishment s.103), **culpable homicide not amounting to murder s.105** with **Exception 5** for consensual killing of an adult, **abetment of suicide s.108**, and **abetment of suicide of a child or person of unsound mind s.107**.
- ***Harish Rana v. Union of India*, 2026 SCC OnLine SC 358 / 2026 INSC 222 (11 March 2026):** the first Supreme Court–sanctioned withdrawal of life support in India. Held **CANH is medical treatment**; laid down a **"best interest" balance-sheet test** incorporating substituted judgement; recognised a **30-day reconsideration period** (waivable where all agree); directed **High Courts** to instruct JMFCs to receive hospital intimations and **CMOs** to maintain SMB panels; and **urged Parliament to enact comprehensive end-of-life care legislation** — while reaffirming that **active euthanasia remains impermissible**.

International legal developments

- **United Kingdom:** the **Terminally Ill Adults (End of Life) Bill** passed the Commons in **June 2025 (314–291)** and had its Lords second reading in September 2025, but **ran out of parliamentary time in April 2026** after more than 1,200 amendments; it is to be reintroduced. Assisting a suicide remains punishable under the Suicide Act 1961.
- **Isle of Man: Assisted Dying Act passed by Tynwald in March 2025** — terminal illness with expected death within **12 months**, capacity, 5 years' residency, verification by two independent doctors.
- **Jersey: Assisted Dying Law approved 26 February 2026 (32–16)** — terminal illness with expected death within **6 months (12 months for neurodegenerative disease such as Parkinson's disease or MND)**; notably permits **clinician administration as well as self-administration**.
- **France: legalised assisted dying in July 2026** — no prognostic time limit, but requires an **advanced, life-threatening illness causing intolerable and incurable suffering**, mental capacity, a voluntary informed request, medical assessment, and protection of **conscientious objection**; **mental illness alone does not qualify**.
- **Scotland:** the Assisted Dying for Terminally Ill Adults (Scotland) Bill remains before the Scottish Parliament.
- Other jurisdictions legalising since 2024 include **Uruguay, Ecuador, the Australian Capital Territory, New York and Illinois**.

Outcome data

- *Oregon Health Authority, 28th Annual DWDA Report (2026, data for 2025):* **637 prescriptions** and **400 deaths**; **88% aged ≥65 years**; diagnoses **cancer 61%, neurological 14%, heart disease 11%**; **92% enrolled in hospice** and **80% died at home**. The three leading end-of-life concerns were **loss of autonomy (89%), inability to participate in enjoyable activities (89%) and loss of dignity (65%)** — **pain was not among the leading reasons**.
- *Health Canada Annual MAiD Report (2025, data for 2024):* **16,499 MAiD deaths**, or **5.1% of all deaths in Canada** (Quebec **7.9%**), with over **76,000 cumulative deaths** since 2016. **Track 2** accounted for **732 deaths**. Roughly **half of recipients reported feeling a burden** on family or caregivers.
- **Canada — expansion to mental illness as the sole underlying condition** has been deferred three times and is currently scheduled for **March 2027**; the **UN Human Rights Committee (2025)** urged Canada to repeal Track 2 and halt further expansion — the central real-world test of the **slippery slope argument**.

Examiner's Tip

Skeleton: Define euthanasia and PAS (WMA wording) → distinguish by **final agent** → classify by **consent** and by **act** → list what is **not** euthanasia (futility, DNAR, double effect, palliative sedation, VSED) → textbook US position (Oregon 1997, low uptake, no compulsion, seven legal myths, futility) → **Indian law:** *Gian Kaur* → *Aruna Shanbaug* → *Common Cause* 2018/2023 → *Harish Rana* 2026, with **BNS s.101/105 and s.108** → arguments for and against → **geriatric angle** → conclude with **palliative care as the answer to the demand for euthanasia**.

"The distinction is not merely semantic: in **euthanasia the physician performs the final act**, in **physician-assisted suicide the patient does** — and in India the first attracts **Section 101 BNS (murder, mitigated to Section 105 by Exception 5 where an adult consents)** while the second attracts **Section 108 BNS (abetment of suicide)**. Only **withholding or withdrawing life-sustaining treatment** under the *Common Cause* guidelines is lawful."

"The Oregon and Canadian data make the geriatric point sharply — the dominant reasons for requesting assisted death are **loss of autonomy, loss of dignity and inability to enjoy life, not uncontrolled pain**, and around **half of Canadian recipients report feeling a burden** — which means that in an ageing society the most effective response to a request for hastened death is **early, accessible palliative care, treatment of depression, and relief of caregiver and social burden**."

Extra Points to Remember — recurring topic

- **The one-line discriminator:** "Who administers the final act?" Doctor = euthanasia. Patient = PAS.
- **Never write "passive euthanasia is legal in India."** Write: "**Withholding and withdrawing life-sustaining treatment** is lawful under Supreme Court guidelines; **euthanasia (active) is not**."
- **Double effect has four conditions:** the act itself must be good or neutral; the bad effect must not be the *means* to the good effect; the **intention** must be relief of suffering; and there must be **proportionate reason**.
- **BNS section numbers replaced IPC from 1 July 2024:** IPC 302 → **BNS 101/103**; IPC 304 → **BNS 105**; IPC 306 → **BNS 108**; IPC 309 (attempt) → overridden by **MHCA s.115**.
- **Slippery slope — the examinable evidence:** Canada's progression from terminal illness (2016) → **Track 2, death not reasonably foreseeable (2021)** → proposed **mental illness alone (deferred to March 2027)**, with MAiD now **5.1% of all deaths**.
- **Closing line that scores:** "The demand for euthanasia is largely a demand for better dying; where palliative care is accessible, the demand falls."

Q3 Informed consent. 15 MARKS

Source: *Essentials of Clinical Geriatrics*, 7th ed., Ch. 17 — Competence and Informed Consent; decisional capacity; informed assent; surrogate decision-making; Table 17-1 Major Ethical Principles. *Pathy's* 6th ed. Indian legal position from *Samira Kohli v. Dr Prabha Manchanda*, (2008) 2 SCC 1, the IMC/NMC Regulations, and *Common Cause* (2018, modified 2023).

A. Definition and Components

1. Informed consent is the doctrine requiring a clinician to **disclose enough information for the patient to make an informed decision** before any investigation, procedure or treatment. It is a **process of communication, not a signature on a form**.
2. Three components must all be satisfied: **capacity to decide**, **voluntariness** (free of coercion or undue influence), and **adequate information** that the patient actually understands.
3. The textbook lists **five items the clinician must disclose**: (i) the **condition being treated**; (ii) the **nature and character** of the proposed treatment or surgery; (iii) the **anticipated results**; (iv) **recognised alternative forms of treatment**; and (v) **recognised serious risks, complications and benefits** — of the proposed treatment, of the alternatives, and of **non-treatment**.
4. Consent is **continuing and revocable**. A patient who makes an informed decision may **change their mind at any time** — a competent person who refuses dialysis does not lose the right to reconsider later.
5. The ethical foundation is **autonomy**, balanced against **beneficence, non-maleficence, justice and fidelity**.

B. Capacity versus Competence

6. **Capacity** is a **clinical judgement** made by the treating clinician about a **specific decision at a specific time**. **Competence is a legal term** — a determination made by a **court of law**.
7. Capacity is **decision-specific, not global**, and follows a **sliding scale** — the more serious the consequences, the higher the threshold demanded.
8. The **four elements of decisional capacity** are: (i) **understanding**; (ii) **appreciation**; (iii) **reasoning**; and (iv) **expressing a choice**.
9. **A diagnosis of dementia is not by itself proof of incapacity.** Intellectual deficits are "spotty" — a patient may forget things yet still decide well **with appropriate prompting**. It is wrong to prejudge, and equally wrong to **infantilise** the patient by directing questions to relatives who answer faster.
10. **Distinguish memory from understanding** — these are not the same thing.
11. **No cognitive screening test determines capacity.** Tests of **executive function** (**Clock Drawing Test, Executive Interview**) correlate better with decisional capacity than the **MMSE**. Formal instruments exist but are difficult for older adults because they demand abstract, hypothetical reasoning.
12. A practical bedside test recommended by the textbook is to **re-check the patient after an interval** and confirm that the understanding and the stated preference are **consistent**.
13. Always look for and correct **reversible causes of apparent incapacity** — **delirium, depression, fear, pain, metabolic derangement, drug effects**, and **hearing or visual impairment**. Capacity may **fluctuate**.
14. Patients who are comatose, or who have **expressive or receptive aphasia** or moderate-to-severe dementia, may be unable to communicate effectively — assess carefully **what they can still understand and express** rather than assuming nothing.

C. When Capacity is Absent

15. Authority passes to a **surrogate** — through an **advance directive / living will**, a **durable power of attorney for health care (proxy)**, or a court-appointed **guardian or conservator**.
16. Surrogate decisions follow a hierarchy: **the patient's known prior wishes → substituted judgement (what the patient would have chosen) → best interests** (weighing benefits against burdens).
17. **"Informed assent"** is a useful option — where the family or proxy does not want to carry the burden of the decision, they may be **explicitly offered the choice to defer to the clinician's judgement**. This must be **clearly documented**.

D. Exceptions and Pitfalls

18. Recognised exceptions are **emergency/necessity** (unconscious patient, life-saving intervention), **patient waiver**, and

therapeutic privilege (narrow and now rarely accepted).

19. Common pitfalls in geriatric practice: **blanket or pre-printed consent forms**, consent taken **on the trolley or after premedication**, **relatives signing when the patient is capable**, and failure to record the discussion. **A signed form without disclosure is not consent.**
20. In geriatrics the evidence itself is often **absent, extrapolated from younger patients, or contradictory** — so risks and benefits are genuinely uncertain, and **clinician values may unconsciously distort how options are framed**. Shared decision-making and confirming understanding (e.g. **teach-back**) are therefore essential.

E. Indian Legal Position

21. India applies the **"real consent"** standard laid down in ***Samira Kohli v. Dr Prabha Manchanda*, (2008) 2 SCC 1** — **not** the American "reasonable/prudent patient" standard.
22. **Key propositions from *Samira Kohli*:** consent for a **diagnostic procedure is not consent for therapy**; consent for **one procedure does not cover another**, and the fact that the extra surgery was **beneficial does not validate it**; **relatives cannot consent to an additional procedure on an unconscious patient** except in a genuine life-threatening emergency; for major surgery **two separate consents** should be taken; and the disclosure standard is the **modified Bolam (professional) standard**.
23. **Legal consequences:** operating **without any consent = battery**; **inadequate disclosure = negligence**; breach of **Regulation 7.16** (written consent before operations) = **professional misconduct**. The emergency/necessity defence lies in **Section 92 IPC, now Section 28 BNS 2023**.

F. Current Practice Update

Textbook position (*Essentials* 7e / *Pathy's* 6e)	**Current position, 2023–2026**
Five-item disclosure list, with no named legal standard	India applies the **modified Bolam professional-disclosure standard** (*Samira Kohli*, 2008), unlike the **patient-centred standard** of *Canterbury v Spence* (USA) and *Montgomery* (UK)
"Informed consent laws do not specify how much must be disclosed"	The **seven propositions of *Samira Kohli*** are now the **operative test** applied by every Indian consumer forum, High Court and the Supreme Court
No screening test determines capacity; executive tests better than MMSE	Still correct; structured instruments (**MacCAT-T, Aid to Capacity Evaluation, CCTI**) are validated, and longitudinal data show **reasoning is the first of the four abilities to fail** in mild-to-moderate dementia
Surrogates and guardians defined by **US state statutes**	India has **no general statutory surrogate hierarchy** for an adult who loses capacity — the closest are the **nominated representative under the Mental Healthcare Act, 2017** and the **guardian named in an advance medical directive**
Advance directives framed by the **US Patient Self-Determination Act**	India's **advance medical directive** became operational after the Supreme Court's order of **24 January 2023**, giving **prospective consent** legal force
Substance of consent matters more than the form	In India **written consent is mandatory for operations** under **Regulation 7.16**, but a **blanket form** ("any procedure found necessary") remains **legally invalid**
Consent framed around **treatment decisions** only	Consent is now **layered** — clinical consent (*Samira Kohli*/NMC), **research consent** (ICMR National Ethical Guidelines 2017), and **health-data consent** (DPDP Act 2023 with DPDP Rules 2025)
Surrogate and family decisions handled informally	For withholding/withdrawing life-sustaining treatment, India now requires the **surrogate's written consent in a prescribed format** plus **two medical boards** and **JMFC intimation**

Latest Updates (2023–Present)

Indian regulatory framework

- **NMC Registered Medical Practitioner (Professional Conduct) Regulations, 2023** (Gazette, 2 August 2023) carried a dedicated **guidance document on informed consent** stating that the consent process **must never be curtailed or neglected because the patient is unable to "fully understand"**, and expressly naming **the elderly, the marginalised and the illiterate** as vulnerable groups requiring extra care. Research consent was excluded and referred to the **ICMR Guidelines, 2017**.
- **These Regulations were held in abeyance on 23 August 2023** after objections from the IMA and other bodies, and the **Indian Medical Council (Professional Conduct, Etiquette and Ethics) Regulations, 2002 — including Regulation 7.16 — were revived and remain in force**. The 2023 Regulations are **not operative** until a further gazette notification. *This is the single most examinable regulatory fact.*
- **Bharatiya Nyaya Sanhita, 2023** (in force 1 July 2024): the good-faith and necessity protections formerly in **IPC ss. 87–92** are renumbered as **BNS ss. 25–28**, with **Section 28 BNS** covering acts done in good faith for a person's benefit **where consent cannot be obtained**.
- ***Common Cause*** (2018, modified **24 January 2023**) and ***Harish Rana*** (2026) confirm that a valid **advance medical directive is a recognised form of consent**, binding the clinician at the future moment of incapacity.

Health-data consent — a new statutory layer

- **Digital Personal Data Protection Act, 2023** with the **DPDP Rules, 2025** (notified 13–14 November 2025) make consent for processing patient data a **separate statutory requirement**. Consent must be **free, specific, informed, unconditional, unambiguous and revocable**, supported by an **itemised notice** in plain language.
- Implementation is **phased**: the **Data Protection Board** from November 2025, **consent managers** from **November 2026**, and the **core compliance obligations** (notice, security, breach reporting, data-principal rights) from **May 2027**. Hospitals are **data fiduciaries**; large ones may be designated **significant data fiduciaries** requiring a **data protection officer**. Clinical establishments have **limited exemptions** where processing is necessary to provide health services.

Capacity assessment and shared decision-making

- *JAGS 2024:* scoping review of **decision-making tools that support substitute decision-makers** for adults with impaired capacity — reflecting a shift from simply *declaring* incapacity to actively *supporting* the surrogate.
- *JAGS 2025:* scoping review of **shared decision-making interventions for deprescribing** in older adults with polypharmacy — the principle being that **consent to stop a drug deserves the same rigour as consent to start one**.
- Internationally, the direction of travel is from **substitute decision-making towards supported decision-making**, driven by **UNCRPD Article 12** and, in India, the **Rights of Persons with Disabilities Act, 2016** and the **Mental Healthcare Act, 2017**, both of which begin from a **presumption of capacity**.

Examiner's Tip

Skeleton: Define consent as a **process, not a form** → three components (**capacity, voluntariness, information**) → the **five disclosure items** → **capacity vs competence** → the **four elements** → dementia ≠ incapacity; **executive tests beat MMSE**; reversible causes → if incapable: **advance directive → proxy → substituted judgement → best interests**, with **informed assent** as an option → exceptions (**emergency, waiver, therapeutic privilege**) → **Indian law: *Samira Kohli* "real consent"**, battery vs negligence, **Regulation 7.16**, **Section 28 BNS** → newer layers: **AMD, ICMR research consent, DPDP data consent**.

> "**Capacity is a clinical determination made by the treating doctor for a specific decision at a specific time; competence is a legal determination made by a court** — and capacity rests on four abilities: **understanding, appreciation, reasoning and expressing a choice**. No cognitive screening test, including the MMSE, establishes capacity; **executive-function measures correlate better**, and a **diagnosis of dementia is never by itself evidence of incapacity**."

> "In India the operative standard is **'real consent'** under *Samira Kohli v. Dr Prabha Manchanda* (2008): **consent for a diagnostic procedure is not consent for therapy, consent for one procedure does not authorise another, and the fact that the additional surgery was beneficial does not validate it** — relatives cannot consent for an unconscious patient's additional procedure except in a genuine life-threatening emergency. A **signed pre-printed form without disclosure is evidence of a signature, not of consent**."

Extra Points to Remember — recurring topic

- **Battery vs negligence — the classic viva discriminator: no consent at all → battery/assault** (trespass to the person, no proof of harm needed); **consent obtained but disclosure inadequate → negligence** (harm and causation must be proved).
- **Three disclosure standards worldwide: Bolam / professional standard** (what a reasonable doctor would disclose) — **India**; **reasonable patient standard** (*Canterbury v Spence*) — USA; **particular patient / material risk standard** (*Montgomery v Lanarkshire*, 2015) — UK.
- **Valid consent in India must be:** voluntary · by a person **≥18 years** of sound mind · informed · specific to the procedure · **written for operations** (Reg. 7.16) · obtained **before premedication** · documented.
- **The three exceptions in one line: Emergency** (necessity — BNS s.28), **Waiver** (patient chooses not to know), **Therapeutic privilege** (disclosure itself would gravely harm — now very narrow and best avoided).
- **Geriatric traps:** consent taken from the son because he speaks English; consent signed on the trolley; the "blanket" form; assuming a hearing-impaired patient is confused. All four are examinable failures.
- **Special consents to name:** HIV testing, organ donation (THOTA), sterilisation, clinical research (ICMR 2017), photography/publication, and **data processing (DPDP 2023)**.

Q4 Decisional capacity — factors affecting it and how to assess it. 15 MARKS

Source: *Essentials of Clinical Geriatrics*, 7th ed., Ch. 17 (Competence and Informed Consent; four elements of decisional capacity) and Ch. 6 (Delirium and Dementia — mental status examination, executive function, reversible causes, Table 6-5 drugs causing cognitive impairment); *Pathy's* 6th ed. — Confusion Assessment Method.

A. Definition and Governing Principles

1. **Decisional capacity** is a **clinical judgement** by the treating doctor that a patient can make **one particular decision at one particular time**. **Competence is a legal term** — determined by a **court of law**.
2. Every adult is **presumed to have capacity** until the contrary is demonstrated. The burden of proof lies on the person asserting incapacity.
3. Capacity is **decision-specific and time-specific, never global**. A patient may lack capacity for a complex surgical decision yet retain capacity to refuse a blood test or to choose where to live.
4. A **sliding (risk-proportionate) scale** applies — the graver the consequences, the greater the certainty required before accepting the patient's choice.
5. **An unwise decision is not incapacity.** Capacity is judged by the **process of reasoning**, not by the **outcome chosen**.
6. Capacity rests on **four elements**: (i) **understanding** the disclosed information; (ii) **appreciation** of what it means for one's own situation; (iii) **reasoning** through risks and benefits; and (iv) **expressing a choice**. **All four must be present.** Denial of illness typically fails at *appreciation*; intact memory with poor planning fails at *reasoning*.

B. Factors Affecting Decisional Capacity

7. **Neurocognitive disorders:** dementia of any type, mild cognitive impairment, stroke, Parkinson's disease. **Executive dysfunction** correlates with capacity far better than memory loss — in frontotemporal dementia, appreciation and reasoning fail early despite preserved recall.
8. **Delirium** is the commonest **reversible** cause in hospital — defined by **acute onset, fluctuation, inattention** and disturbed consciousness; diagnose with the **CAM** (features 1 and 2 plus either 3 or 4). Frequently **superimposed on dementia** and often missed in its **hypoactive** form.
9. **Psychiatric illness:** depression (hopelessness distorts appreciation and the weighing of risk — **"depressive pseudodementia"** causes patchy deficits and frequent "don't know" answers), psychosis, mania, severe anxiety or fear.
10. **Drugs** — a major and correctable contributor: **anticholinergics, antihistamines, opioids, benzodiazepines and sedative-hypnotics, antipsychotics, anticonvulsants, antiparkinsonian drugs, steroids, digoxin, H_2-receptor antagonists, muscle relaxants and alcohol**. Recent **anaesthesia and premedication** matter — consent taken after premedication is invalid.
11. **Medical and metabolic causes:** hypoxia, hypoglycaemia, hyponatraemia, uraemia, hepatic encephalopathy, hypothyroidism, B_{12} deficiency, sepsis, dehydration, uncontrolled **pain**, sleep deprivation, and — classically in geriatrics — **faecal impaction and urinary retention**, whose relief can produce dramatic improvement.
12. **Sensory and communication barriers:** deafness, poor vision, dysarthria, and **expressive or receptive aphasia**. Older patients are often mislabelled "confused" or "senile" simply because they were **not given adequate time to answer** or could not hear the question.
13. **Educational, linguistic and cultural factors:** low literacy, an unfamiliar language or dialect, technical jargon, and family-centred decision norms in which the patient defers to relatives.
14. **Decision-related factors:** complexity, novelty, number of options, volume of information, and **urgency or time pressure**.
15. **Environmental and assessor factors:** a noisy unfamiliar ward, time of day (**sundowning**), a rushed disclosure, a coercive relative in the room, and the clinician's own **framing bias**.
16. **Capacity fluctuates.** It must be reassessed at the patient's best time and again after reversible factors have been treated.

C. How to Assess Decisional Capacity — Seven Steps

17. **Step 1 — Optimise before assessing.** Treat delirium, pain and depression; stop offending drugs; correct metabolic derangement; supply hearing aids and spectacles; use the patient's own language or an interpreter; choose a quiet room and the best time of day; allow unhurried time.

18. **Step 2 — Disclose properly.** Plain language in short chunks, repeat, use written or pictorial aids, then confirm with **teach-back** — "tell me in your own words what we have decided".
19. **Step 3 — Structured interview mapped to the four abilities**, using the patient's own words:
    - **Understanding** — "What is wrong with you? What treatment have I suggested? What could go wrong?"
    - **Appreciation** — "Why do you think this has been advised for *you*? What will happen if you do not have it?"
    - **Reasoning** — "How did you arrive at that decision? What made this option better than the other?"
    - **Expressing a choice** — "So what have you decided?"
20. **Step 4 — Screening tests support only. No cognitive screening test determines capacity.** The **MMSE alone is inadequate**; tests of **executive function** — the **Clock Drawing Test**, the **Mini-Cog** (three-item recall plus clock drawing) and the **Executive Interview** — correlate far better.
21. **Step 5 — Structured instrument in difficult or contested cases.** The **MacArthur Competence Assessment Tool for Treatment (MacCAT-T)** is the reference standard and scores all four abilities; the **Aid to Capacity Evaluation (ACE)** is short, free and practical at the bedside; **MacCAT-CR** and **UBACC** are used for research consent. These tools demand abstract, hypothetical reasoning and are therefore difficult for many older adults.
22. **Step 6 — Re-check for consistency** after an interval to confirm that understanding and the stated preference are **stable**, as the textbook specifically recommends.
23. **Step 7 — Document and escalate.** Record the **specific decision**, the information given, the patient's **verbatim responses under each of the four abilities**, the reversible factors addressed, the conclusion and the plan for reassessment. Seek a **second opinion (psychiatry or neuropsychology)** when stakes are high, the assessment is contested, capacity fluctuates, or the choice conflicts with the person's lifelong values.
24. **If capacity is absent**, never make a **blanket declaration**. Support what the patient can still do (**supported decision-making**), then fall back to **advance directive → nominated representative or proxy → substituted judgement → best interests**, and **reassess once reversible factors are corrected**.

D. Current Practice Update

Textbook position (*Essentials* 7e / *Pathy's* 6e)	Current position, 2023–2026
Four elements of capacity described narratively	Formalised as the **Appelbaum–Grisso four-abilities model**, now the international standard and the basis for the **MacCAT-T** and **MacCAT-CR**
"No cognitive screening test is appropriate"; executive tests beat the MMSE	**Unchanged and reinforced** — the **Mini-Cog** (recall + clock drawing) is the preferred bedside screen for who needs fuller assessment
Capacity instruments described as "difficult for older adults"	**23 instruments** now catalogued, with **considerable heterogeneity** in criteria and operationalisation; MacCAT-T remains the reference standard, **ACE** the practical bedside choice
Four-abilities model presented as settled	Recent commentary documents **substantial inter-rater variability**, poorer performance in **MCI**, demonstrated **racial bias**, and neglect of **emotion and values** — the call is for **safeguards and flexibility**, not abandonment
Capacity framed as a binary clinician determination	Shift towards **supported decision-making** under **UNCRPD Article 12**, India's **RPWD Act 2016** and **MHCA 2017** — support first, substitute only as a last resort
Surrogate arrangements described through **US guardianship and proxy statutes**	**India: MHCA 2017 Section 4** codifies a three-limb capacity test with a **presumption of capacity** and a **nominated representative**; for general medical decisions there is still **no statutory surrogate hierarchy** outside the advance medical directive route
Capacity assessed as a one-off event	Emphasis now on **serial reassessment**, because delirium persists in a substantial minority and capacity is dynamic

Latest Updates (2023–Present)

Indian statutory framework

- **Mental Healthcare Act, 2017, Section 4** is the only Indian statute that defines capacity. Its three limbs are: ability to **understand information relevant to the decision**; ability to **appreciate any reasonably foreseeable consequence of a decision or of the lack of a decision**; and ability to **communicate the decision by speech, expression, gesture or any other means**. The Act begins from a **presumption of capacity**, endorses **supported decision-making**, and restricts the **nominated representative** to those found to lack capacity.
- *Indian Journal of Psychiatry* commentary highlights a drafting problem: the statute joins the three limbs with **"or"** rather than "and", which literally read would allow a person satisfying only one limb to be deemed capacitous. Commentators and clinical practice hold that **all three must be satisfied**; there have been repeated calls for an amendment and a **Code of Practice** for capacity assessment.
- For general medical (non-psychiatric) decisions, the operative rules remain the **capacity limb of "real consent"** in *Samira Kohli* (2008) and the **advance medical directive** framework under *Common Cause* (2018, modified 2023) and *Harish Rana* (2026).

Evidence on the assessment model itself

- *Journal of the American Academy of Psychiatry and the Law, 2024:* the four-skills model shows **considerable variability between evaluators**, especially pronounced in **mild cognitive impairment due to Alzheimer's disease**, and has shown **high levels of racial bias**. The authors argue the model is often applied **too inflexibly** and to situations for which it was not designed, and stress that in many circumstances loss of capacity is **temporary or reversible**.
- *European Psychiatry, 2025* (Kupsch et al., systematic review to March 2024): identified **23 instruments** assessing capacity to consent to treatment, with **considerable heterogeneity** in criteria and operationalisation — a caution against treating any single tool's cut-off as definitive.
- *BMC Psychiatry, 2025* (Norwegian stakeholder study): the four-abilities model is the primary basis of capacity assessment in specialist care, but **systematic training, validated tools and further research are needed** to improve assessment quality in routine practice.
- *JAGS 2024:* scoping review of **decision-making tools that support substitute decision-makers** — emphasis has shifted from merely *declaring* incapacity to actively *supporting* the surrogate.
- *JAGS 2025:* scoping review of **shared decision-making interventions for deprescribing** in older adults with polypharmacy — consent to **stop** a drug requires the same capacity assessment as consent to start one.

Epidemiology that drives practice

- Meta-analytic data in hospitalised older adults show a **delirium prevalence of about 21%** and a **frailty prevalence of about 34%**, with frailty an **independent risk factor for delirium (RR 1.66)** — a large minority of ward patients have a **potentially reversible** barrier to capacity at any moment.
- **Persistent delirium** is common: pooled prevalence at discharge around **36%**, and modelling suggests roughly **16% at 12 months** — capacity must be **reassessed serially**, not certified once.
- Dementia prevalence in older general-hospital inpatients ranges from **13% to 63%**, and fewer than one-third of studies screened for **delirium or depression** — misclassification of reversible impairment as dementia is frequent.
- Longitudinal **MacCAT-T** data in mild-to-moderate dementia show that **reasoning is the ability that fails first and most often** (about 23% impaired at 9 months; about 26% impaired overall), while **appreciation and expressing a choice are relatively preserved**. Clinically, **probe reasoning hardest**.

Examiner's Tip

Skeleton: Capacity (clinical) vs competence (legal) → presumption of capacity → **decision-specific, time-specific, sliding scale**; an unwise decision is not incapacity → the **four abilities** with model questions → **factors**: neurocognitive, delirium, psychiatric, drugs, metabolic, sensory, educational-cultural, decision complexity, environment and assessor bias, fluctuation → **assessment**: optimise → disclose with teach-back → four-ability interview → screening tests support only (**Mini-Cog/Clock/EXIT, not MMSE alone**) → **MacCAT-T / ACE** if contested → re-check consistency → document → second opinion → if incapable: **support first**, then AD → nominated representative → substituted judgement → best interests, and **reassess**.

"**Capacity is decision-specific, time-specific and risk-proportionate — it is never a global label**, and it is judged by the **process of reasoning, not by the choice made**. The four abilities that must all be present are **understanding, appreciation, reasoning and expressing a choice**; in dementia, **reasoning is the ability that fails first**, so it is the one to probe hardest."

"**No cognitive screening test determines capacity** — the MMSE least of all. Tests of **executive function (Clock Drawing, Mini-Cog, Executive Interview)** correlate far better, and the **MacCAT-T** or the **Aid to Capacity Evaluation** should be used when the case is contested. But before any of this, the first duty is to **remove the reversible barrier** — delirium, pain, depression, an anticholinergic, hyponatraemia, faecal impaction, or simply a missing hearing aid — because in geriatric practice most apparent incapacity is **treatable, fluctuating and temporary**."

Extra Points to Remember — recurring topic

- **Mnemonic for the four abilities — "U-A-R-C": U**nderstanding · **A**ppreciation · **R**easoning · **C**hoice.
- **Mnemonic for reversible causes — "DELIRIUM": D**rugs · **E**lectrolytes and endocrine · **L**ack of drugs (withdrawal) · **I**nfection · **R**educed sensory input · **I**ntracranial · **U**rinary/faecal retention · **M**yocardial and pulmonary (hypoxia).
- **The three commonest wrong answers in vivas:** (i) "MMSE <24 means no capacity" — false; (ii) "he refused surgery so he lacks capacity" — outcome bias; (iii) "he has dementia so the son will sign" — capacity was never assessed.
- **Delirium vs dementia in one line:** delirium = **acute, fluctuating, inattention, altered consciousness**; dementia = **gradual, progressive, consciousness preserved until late**.
- **Assent vs consent:** where the patient lacks capacity, still seek **assent** — their agreement at their own level — and note dissent, which should carry real weight in the best-interests calculation.
- **Capacity is also needed for non-medical decisions** that a geriatrician is often asked about: testamentary capacity (making a will), financial capacity, capacity to drive, capacity to live alone, and capacity to consent to sexual activity in a care home. **Each is assessed separately.**

Q5 How will you communicate and break bad news to an older patient? 15 MARKS

Source: *Essentials of Clinical Geriatrics*, 7th ed., Ch. 3 — Evaluating the Geriatric Patient (Table 3-2, Potential Difficulties in Taking Geriatric Histories) and Ch. 17; *Pathy's* 6th ed. — Communication Disorders and Dysphagia, and Ch. 8 Health Literacy and Cultural Sensitivity (Tables 8.5 and 8.6, barriers and areas of cultural conflict).

A. General Principles of Communication in Geriatrics

1. Effective communication means a message is conveyed **and correctly interpreted**. It depends on hearing, vision, attention, arousal, memory and cognition — **every one of which may be impaired in an older patient**.
2. The textbook lists three core barriers in geriatric history-taking: **diminished hearing, diminished vision and slowed psychomotor performance**.
3. Practical corrective measures are examinable verbatim: use a **well-lit room**, **eliminate extraneous noise**, **speak slowly in a deep tone**, **face the patient so the lips can be seen**, use a **simple amplification device** for severe hearing loss, **write questions in large print** if needed, and **leave enough time to answer**.
4. **Patience is truly a virtue.** Thought and verbal processes are slower; hurrying loses important information. The rule of open-ended questions may need to be **tempered** to fit the time available.
5. Older patients **underreport symptoms** because of health beliefs, fear, depression and cognitive impairment. Ask **specific direct questions** and use **collateral history** from relatives and caregivers.
6. **Non-verbal communication carries a great deal of information.** Cultures differ in pace of conversation, tolerance of interruption, physical proximity, eye contact, touch and finger-pointing. Attitudes to the **direct discussion of death** vary widely.
7. The **electronic record is itself a communication barrier** — patients perceive it as disrespectful when the clinician interacts more with the screen than with the person.
8. Use **professional interpreters, not children**. Misinterpretation happens even in a shared language — a patient saying "stomach ache" may mean pain anywhere in the abdomen.
9. Avoid **stereotyping**. Rather than memorising every culture, **ask the patient or interpreter** what is acceptable, record it, and revisit it at follow-up. **There is no substitute for asking the older adult what their goals are.**
10. In **dementia**, use the speech-therapy strategies given in *Pathy's*: **use names instead of pronouns**, **avoid open-ended questions by offering definite options**, **encourage gesture**, and use **communication prompts such as pictures and charts**. Training caregivers with **memory books and wallets** improves interaction.
11. Avoid **ageist language and elderspeak**. Do not direct questions to the relative who answers faster — that infantilises the patient.

B. Breaking Bad News — the SPIKES Protocol

12. **S — Setting.** Private, quiet, well-lit room; **sit down at eye level**; ensure the **hearing aid and spectacles are in place**; have an amplifier ready; silence the phone; ask **who the patient wants present**; allow uninterrupted time.
13. **P — Perception. Ask before you tell:** "Before I give you the results, tell me what you understand about what has been happening."
14. **I — Invitation.** Ask **how much the patient wants to know** and in what detail. Some want full details, others want a relative told. **The right not to know must be respected.**
15. **K — Knowledge.** Give a **warning shot** ("I'm afraid I have some difficult news"), then deliver it in **plain language in small chunks**. Use the real word — "cancer", "dementia" — not euphemisms. **Pause frequently** and check understanding with **teach-back**.
16. **E — Emotions and empathy.** Expect silence, tears, denial or anger. Respond with **NURSE** statements — **N**ame the emotion, express **U**nderstanding, show **R**espect, offer **S**upport, and **E**xplore. **Never answer an emotional statement with more data.**
17. **S — Strategy and summary.** Agree the next steps, address symptom control, give a **written summary**, a **contact person** and a **follow-up date**. **Never say "there is nothing more we can do"** — reframe to what *can* be done.

C. Other Frameworks Worth Naming

18. **Ask–Tell–Ask** for each chunk of information; **REMAP** (Reframe, Expect emotion, Map values, Align, Plan) for goals-of-care conversations; **Best Case / Worst Case** and **time-limited trials** for treatment decisions; the **Serious Illness Conversation Guide** for values and prognosis.

D. Geriatric- and India-Specific Issues

19. Disclose to the **patient first** if capacity is present. If capacity is impaired, work through the **advance directive** and the **nominated surrogate**, but still tell the patient at their level — **dementia does not abolish the right to know**.
20. **Collusion** — the family asking the doctor to withhold the truth — is common in India. Do not simply comply, and do not simply override. **Explore the family's fear**, ask what they think will happen if the patient is told, obtain their permission to ask the patient how much they wish to know, and then use the **patient's own invitation** as the ethical route through.
21. **Prognostic communication:** give **ranges, not point estimates** ("hours to days", "weeks to months"), acknowledge uncertainty honestly, and hold **hope and preparedness together** — "hope for the best, prepare for the worst".
22. **Document** who was present, what was disclosed, the questions asked and the plan; then **brief the whole team** so the message is consistent. **Mixed messages from different team members are a leading cause of complaints.** Debrief afterwards — most clinicians report feeling low after breaking bad news.

E. Current Practice Update

Textbook position (*Essentials* 7e / *Pathy's* 6e)	**Current position, 2023–2026**
Generic communication tips and cultural-sensitivity advice	Formalised, teachable, evidence-based protocols — **SPIKES, Ask–Tell–Ask, NURSE, REMAP, Best Case/Worst Case** and the **Serious Illness Conversation Guide**
No named standard for **dementia diagnosis disclosure**	**AAN / Alzheimer's Association multi-stakeholder Delphi best practices (2024)**, the **DETeCD-ADRD guideline (2025)** for primary care, and the **CLEAR guidelines (2025)** for culturally sensitive disclosure
Family involvement framed as culturally appropriate	Reframed as **patient-directed** — the patient decides who is present and how much is disclosed; **collusion is managed explicitly, not accommodated by default**
Disclosure treated as a single event	Now a **longitudinal, repeated conversation**, with a **written summary and care plan**, because patients and families recall very little from the disclosure visit
Communication skill assumed to be acquired by experience	**Systematic review evidence (2025)** shows structured training improves communication behaviours in **93%** of studies and self-efficacy in **96%**, largely sustained at follow-up
Prognosis discussion left to individual judgement	**Structured tools reliably improve process but not yet outcomes** — they increase documentation of goals-of-care discussions, with mixed effects on concordant care and utilisation
Electronic record noted as a barrier	Compounded by **tele-consultation and AI scribes**; the principle is unchanged — **attend to the person, not the screen**

Latest Updates (2023–Present)

Dementia diagnosis disclosure — the biggest change

- *Neurology Clinical Practice, 2024* (AAN process, multi-stakeholder modified Delphi including a patient and caregivers) issued **seven best-practice statements**. The strongest is that **clinicians must show compassion and empathy when delivering a diagnosis of dementia (Level A)**. Clinicians should also **ask about the patient's diagnosis preferences, instil realistic hope, provide practical strategies, provide education and links to high-quality resources, and connect caregivers to support**. A **written record or care plan** is recommended because patients and families recall the disclosure visit poorly and often differently.
- **The driving statistic:** in **about 50%** of people whose billing records indicate dementia, the **patient or care partner reports never having been told the diagnosis** — even though the **vast majority want to know**.
- **Alzheimer's Association DETeCD-ADRD clinical practice guideline** (*Alzheimer's & Dementia, 2025*) covers diagnostic evaluation, testing, counselling and disclosure for primary care, and reports that **39% of primary care physicians are "never or only sometimes" comfortable making a dementia diagnosis**.
- **O'Brien, Largent and Karlawish** (*Alzheimer's & Dementia, 2025;* summarised in *JAMA, 2025*) give practical guidance: before disclosing, assess the patient's **understanding and appreciation of their symptoms, their goals for the evaluation, and their desire for information**. Because Alzheimer's disease itself distorts symptom perception, an **informant or care partner should be included**.
- **CLEAR guidelines** (*Alzheimer's & Dementia, 2025*) — *Compassionate Language and Empathetic Approaches for Respectful dementia disclosure* — a culturally sensitive framework encouraging disclosure of **aetiology, type, prognosis and support resources**, together with **active listening, empathy and explicit emotional support**, and preparation for a difficult conversation.

Evidence on structured communication tools and training

- *BMJ Supportive & Palliative Care, 2026* — systematic review and meta-analysis of **22 randomised trials** of communication tools and decision aids for hospitalised adults (2015–2025). Tools **increased documentation of goals-of-care discussions (OR 1.46, 95% CI 1.04–2.05)**; effects on resuscitation preferences were **inconsistent (OR 1.84, 95% CI 0.95–3.60)**; effects on **concordant care, psychosocial outcomes and healthcare utilisation were mixed**. The honest verdict: structured tools reliably improve **process**, not yet **outcomes**.
- *BMC Medical Education, 2025* — systematic review of communication skills training in chronic care: **communication behaviours improved in 93% (37/40) of studies**, **self-efficacy and confidence in 96% (26/27)**, and attitudes in **80%**, with gains **largely sustained at follow-up**. The gap identified is **standardisation and scalability**.
- *Journal of Pain and Symptom Management, 2025* — an **ICU-tailored Serious Illness Conversation Guide** proved suitable for critical care, produced more comprehensive skills among fellows and self-reported practice change among faculty.
- **AI-simulated patients** for communication training are now under **randomised evaluation (2025–2026)** — potentially scalable, but as yet unproven against standardised-patient teaching.

Indian context

- **Collusion — family-led non-disclosure — remains widespread.** Surveys of Indian oncologists show that although the great majority favour truth-telling, **only a minority will refuse a family's request to withhold the diagnosis**, and most agree to tell the truth **only if the patient asks**.
- A structured-interview study of **127 patients** at a tertiary oncology centre in **Bengaluru** found that **72% wanted disclosure of a cancer diagnosis** — a preference associated with **higher education and English proficiency** — and that **most also wanted their family involved**. The examinable implication: **the default assumption of non-disclosure is not supported by Indian patient-preference data**; family involvement and patient disclosure are **not alternatives**.
- **PENS**, a four-step abbreviated protocol (*Supportive Care in Cancer, 2020*), has been proposed as an India-adapted alternative to SPIKES for the busy oncology outpatient department, where a full SPIKES conversation is often impractical.
- Indian surveys also report that most clinicians **have received no formal training** in breaking bad news and want dedicated teaching, and that a **majority feel depressed after breaking bad news** — supporting the case for structured training and team debriefing.

Examiner's Tip

Skeleton: Define communication and its geriatric barriers (**hearing, vision, slowed processing**) → the textbook's practical measures (**quiet, well-lit room, deep slow voice, face the patient, amplifier, large print, time**) → underreporting and collateral history → non-verbal and **cultural** factors, interpreters, avoiding stereotyping, the EMR barrier → dementia-specific strategies (**names not pronouns, definite options, gesture, picture prompts**) → **SPIKES step by step**, with **NURSE** for emotion → **Ask–Tell–Ask, REMAP, Best Case/Worst Case, SICG** → geriatric issues: capacity, assent, surrogate, **collusion**, prognosis in ranges → document, brief the team, debrief.

> "Before a single word of bad news is spoken, the **preconditions for communication** must be met — a quiet, well-lit private room, the patient's **hearing aid and spectacles in place**, the clinician seated at eye level and facing the patient so the lips can be read, and **enough unhurried time**. In geriatrics, most 'poor communication' is not a failure of empathy but a failure to correct a **sensory barrier**."

> "The single strongest recent recommendation is that **clinicians must show compassion and empathy when delivering a diagnosis of dementia (Level A, AAN / Alzheimer's Association Delphi 2024)** — and the reason it was needed is that **about half of people with dementia report never having been told their diagnosis, although the vast majority want to know**. In the Indian setting, **collusion is managed neither by complying nor by overriding**: explore the family's fear, then obtain the **patient's own invitation** to determine how much they wish to know."

Extra Points to Remember — recurring topic

- **SPIKES expanded: S**etting · **P**erception · **I**nvitation · **K**nowledge · **E**motions · **S**trategy and summary. **NURSE** for empathy: **N**aming · **U**nderstanding · **R**especting · **S**upporting · **E**xploring.
- **The three golden rules: Ask before you tell** · **Fire a warning shot** · **Answer emotion with empathy, never with data**.
- **Handling collusion in four moves:** (1) meet the family separately and **listen to the fear**; (2) ask "what do you think will happen if he is told?"; (3) explore the **cost of concealment** — isolation, mistrust, unfinished business, inability to consent; (4) ask permission to **ask the patient how much he wishes to know**, and be guided by the patient's own answer.
- **Never use:** "There is nothing more we can do" · "We are withdrawing care" (say **withdrawing a treatment that is no longer helping**) · "Do you want us to do everything?" (an unanswerable question — map values instead).
- **Prognosis:** use **ranges** — hours to days, days to weeks, weeks to months, months to years — and pair with the **"hope for the best, prepare for the worst"** dual framing.
- **Culture is fluid — ask, do not assume.** Record the patient's stated preferences about disclosure and family involvement in the notes, and revisit them at follow-up.

Rapid Revision Grid — Bioethics & Medico-Legal Geriatrics

1. Indian Case Law — the Five Landmarks

Case	Holding to quote
***Gian Kaur v. State of Punjab*, 1996**	Right to life under Article 21 **does not include a right to die**. Overruled *P. Rathinam*.
***Aruna Shanbaug v. Union of India*, 2011**	Permitted **passive euthanasia with High Court approval**; active euthanasia illegal.
***Common Cause v. Union of India*, (2018) 5 SCC 1**	Five-judge Bench: **right to die with dignity is a fundamental right under Article 21**; legalised **living wills** and WH/WD-LST.
SC Order, 24 January 2023 (MA 1699/2019)	Simplified the procedure — **notary/gazetted officer** replaces JMFC countersignature; **48-hour** board timelines; **5 years'** experience; multiple nominees; JMFC only **informed**.
***Harish Rana v. Union of India*, 2026 INSC 222 (11 Mar 2026)**	**CANH is medical treatment** and may be withdrawn; **best-interest balance-sheet test**; **30-day** reconsideration; term "passive euthanasia" retired; **active euthanasia remains impermissible**.
***Samira Kohli v. Dr Prabha Manchanda*, (2008) 2 SCC 1**	**"Real consent"** standard; modified **Bolam** disclosure; diagnostic consent ≠ therapeutic consent; relatives cannot consent for extra surgery on an unconscious adult.

2. Statutes and Regulations — Numbers to Get Right

Provision	Content
BNS s.101 (was IPC 302)	Murder — **active euthanasia**. Punishment s.103.
BNS s.105, Exception 5 (was IPC 304)	Culpable homicide not amounting to murder where an **adult >18 consents** — mitigates, does not legalise.
BNS s.108 (was IPC 306)	**Abetment of suicide** — physician-assisted suicide. Up to **10 years** + fine.
BNS s.107	Abetment of suicide of a **child or person of unsound mind**.
BNS s.28 (was IPC 92)	Act done in **good faith** for benefit **where consent cannot be obtained** — the emergency/necessity defence.
MHCA 2017, s.115	Attempted suicide — **presumption of severe stress**; not to be punished. Effectively decriminalised IPC 309.
MHCA 2017, s.4	Only Indian statutory **capacity test**: understand · appreciate consequences · communicate. **Presumption of capacity**; nominated representative.
IMC Regulation 7.16 (2002)	**Written consent mandatory before operations**; breach = professional misconduct. **Currently operative.**
NMC RMP Regulations 2023	Notified 2 Aug 2023 with an informed-consent guidance document; **held in abeyance 23 Aug 2023**. **2002 Regulations remain in force.**
BNS in force	**1 July 2024** — replaced the IPC.
DPDP Act 2023 + Rules 2025	Health-data consent: free, specific, informed, unconditional, unambiguous, **revocable**. Board Nov 2025 → consent managers Nov 2026 → core obligations **May 2027**.

3. Living Will — the Procedure in Numbers

Element	Requirement (post-January 2023)
Executor	Adult, sound mind, voluntary, understands consequences
Form	**Written**, unambiguous; verbal directives **not valid**
Witnesses	**Two** attesting witnesses
Attestation	**Notary OR Gazetted Officer** (JMFC countersignature removed)
Nominees	**One or more** guardians / close relatives

Copies to	Nominee(s) · **family physician** · family · **local-government custodian** · optionally digital health records
Primary Medical Board	Treating physician + **2 experts**, each **≥5 years**; opinion in **48 hours**
Secondary Medical Board	Different clinicians ≥5 years + **1 CMO nominee**; **48 hours**
JMFC	**Informed only** — no approval required
Dispute pathway	**High Court, Article 226** → Division Bench → independent **3-doctor** committee
Reconsideration period	**30 days** (*Harish Rana* 2026); waivable if all stakeholders unanimous

4. Global Assisted-Dying Status — Facts and Figures

Jurisdiction / source	Key data
Oregon DWDA — first US law	**1997**; residency requirement **removed 2023**
Oregon 28th Annual Report (2026, data 2025)	**637** prescriptions, **400** deaths; **88% aged ≥65**; cancer **61%**, neurological **14%**, cardiac **11%**; hospice **92%**; died at home **80%**
Oregon — top three reasons	Loss of **autonomy 89%** · inability to enjoy activities **89%** · loss of **dignity 65%**. **Pain not in the top three.**
Canada MAiD (2025 report, data 2024)	**16,499** deaths = **5.1% of all deaths** (Quebec **7.9%**); **>76,000** cumulative since 2016; **Track 2 = 732**; ~**half** felt a burden
Canada — mental illness alone	Deferred three times; currently **March 2027**. UN HRC (2025) urged repeal of Track 2.
UK	Terminally Ill Adults Bill passed Commons **June 2025 (314–291)**; **ran out of time April 2026**; to be reintroduced
Isle of Man	Act passed **March 2025**; prognosis **≤12 months**; 5 years' residency
Jersey	Approved **26 February 2026 (32–16)**; **≤6 months**, or **12 months** for neurodegenerative disease; permits **clinician administration**
France	Legalised **July 2026**; **no prognostic time limit**; mental illness alone excluded
WMA (2019)	**Firmly opposed**; no physician forced to participate **or to refer**

5. Consent, Capacity and Communication — Frameworks

Framework	Components
Three components of consent	**Capacity** · **Voluntariness** · **Information**
Five disclosure items (*Essentials*)	Condition · nature of treatment · anticipated results · alternatives · risks and benefits, **including of non-treatment**
Four abilities — capacity (U-A-R-C)	**U**nderstanding · **A**ppreciation · **R**easoning · expressing a **C**hoice. **Reasoning fails first in dementia.**
Three exceptions to consent	**Emergency/necessity** · **Waiver** · **Therapeutic privilege**
Three disclosure standards	**Bolam / professional** — India · **Reasonable patient** (*Canterbury*) — USA · **Material risk** (*Montgomery* 2015) — UK
Surrogate hierarchy	Prior known wishes → **substituted judgement** → **best interests**
SPIKES	**S**etting · **P**erception · **I**nvitation · **K**nowledge · **E**motions · **S**trategy/summary
NURSE (empathy)	**N**aming · **U**nderstanding · **R**especting · **S**upporting · **E**xploring
REMAP (goals of care)	**R**eframe · **E**xpect emotion · **M**ap values · **A**lign · **P**lan
Capacity instruments	**MacCAT-T** (reference standard) · **ACE** (bedside) · MacCAT-CR, UBACC (research). **23 instruments** catalogued (Eur Psychiatry 2025)

Cognitive screens	**Mini-Cog** = 3-item recall + clock drawing. **Executive tests > MMSE** for capacity. **No test determines capacity.**
Delirium	**CAM** = features 1 **and** 2, plus 3 **or** 4: acute/fluctuating · inattention · disorganised thinking · altered consciousness

6. Numbers from Recent Evidence

Finding	Figure
Dementia diagnosis never disclosed	**~50%** of those with billing-record dementia (AAN 2024)
PCPs uncomfortable diagnosing dementia	**39%** "never or only sometimes" (DETeCD-ADRD, 2025)
Communication skills training — behaviours improved	**93% (37/40)** of studies; self-efficacy **96% (26/27)** (BMC Med Educ 2025)
Communication tools → GOCD documentation	**OR 1.46 (1.04–2.05)**; resuscitation preferences **OR 1.84 (0.95–3.60), NS** (BMJ Support Palliat Care 2026)
Indian patients wanting cancer diagnosis disclosed	**72%** of 127, Bengaluru tertiary centre
Delirium in hospitalised older adults	**~21%**; frailty **~34%**; frailty → delirium **RR 1.66**
Persistent delirium	**~36%** at discharge; **~16%** at 12 months
Dementia in older inpatients	**13–63%**; <1/3 of studies screened for delirium or depression
MacCAT-T in mild–moderate dementia	**Reasoning impaired first** — ~**23%** at 9 months, ~**26%** overall

Six lines that will earn marks in almost any bioethics answer

- "**Capacity is clinical and decision-specific; competence is legal and determined by a court.**"
- "**An unwise decision is not incapacity** — capacity is judged by the process of reasoning, not the outcome chosen."
- "**Withholding and withdrawing life-sustaining treatment is lawful in India; active euthanasia is not.**"
- "**A signed form without disclosure is evidence of a signature, not of consent.**"
- "Since the order of **24 January 2023**, the JMFC is **informed, not asked to approve**."
- "In geriatrics, most apparent incapacity is **treatable, fluctuating and temporary** — correct the reversible barrier before you certify anything."

Session index — questions in this compilation

1. Living will — procedure and execution in India • **2.** Euthanasia and physician-assisted suicide • **3.** Informed consent • **4.** Decisional capacity — factors and assessment • **5.** Communication and breaking bad news

Outstanding in the broader syllabus: potassium, calcium and magnesium disorders.

MD GERIATRIC MEDICINE · EXAMINATION REVISION COMPENDIUM

Falls, Fracture Prevention and Pressure Injuries

Structured high-yield answers with current evidence to 2026 and Indian clinical context

Module: Falls · Fracture Prevention · Pressure Injuries
Questions covered: Five (15-mark format)
Primary references: Pathy's Principles and Practice of Geriatric Medicine, 6th edition
Kane's Essentials of Clinical Geriatrics
Evidence updated to: August 2026

Prepared for Dr. Sudarsan · Night-before-examination revision format · All values in Indian conventional units

Contents

Q1 Assessment of Fall Risk and Prevention of Falls in the Elderly

Definition and burden · risk factors · STEADI · performance testing · multifactorial intervention · World Falls Guidelines · NICE NG249 · USPSTF 2024

Q2 Tools for Falls Risk Assessment

Screening tools · performance-based gait and balance tests · cut-offs · inpatient scales · fear-of-falling scales · supporting assessments

Q3 Role of Sensor Devices in the Prevention of Falls

Wearable and ambient sensors · machine learning risk indices · context-aware alerting · virtual reality · fall detection · barriers and evidence gaps

Q4 Prevention of Fractures

Fragility fracture · FRAX · hip protectors · bisphosphonates and anabolic agents · sequential therapy · Fracture Liaison Services · ISBMR

Q5 Pressure Sores: Stages, Common Sites, Prevention and Treatment

NPIAP staging · DTPI and unstageable · site by posture · Braden/Norton/Waterlow · repositioning · stage-wise treatment · 2025 International Guideline

How each answer is structured. Every question follows the same four-part architecture: **(1)** numbered high-yield points under lettered subheadings, keywords in bold; **(2) Latest Updates (2023–Present)** — named trials, guidelines and policy, cited inline; **(3) Current Practice Update** — a table contrasting the textbook position with current evidence; **(4) Exam One-Liners** — rapid-recall statements for final revision.

QUESTION 1

Assessment of Fall Risk and Prevention of Falls in the Elderly

15 MARKS | 20 HIGH-YIELD POINTS

A. Definition and Burden

1. A **fall** is defined as an *unexpected event in which the person comes to rest on the ground, floor, or lower level*. Syncope and seizure must first be excluded by history.
2. About **one-third of community-dwelling adults ≥65 years fall each year**. Among **nursing home residents, one in two falls each year**. **One in five falls** causes serious injury such as fracture or head trauma.
3. In India, **LASI wave-1** data show **about 11.6% of adults ≥60 years report a fall**, with higher rates in **women** and with **advancing age**; falls are heavily under-reported.
4. Complications include fractures of **hip, femur, humerus, wrist and ribs**, **subdural haematoma** (may present days to weeks later), immobility, **fear of falling**, loss of confidence, institutionalisation and death.

B. Why Falls Occur

5. Falls are **multifactorial** — an *acute trigger* acting on *accumulated chronic impairments*. **Rarely is a fall due to a single factor.**
6. Age-related changes: **reduced proprioception**, slowed righting reflexes, **increased antero-posterior postural sway**, loss of **type II (fast-twitch) fibres**, stiffer joints. **Gait speed declines 12–16% per decade.**
7. **Dual-tasking** (walking while talking or counting) becomes difficult with age; institutionalised elders who cannot converse while walking have high fall risk.

C. Risk Factors

8. Divided into **intrinsic** (ageing neuromuscular physiology, disease) and **extrinsic** (medications, environment). Risk rises **exponentially** as factors accumulate.
9. Useful mnemonic — **AGAIN I'VE FALLEN**: **A**gain fallen (previous fall), **G**ait/balance problems, **A**DL loss, **I**mpaired mental status, **N**umber of drugs (>4), **I**llness/delirium, **V**estibular disorders, **E**yes/Ears, **F**oot disorders, **A**lcohol, **L**ower-limb weakness, **L**ow BP (postural), **E**nvironmental hazards, **N**eurological disorders.
10. **Medications are the commonest and most modifiable risk factor** — benzodiazepines, Z-drugs, antipsychotics, all antidepressants (TCA/SSRI/SNRI),

opioids, NSAIDs, muscle relaxants, antihistamines/anticholinergics, alpha-blockers, diuretics, antihypertensives, digoxin, amiodarone, insulin/sulphonylureas.

11. Risk is **highest in the days to weeks immediately after starting a drug or changing its dose** — start new drugs during periods of supervision and warn the patient.
12. Disease-related risk: **dementia (2–8 times higher fall rate)**, stroke, **Parkinson's disease**, arthritis, peripheral neuropathy, chronic pain, **orthostatic hypotension**, carotid sinus hypersensitivity, urinary urgency and nocturia.
13. **Fear of falling (FoF)** is itself an independent predictor of future falls — it causes activity restriction, deconditioning and consumes cognitive reserve needed for gait.
14. **Delirium is the leading cause of falls in hospital**; environmental triggers include wet floors, poor lighting, loose rugs, clutter, steps and unstable furniture.

D. Assessment

15. **Screening (STEADI algorithm, CDC):** ask **every older adult whether they have fallen in the past year**. Those who screen positive (or through the **Stay Independent 12-item questionnaire** / "three key questions") undergo full risk assessment.
16. **History:** exclude syncope/seizure, circumstances of the fall, relation to meals or medication, premonitory symptoms (palpitations, focal deficit), loss of consciousness, alcohol, and full drug list.
17. **Examination: BP lying, sitting and standing** (fall of **>20 mmHg systolic** = orthostatic hypotension), visual acuity, cardiac murmurs/arrhythmia/carotid sinus, neurological and cerebellar signs, joints, **feet and footwear**, and inspection of walking aids for defects.
18. **Performance-based tests (must know):**
    - **Gait speed** over 4 m — **<0.6 m/sec is abnormal**; every **0.1 m/sec fall in speed = 7% rise in fall risk**
    - **Timed Get Up and Go Test (GUGT/TUG) — >12 seconds = high risk** (sensitivity 77%, PPV 93%)
    - **30-second chair stand** and **4-stage balance test**
    - **Tinetti Mobility Scale (POMA) — score <24/28 indicates fall risk**
    - **Berg Balance Scale** (0–56) and **Short Physical Performance Battery (SPPB)** (0–12)
19. **Investigations: no routine laboratory or imaging tests are needed.** Minimum work-up is **lying/standing BP and a 12-lead ECG**. MRI only if a treatable neurological cause is suspected (infarcts, hydrocephalus, cerebellar degeneration); EEG only with strong suspicion of seizure.

E. Prevention and Management

20. **Multifactorial, targeted intervention is the cornerstone:**
    - **Exercise (best evidence): balance and functional exercise reduces fall rate by 24%**; **multiple exercise types (balance + resistance) by 34%**; **Tai chi by 19%**. Walking alone or resistance training alone is of uncertain benefit.
    - **Medication review and withdrawal of psychotropics** — proven to reduce falls.
    - **Home hazard assessment and modification**, most effective when done by an **occupational therapist** (grab bars, lighting, removing rugs, raising bed/chair height).
    - **Vision: cataract surgery** (first eye) reduces falls; replace **multifocal with single-vision lenses** for outdoor use.
    - **Vitamin D:** benefit mainly in those with **low baseline levels** and in **nursing-home residents**.
    - **Cardiac pacing** for cardioinhibitory **carotid sinus hypersensitivity**.
    - **Hip protectors:** small benefit in care homes (RR 0.82); **no clear benefit in the community**; adherence is poor.
    - **In hospital/nursing homes:** prevent and treat **delirium**. **Physical restraints, bed rails and bed alarms do NOT reduce falls** and may cause harm.

LATEST UPDATES (2023–PRESENT)

1. **World Falls Guidelines (Age and Ageing) — three-tier risk stratification, now the global standard.** Older adults are stratified into **low risk** (education and general exercise), **intermediate risk** (targeted exercise or physiotherapy referral for balance and strength), and **high risk** (full multifactorial falls risk assessment with individualised tailored interventions). High-risk features include recurrent falls, injurious falls, frailty and balance problems.
2. **NICE NG249 (April 2025) — replaces the 2013 CG161 guideline.** It covers everyone aged 65 and over, **plus people aged 50–64 who are at higher risk**. Key shifts:
    - **Do not use formal falls risk-prediction tools.** Instead, directly ask all people ≥65 (and 50–64 with risk-enhancing conditions such as dementia, arthritis, Parkinson's disease, stroke, diabetes or learning disability) about **any fall in the past year**.
    - Comprehensive falls assessment is offered to those meeting defined criteria, including **two or more falls in the last year**; those who fell once without these criteria have their **gait and balance assessed**.
    - **Offer a home hazard assessment using a validated tool**, preferably delivered by an **occupational therapist**; **consider cognitive behavioural interventions** where concern about falling is not helped by strength and balance exercises; **investigate carotid sinus hypersensitivity and consider cardiac pacing** for unexplained falls.

    - **Vitamin D is not recommended specifically to reduce falls** — evidence is insufficient — though people are encouraged to take it for bone and muscle health.

3. **USPSTF 2024 (JAMA) — exercise upgraded, multifactorial downgraded. Exercise interventions are recommended (Grade B)** to prevent falls in community-dwelling adults ≥65 at increased risk. The decision to offer **multifactorial interventions must be individualised (Grade C)**, weighing prior falls, comorbidities and patient preference, because the **overall net benefit of routinely offering them is small**.
4. **USPSTF draft statement (December 2024) — vitamin D for falls.** A **Grade D (recommend against)** was proposed for **vitamin D, with or without calcium, to prevent falls or fractures** in community-dwelling postmenopausal women and men ≥60 years. This does **not** apply to those with diagnosed osteoporosis or vitamin D deficiency.
5. **Cochrane Review 2025 (Dyer et al., CD016064) — care facilities. Multifactorial interventions implemented with facility staff engagement and tailored to individual residents probably reduce the rate of falls and risk of falling, and may be cost-effective**; among single interventions, **exercise probably reduces falls**. **Vitamin D supplementation** also likely reduces falls in care facilities, and increasing dietary dairy servings may reduce the number of people falling.
6. **STOP-FALLS trial (JAMA Network Open, 2024) — deprescribing alone is not enough.** In 2,367 older adults, a health-system-embedded deprescribing intervention targeting **CNS-active medications** (opioids, benzodiazepines/Z-drugs, muscle relaxants, TCAs, first-generation antihistamines) **did not significantly reduce medically treated falls** compared with usual care; modest prescribing improvements occurred without a measurable effect on clinical outcomes.
7. **STOPPFall (EuGMS Task and Finish Group on Fall-Risk-Increasing Drugs).** A validated Delphi-derived tool listing **14 medication classes** (mostly psychotropics) that increase fall risk, combined with **practical stepwise deprescribing guidance**. This is now the preferred structured framework for FRID review.
8. **Indian evidence (LASI wave-1 analyses, 2022–2026). 11.6% of Indian older adults report falls**, higher in women and with advancing age; independent determinants are **female sex, underweight BMI, tobacco/alcohol use, chronic joint disease, prior injury or fracture, sensory impairment, depression, ADL limitation and poor physical performance**. Environmental and drug factors dominate — **slippery floors (OR 2.28)**, **polypharmacy (OR 1.71)**, **hearing loss (OR 1.83)** and **depression (OR 1.62)**. **Frail** older Indians have higher falls (15.4% vs 11.9%), multiple falls (7.7% vs 5.3%) and fall-related injuries (6.7% vs 5.3%) than non-frail.
9. **Indian service delivery.** Fall risk screening is delivered through **NPHCE** — geriatric clinics at PHC/CHC level, **rehabilitation units and physiotherapy at district hospitals**, **Regional Geriatric Centres**, and **domiciliary visits by**

rehabilitation workers for bed-bound elders. Assistive devices and mobility aids are supplied through **ADIP/RVY via ALIMCO and DDRCs**.

10. **Emerging directions (2023–2026).** Sensory impairment — especially **hearing loss and dizziness** — is being proposed for inclusion in risk-stratification algorithms. Modified algorithms using a **shorter TUG cut-point (10 seconds)** are being tested to improve stratification into the intermediate-risk group. Wearable fall-detection devices and tele-rehabilitation are expanding access where multifactorial clinics are unavailable.

CURRENT PRACTICE UPDATE

Domain	Textbook Position	Current Practice (2023–Present)
Risk identification	STEADI screening with structured questionnaires and risk assessment	**NICE NG249 advises against formal falls risk-prediction tools**; simply ask about falls in the past year and stratify into **low / intermediate / high risk** per World Falls Guidelines
Target population	Adults ≥65 years	Extended to **50–64 years with risk-enhancing conditions** (dementia, Parkinson's, stroke, arthritis, diabetes, learning disability)
Exercise	Balance and functional exercise beneficial; Tai chi effective	**Strongest and most consistent intervention (USPSTF Grade B)**; must be **progressive, tailored and supervised** (Otago, FaME)
Multifactorial intervention	Recommended for all recurrent fallers	**Individualise the decision (USPSTF Grade C)** — net benefit is small if applied routinely; reserve for **high-risk** patients
Vitamin D	Useful when baseline levels are low and in nursing-home residents	**Not recommended solely for falls prevention in the community** (NICE 2025; USPSTF draft Grade D); **still supported in care-facility residents** (Cochrane 2025)
Medication review	Withdrawal of psychotropics reduces falls	Structured **STOPPFall** review preferred; **STOP-FALLS (2024) showed deprescribing alone did not reduce falls** — must be combined with exercise and other measures
Fear of falling	Recognised as a predictor; no specific therapy named	**Cognitive behavioural intervention** now recommended when exercise alone does not relieve concern about falling
Care homes	Evidence for exercise unclear in nursing homes	**Staff-engaged, individually tailored multifactorial programmes +**

		exercise + vitamin D probably reduce falls (Cochrane 2025)
Hospital	Restraints, bed rails, bed alarms ineffective	Unchanged; emphasis added on **preventing deconditioning** — encourage patients to dress, stand and walk; refer to community services **at discharge**

EXAM ONE-LINERS

- **Fall** = unexpected event coming to rest on ground/floor/lower level.
- **One-third** of community elders and **one-half** of nursing-home residents fall yearly; **1 in 5** falls injures.
- **India (LASI): 11.6%** prevalence; slippery floors, polypharmacy, hearing loss and depression are key drivers.
- **Mnemonic: AGAIN I'VE FALLEN.**
- **Medications = most modifiable risk factor**; danger window is **immediately after starting or changing a drug**.
- **Gait speed <0.6 m/sec** abnormal; **0.1 m/sec drop = 7% ↑ risk**.
- **TUG >12 seconds = high fall risk.**
- **Tinetti POMA <24/28 = fall risk.**
- **Orthostatic hypotension = fall of >20 mmHg systolic** on standing.
- **No routine labs**; minimum = **lying/standing BP + 12-lead ECG**.
- **Delirium = leading cause of inpatient falls.**
- **Balance + functional exercise ↓ falls 24%; multiple exercise types ↓ 34%; Tai chi ↓ 19%.**
- **Restraints, bed rails and bed alarms do NOT prevent falls.**
- **Hip protectors** — modest benefit in care homes (RR 0.82), none in the community.
- **Cardiac pacing** for **cardioinhibitory carotid sinus hypersensitivity** with unexplained falls.
- **NICE NG249 (2025): do not use falls risk-prediction tools.**
- **USPSTF 2024: exercise = Grade B; multifactorial = Grade C.**
- **STOPPFall = 14 fall-risk-increasing drug classes.**
- **Fear of falling** is both a consequence and an independent predictor — treat with **CBT**.

QUESTION 2

Tools for Falls Risk Assessment

15 MARKS | 20 HIGH-YIELD POINTS

A. General Principles

1. Falls assessment tools serve **three distinct purposes**: (i) **screening / case-finding**, (ii) **risk stratification**, and (iii) **objective measurement of gait and balance deficits** to target intervention and monitor progress.
2. **No single tool is adequate alone.** Tools supplement — never replace — a **detailed history, medication review, postural blood pressure and targeted physical examination**.
3. The most powerful single predictor is not a scale but a simple question — **"Have you fallen in the last one year?"** A previous fall is the strongest predictor of a future fall.

B. Screening / Case-Finding Tools

4. **STEADI algorithm (Stopping Elderly Accidents, Deaths and Injuries — CDC):** a **stepwise three-stage approach** — *Screen* → *Assess* → *Intervene*. It is the standard framework for community falls screening.
5. **"Three Key Questions" (STEADI):**
   - Have you **fallen in the past year**?
   - Do you feel **unsteady when standing or walking**?
   - Do you **worry about falling**?

   A "yes" to any question triggers full risk assessment.
6. **Stay Independent 12-item questionnaire:** a self-administered checklist; a **score of ≥4 indicates increased fall risk** and mandates further assessment.
7. **AGAIN I'VE FALLEN mnemonic:** a bedside checklist for systematic risk-factor identification — **A**gain fallen, **G**ait/balance problems, **A**DL loss, **I**mpaired mental status, **N**umber of drugs (>4), **I**llness/delirium, **V**estibular disorders, **E**yes/Ears, **F**oot disorders, **A**lcohol, **L**ower-limb weakness, **L**ow BP (postural), **E**nvironmental hazards, **N**eurological disorders.

C. Performance-Based Gait and Balance Tools

8. **Gait speed** over a pre-measured **4-metre** course. **0.6–0.8 m/sec = high-risk range; <0.6 m/sec is the accepted cut-off for abnormal gait.** Every **0.1 m/sec fall in speed carries a 7% rise in fall risk**. It also predicts disability, hospitalisation, institutionalisation and mortality.

9. **Timed Get Up and Go Test (GUGT / TUG):** patient rises from a standard armchair, walks **3 metres (10 feet)**, turns, returns and sits. Walking aid permitted. **>12 seconds = high fall risk** (sensitivity 77%, positive predictive value 93% in the community; sensitivity 91% but specificity only 22% in inpatients). Even an **untimed** observation is clinically useful.
10. **30-Second Chair Stand Test:** number of full sit-to-stands in 30 seconds with **arms folded across the chest** — measures lower-limb strength and endurance.
11. **Four-Stage Balance Test:** progressive stances — **feet together → semi-tandem → tandem → single leg**, each held for **10 seconds**. Inability to hold **tandem stance for 10 seconds** indicates increased fall risk.
12. **Standing unassisted from a sitting position:** sensitivity **31%**, specificity **90%**; risk rises if the person **cannot stand or takes more than 2 seconds** to do so.
13. **Tinetti Performance-Oriented Mobility Assessment (POMA / Tinetti Mobility Scale): 14 performance tasks** split into **Balance (0–16)** and **Gait (0–12)**, total **0–28**. **Score <24 indicates fall risk** (mean sensitivity 82%, specificity 65%). Takes **10–15 minutes** — its main drawback in a busy clinic.
14. **Berg Balance Scale (BBS): 14 items**, each scored 0–4, total **0–56**. Interpretation: **0–20 = wheelchair-bound; 21–40 = walks with assistance; 41–56 = independent.** Best suited to rehabilitation and stroke settings.
15. **Short Physical Performance Battery (SPPB):** three components — **balance (side-by-side, semi-tandem, tandem), 8-foot (2.44 m) walk, and five repeated chair stands** — each scored 0–4, total **0–12**. Predicts **mortality, nursing-home admission and disability** in a graded fashion.
16. **Dual-task testing:** walking while **counting backwards from 100 by sevens**, TUG **while holding a full glass of water**, or **one-leg stand with eyes closed**. Institutionalised elders who cannot converse while walking are at high fall risk; clinical utility remains uncertain because of variability across studies.
17. Other balance measures compared psychometrically against TUG include the **one-leg (single-limb) stance test** and the **Functional Reach Test**.

D. Supporting and Setting-Specific Tools

18. **Fear of falling:** measured with the **Falls Efficacy Scale–International (FES-I)** and the **Short FES-I (7-item)**; the **ABC (Activities-specific Balance Confidence) Scale** is an alternative. Fear of falling is an **independent predictor** of future falls.
19. **Supporting assessments that function as tools:**
    - **Postural BP** — lying, sitting, and at 1 and 3 minutes standing; **fall of >20 mmHg systolic = orthostatic hypotension**
    - **Medication review tools — STOPPFall**, **Beers criteria**, **STOPP/START**
    - **Home hazard tools — HOME FAST**, occupational-therapist home safety checklists
    - **Vision/hearing** — Snellen chart, whispered voice test, Hearing Handicap Inventory for the Elderly

- **Cognition/mood** — MMSE/MoCA/SLUMS, GDS (dementia and depression both raise fall risk)

20. **Hospital and institutional fall risk scales** (used mainly by nursing services for care planning):
    - **Morse Fall Scale (MFS)** — 6 items, score 0–125; **≥25 moderate, ≥45 high risk**; most widely used in acute care
    - **STRATIFY (St Thomas's Risk Assessment Tool in Falling Elderly Inpatients)** — 5 items; **score ≥2 = at risk**
    - **Hendrich II Fall Risk Model** — 7 items including a **Get-Up-and-Go component** and confusion/depression; **score ≥5 = high risk**
    - **Johns Hopkins Fall Risk Assessment Tool (JHFRAT)**

Rapid Cut-Off Table

Tool	Measures	Cut-off / Score
Gait speed (4 m)	Global function	**<0.6 m/sec abnormal**
Timed Up and Go	Mobility, dynamic balance	**>12 sec = high risk**
4-Stage Balance	Static balance	**Tandem <10 sec = risk**
Sit-to-stand	Lower-limb power	**>2 sec or unable = risk**
Tinetti POMA	Gait + balance	**<24 / 28 = fall risk**
Berg Balance Scale	Balance	**0–56; <45 = fall risk**
SPPB	Lower-limb function	**0–12; ≤8 = impairment**
Stay Independent	Screening	**≥4 = increased risk**
Morse Fall Scale	Inpatient	**≥45 high, ≥25 moderate**
STRATIFY	Inpatient	**≥2 = at risk**
Hendrich II	Inpatient	**≥5 = high risk**
Postural BP	Orthostatic hypotension	**↓ >20 mmHg systolic**

LATEST UPDATES (2023–PRESENT)

1. **NICE NG249 (April 2025) — a major reversal on risk-prediction tools.** The guideline replaces CG161 (2013) and covers everyone aged 65 and over plus people aged 50–64 at higher risk. It **explicitly recommends against using formal falls risk-prediction tools**. Clinicians should instead **directly ask everyone ≥65 (and 50–64 with risk-enhancing conditions such as dementia, arthritis, Parkinson's disease, stroke, diabetes or learning disability) about any fall in the past year**, opportunistically at routine appointments and annual

health checks. Those with defined criteria — including **two or more falls in the last year** — go straight to a **comprehensive falls assessment**; those who fell once without these criteria have their **gait and balance assessed**.

2. **World Falls Guidelines — the three-tier stratification model.** Older adults are placed in **low risk** (education, general exercise), **intermediate risk** (targeted exercise or physiotherapy referral for balance and strength) or **high risk** (**full multifactorial falls risk assessment** with individualised interventions), determined either by opportunistic case-finding or at presentation with a fall. High-risk markers are recurrent falls, injurious falls, frailty and balance problems.
3. **Real-world testing of the World Guidelines algorithm (BMC Geriatrics 2024).** In 392 emergency-department patients assessed by a frailty intervention team, almost all fell into the **high-risk (43.4%) or low-risk (38.0%)** groups with only **4.8% intermediate**, suggesting the intermediate criteria need widening. **Hearing loss and dizziness were significantly associated with falls, whereas vision and balance were not**, supporting formal inclusion of sensory measures in the algorithm.
4. **Proposed algorithm refinement (Age and Ageing, 2025). Broader use of the TUG test with a shorter cut-point of 10 seconds** stratified a community sample into three sizeable, distinct risk groups, allowing more timely and appropriately tailored prevention.
5. **USPSTF 2024 — the accuracy gap in existing tools is now formally acknowledged.** In response to public comment on its 2024 falls statement, the USPSTF agreed that **more accurate risk assessment tools are needed** and listed the development and validation of **primary-care-feasible tools that accurately predict fall risk** as a formal research gap.
6. **Inpatient scales perform poorly in older patients.** A large retrospective study (2024; 303 fall events vs 47,146 non-fall events) found the **Hendrich II model at its standard cut-off ≥5 had sensitivity of only 29.5%, specificity 86.3% and AUC 0.57** — limited discriminative ability. Lowering the cut-off to **≥2 raised the AUC to 0.75** and identified 101 additional fall events, showing that **cut-off selection matters more than the tool itself**. In the same analysis, **STRATIFY failed to predict falls in geriatric wards and in those aged ≥75 years**, and MFS and JHFRAT showed lower sensitivity and specificity than Hendrich II.
7. **Two-stage screening models under evaluation (International Nursing Review, 2025).** The **Morse Fall Scale** showed higher specificity and performed well in surgical wards (AUC 0.887) and in patients aged ≥75 (AUC 0.837), while a **Clinical Judgment Method** offered higher sensitivity for rapid screening; a combined **"screening–confirmation" two-stage model** is proposed but not yet validated.
8. **Indian evidence for tool selection.** LASI wave-1 analyses show that **poor grip strength, slow gait speed, ADL limitation, depression and sensory impairment** are independent determinants of falls in Indian elders — supporting the use of **grip strength, gait speed, GDS and simple sensory screening** as the core, low-cost battery in Indian NPHCE geriatric clinics, where formal balance laboratories are unavailable.

9. **STOPPFall as the standard medication-side tool.** A validated Delphi-derived list of **14 fall-risk-increasing drug classes**, mostly psychotropics, combined with **stepwise practical deprescribing guidance** for medication review.
10. **Emerging technology.** Wearable inertial sensors, smartphone-based TUG, instrumented gait analysis and machine-learning models are being developed to replace static questionnaires, but none has yet entered guideline recommendations.

CURRENT PRACTICE UPDATE

Point	Textbook Position	Current Practice (2023–Present)
Use of prediction tools	STEADI screening with Stay Independent questionnaire, followed by performance testing	**NICE NG249 (2025) advises against formal falls risk-prediction tools**; use a **single direct question about falls in the past year** plus **World Falls Guidelines three-tier stratification**
TUG cut-off	**>12 seconds** = high risk	A **shorter cut-point of 10 seconds** with broader application is proposed to better identify the **intermediate-risk** group
Sensory assessment	Vision and hearing noted as risk factors	**Hearing loss and dizziness** proposed for **formal inclusion in the stratification algorithm** — in some cohorts they outperform vision as predictors
Inpatient scales	Not emphasised as core geriatric tools	Widely used for nursing care plans but **discrimination is poor in the very old**; **STRATIFY fails in geriatric wards and in age ≥75**; cut-off calibration is critical
Purpose of scoring	Identify who is at risk	Shift from **prediction to identification of modifiable risk factors** — the score matters less than what it triggers
Medication review	Table of offending drug classes	Structured **STOPPFall** (14 classes) with stepwise deprescribing guidance
Fear of falling	Recognised as a predictor	Formally measured with **FES-I / Short FES-I**; if unresponsive to exercise, **NICE now recommends cognitive behavioural intervention**

EXAM ONE-LINERS

- **Best single screening question:** "Have you fallen in the last year?"

- **STEADI = Screen → Assess → Intervene** (CDC three-step algorithm).
- **Stay Independent questionnaire: 12 items, score ≥4 = increased risk.**
- **Three Key Questions:** fallen / unsteady / worried about falling.
- **Gait speed <0.6 m/sec = abnormal;** each **0.1 m/sec drop = 7% ↑ fall risk**.
- **TUG >12 seconds = high fall risk** (sens 77%, PPV 93%).
- **TUG distance = 3 metres (10 feet).**
- **Tinetti POMA: 14 tasks, total 28; <24 = fall risk** (sens 82%, spec 65%).
- **Berg Balance Scale: 14 items, 0–56;** 41–56 independent, 21–40 needs assistance, 0–20 wheelchair-bound.
- **SPPB: balance + 8-foot walk + 5 chair stands = 0–12.**
- **Four-Stage Balance Test:** feet together → semi-tandem → tandem → one leg, **10 seconds each**.
- **Morse Fall Scale ≥45 = high risk; STRATIFY ≥2; Hendrich II ≥5.**
- **Orthostatic hypotension = systolic fall >20 mmHg on standing.**
- **STOPPFall = 14 fall-risk-increasing drug classes.**
- **FES-I** measures fear of falling.
- **NICE NG249 (2025): do NOT use falls risk-prediction tools** — ask about falls instead.
- **World Falls Guidelines: low / intermediate / high risk** stratification.
- **No routine laboratory test is part of a falls assessment** — minimum is **lying/standing BP and 12-lead ECG**.

QUESTION 3

Role of Sensor Devices in the Prevention of Falls

15 MARKS | 20 HIGH-YIELD POINTS

A. Rationale and Classification

1. Falls are **multifactorial and episodic**. Clinic-based tests (TUG, Tinetti, Berg) give only a **single snapshot in an artificial setting**. Sensors provide **continuous, objective, real-world ("free-living") data** on gait, balance and behaviour.
2. Conventional disability and rehabilitation scoring systems are **subjective, prone to inter- and intra-observer variability, and time-consuming**, diverting therapists from direct patient care. **Remote sensors record quantitative data objectively.**
3. **Sensor devices serve four distinct roles:**
    - **Fall risk identification** (predicting who will fall)
    - **Fall prevention** (real-time alerting and feedback-driven exercise)
    - **Fall detection** (post-fall alarm to prevent the "long lie")
    - **Rehabilitation monitoring** (objective measurement of progress and adherence)
4. **Classification of sensors:**
    - **Wearable / body-worn: accelerometers, gyroscopes, inertial measurement units (IMUs)** worn on trunk, waist, wrist or lower limb; **smartphone accelerometer**; GPS trackers
    - **Ambient / non-wearable (environmental): PIR motion sensors**, **pressure and piezoelectric sensors** in bed or floor, **acoustic sensors** that identify the specific sound a fall makes, **video-based detectors**, radar, WiFi-sensing and thermal sensors
    - **Multimodal / hybrid systems:** fuse information from several sensor types to improve accuracy

B. Sensors in Fall Risk Assessment

5. Sensors quantify **gait speed, step length, step variability, double-limb support time, postural sway and turning quality** — parameters that predict falls better than a single timed test.
6. **Machine learning technologies** (artificial neural networks, deep learning) integrate **comprehensive clinical assessment data with continuous in-home sensor data** to compute an **individual fall risk index** that is **constantly updated**.
7. A person's **activity pattern** builds a **baseline behavioural model**; any **deviation from that pattern flags functional decline** and rising fall risk before a fall occurs.

8. A simple **accelerometer may be more sensitive than a traditional rating scale** in demonstrating subtle but important gains in mobility — for example, response to a new drug in Parkinson's disease.

C. Sensors in Active Fall Prevention

9. **Real-time context-aware fall risk communication:** the system detects the *intention* to act through an **activity recognition module** and alerts the person **before** a high-risk manoeuvre. *Classic example:* an older adult with **orthostatic hypotension** attempting to rise from a couch is warned **just before standing up**.
10. **Night-time guidance: PIR sensors linked to automatic light faders** illuminate the path to the toilet — targeting **nocturia**, a major and modifiable cause of night falls.
11. **Home safety automation: smart taps and smart stoves** reduce scald, burn and wet-floor hazards.
12. **Adherence monitoring: smart pill dispensers** track drug intake (important for both under-dosing and inadvertent overdose of fall-risk-increasing drugs); **motion, GPS or video sensors** monitor adherence to prescribed exercise programmes and generate warnings when the person defaults.
13. **Virtual reality (VR) platforms** provide visual, auditory and tactile input. By **altering sensory inputs and enhancing corrective postural strategies**, VR augments the adaptive mechanisms that decline with age, promoting **postural stability** in a **safe environment that reduces physical risk and builds patient confidence**.
14. **Video-capture and gaming technology** is much cheaper and widely available — the **Nintendo Wii** with built-in accelerometers and infrared camera is the model. Effective rehabilitation games must **provide feedback, avoid conveying a sense of failure, and match the level of challenge to the player's capability**.
15. **Robotic assist devices** (e.g. the **Honda Walking Assist Device with body-weight support**) can augment recovery in neuromuscular deficits and provide ongoing help with ADLs including stair-climbing. A **"robotic glove" plus VR** optimises motor function after hemiparetic stroke.
16. Sensor-based gait analysis allows **individual tailoring** — for example, accurately characterising a maladaptive gait in knee osteoarthritis to select the **most appropriate orthotic device**.

D. Sensors in Fall Detection and Home-Based Care

17. Immediate detection of a fall **triggers an early attention protocol**, preventing the **"long lie"** (with its risks of dehydration, hypothermia, rhabdomyolysis, pressure ulcers and pneumonia). Even where no injury has occurred, it **generates a warning that the person is at high risk of falling again**.
18. In a **healthcare-enabled Smart Home**, the general practitioner can **access the continuously collected data** to decide on action (face-to-face visit, virtual visit,

treatment change, specialist referral), and **emergency services** can use both the stored data and established communication channels to judge whether to respond.

19. **Remote sensors, wireless data transfer, GPS and mobile computing facilitate early transfer from hospital-based to home-based rehabilitation**, shortening admission and reducing hospital-acquired deconditioning.

E. Limitations and Barriers

20. **Barriers to wider use:**
    - **Cost, lack of technical support, and a weak evidence base for efficacy** are the three principal barriers
    - Older devices were **bulky, heavy, difficult to attach, with poor battery life** and data-storage problems — largely solved by **miniaturisation and wireless transmission**, but **scientific validation in older patients remains limited**
    - The **digital divide** — income, education, age and geography — affects technology penetration; the aged suffer most from this gap
    - Older users have **reduced sensory and motor capability** (high-frequency hearing loss, poor visual acuity, impaired dexterity, reduced strength) and cannot be modelled like young users
    - **Acceptance** is driven by **perceived usefulness and trust**; **perceived risk (privacy) discourages adoption**
    - **Infrastructure needed:** professional needs-assessment before installation, trained installers, an operational backup for remote care, and a **rapid local technical maintenance service**
    - Critically — **bed alarms and physical restraints do NOT reduce falls in hospital**; sensor alarms are **not a substitute for multifactorial intervention**

LATEST UPDATES (2023–PRESENT)

1. **Systematic review of 130 studies (2008–2025) — the field has shifted from detection to prediction.** Three dominant sensor categories are identified: **wearable systems (chiefly inertial measurement units)**, **ambient systems (vision-based, radar, WiFi, LiDAR)** and **hybrid systems fusing multiple data sources**; the notable emerging trend is a move **from reactive fall detection to proactive, pre-impact fall risk prediction**. **Deep learning is now the dominant computational paradigm**, with hybrid **CNN-LSTM and Transformer models** performing best. No modality is universally superior — there is a **trade-off between the continuous, person-centric monitoring of wearables and the non-intrusive, privacy-preserving nature of ambient systems such as radar and LiDAR**.
2. **Hospital sensor technologies — the evidence gap is now formally documented (2025 systematic review, PROSPERO-registered).** A search of five databases and two trial registries for RCTs published 2000–2025 **found no eligible randomised controlled trial assessing the isolated effectiveness of sensor-based technologies for fall prevention in hospitalised older adults**.

Widely implemented technologies such as **bed and chair alarms have shown limited effectiveness and may cause alarm fatigue, reducing staff responsiveness**.

3. **Diagnostic accuracy of wearables is probably over-estimated.** Reviewers have raised concerns of **over-inflated diagnostic accuracy in wearable sensor-based fall risk testing**, attributed to **small sample sizes, questionable modelling decisions and inappropriate validation methodology**. Predictive accuracy is strongly influenced by **sensor location, sensor type, features selected, and the data-processing and modelling technique used** — meaning results are not transferable between systems.
4. **Formal meta-analysis of predictive accuracy (Frontiers in Public Health, 2026).** A systematic review and meta-analysis (search to October 2025) pooled studies in adults ≥60 years across community, residential care and healthcare settings using **accelerometer-, gyroscope- or IMU-equipped wearables placed on trunk, waist or lower limb** to model **future** fall risk — the first quantitative synthesis of an evidence base that had previously been only descriptive.
5. **WHO/ITU engagement with AI-based falls stratification.** The **Topic Group on "Falls among the elderly" of the ITU/WHO Focus Group on Artificial Intelligence for Health** published a formal commentary on fall risk stratification of community-living older people alongside the World Falls Guidelines (*Age and Ageing*, 2023) — signalling that **sensor and AI-derived risk scores are moving towards guideline integration**.
6. **Technology-assisted rehabilitation compared head-to-head.** A network meta-analysis of randomised trials in adults ≥60 years compared **VR exergaming, serious gaming, wearables and telerehabilitation** for balance and functional mobility — the first attempt to rank these modalities against each other rather than against usual care alone.
7. **Scoping review of digital tools 2019–2024.** Digital and smart-home safety technologies are recognised as promising avenues for **timely intervention**, with **AI emergence** driving a new generation of tools; a PRISMA-ScR review synthesised recently published evaluations of digital tools for **fall prevention, detection and risk assessment**.
8. **NICE NG249 (April 2025) — no endorsement of sensor devices.** The guideline recommends **exercise programmes, home hazard assessment by an occupational therapist, medication review and cataract referral**, and mentions **movement sensors** only as equipment that residents in care homes should be taught to use. **No sensor technology is recommended as a fall-prevention intervention.**
9. **Indian relevance.** Sensor-based approaches are attractive in India because of **high smartphone penetration and a shortage of trained falls-clinic personnel** — a **smartphone accelerometer-based TUG or gait speed** can be delivered at NPHCE geriatric clinics and health and wellness centres at negligible cost. Barriers are the **rural digital divide, electricity and connectivity gaps, absence of reimbursement under AB-PMJAY for assistive monitoring technology, and low digital literacy in the oldest old**.

10. **Practical position in 2026.** Sensors are currently **best supported for objective risk stratification, rehabilitation feedback and post-fall detection — not** as stand-alone preventive interventions. Every consensus document still places **progressive balance and functional exercise plus multifactorial risk-factor modification** as the core of prevention, with technology as an **adjunct**.

CURRENT PRACTICE UPDATE

Domain	Textbook Position	Current Practice (2023–Present)
Primary sensor role	Objective measurement in rehabilitation and fall detection	Shift **from reactive detection to proactive, pre-impact fall prediction** using deep learning
Dominant technology	Accelerometers, video capture, VR, Nintendo Wii–type gaming	**IMUs, radar, LiDAR, WiFi-sensing and hybrid multimodal systems**; **CNN-LSTM and Transformer models** now standard
Evidence base	"Lack of a strong evidence base for efficacy" identified as a barrier	Barrier persists — **no RCT isolates sensor effectiveness in hospitalised older adults (2025)**; reported accuracy is likely **over-inflated** by small samples and weak validation
Bed/chair alarms	Bed alarms not associated with reduced falls	Confirmed — **limited effectiveness plus alarm fatigue**, which itself reduces staff responsiveness
Guideline status	Emerging technology, promising	**Not recommended as a preventive intervention by NICE NG249 (2025)**; WHO/ITU AI Focus Group working towards eventual integration
Privacy	Not emphasised	Now a **central design consideration** — ambient radar/LiDAR favoured for privacy preservation over video
Where sensors clearly help	Rehabilitation progress measurement	**Objective risk stratification, exercise feedback and adherence, and post-fall alerting to prevent the long lie**

EXAM ONE-LINERS

- Sensors give **continuous, objective, real-world data** — clinic tests give only a **snapshot**.
- **Four roles:** risk prediction, active prevention, fall detection, rehabilitation monitoring.

- **Wearable = accelerometer, gyroscope, IMU, smartphone.**
- **Ambient = PIR, pressure/piezoelectric, acoustic, video, radar, WiFi, LiDAR, thermal.**
- **Multimodal/hybrid systems** fuse several streams for higher accuracy.
- **Machine learning + clinical assessment + in-home sensors = continuously updated individual fall risk index.**
- **Context-aware alerting:** warns the patient with orthostatic hypotension *before* they stand up.
- **PIR sensors + automatic light faders** = night-time guidance for nocturia-related falls.
- **Smart pill dispensers** monitor drug adherence; **motion/GPS sensors** monitor exercise adherence.
- **Deviation from the baseline behavioural model = early marker of functional decline.**
- **Fall detection prevents the "long lie"** and flags high risk of recurrence.
- **VR promotes postural stability** by altering sensory input and augmenting corrective strategies in a safe environment.
- **Nintendo Wii** = accelerometer + infrared camera; rehab games must give feedback and avoid a sense of failure.
- **Honda Walking Assist Device** = robotic gait assistance.
- **Three barriers: cost, lack of technical support, weak evidence base.**
- **Digital divide** — income, education, age, geography.
- **Adoption driven by perceived usefulness and trust; discouraged by perceived risk.**
- **Bed alarms and restraints do NOT prevent falls** — alarm fatigue is a real harm.
- **NICE NG249 (2025) does not recommend any sensor device for fall prevention.**
- **Exercise remains the intervention with the strongest evidence** — technology is an **adjunct, not a substitute**.

QUESTION 4

Prevention of Fractures in the Elderly

15 MARKS | 20 HIGH-YIELD POINTS

A. Concept and Burden

1. A **fragility (osteoporotic) fracture** results from **low-energy trauma — typically a fall from standing height or less**. It is the product of **two independent factors: the fall (impact) and bone made fragile by ageing (osteoporosis)**. Prevention must therefore address **both**.
2. The **commonest sites are hip, forearm (distal radius) and vertebrae**; humerus and ribs follow. **Maintenance of musculoskeletal health during ageing is a key determinant of functional ability.**
3. In elderly patients, **only 3% of hip fractures are pathological** (metastasis, myeloma, bone cysts, Paget's disease); **over half of patients are osteoporotic and almost all have osteopenia**.
4. After a proximal femoral fracture, in the **first year: 56% fall again, 28% have repeated falls, 12% sustain a new fracture, and 5% fracture the opposite hip** — making **secondary prevention mandatory**.
5. **Three pillars of fracture prevention:**
    - **Assessing fall risk and delivering preventive interventions**
    - **Protecting the trochanteric region (hip protectors)**
    - **Assessing and treating osteoporosis**

B. Fracture Risk Assessment

6. **FRAX** integrates clinical risk factors **with or without BMD** to compute the **10-year probability of hip fracture and of major osteoporotic fracture**. It can be modulated by **BMD, trabecular bone score (TBS), glucocorticoid therapy, fall history, type 2 diabetes and hip axis length**.
7. **Major risk factors: age, female sex, low BMI, prior fragility fracture, parental hip fracture, height loss >4 cm, secondary osteoporosis, glucocorticoid therapy, excess alcohol and smoking**.
8. **Diagnosis:** osteoporosis can be diagnosed **clinically in a patient with a minimal-trauma fracture without any other test**. Otherwise **DXA T-score ≤ −2.5**. Note that **after age 80 a woman with BMD normal for her age already has a T-score of −2.5**, so **DXA for diagnostic purposes alone is chiefly indicated in women under 75**.
9. **Exclude secondary osteoporosis** in every fragility fracture: full history of **corticosteroids and alcohol**, plus **complete blood count, liver and kidney**

function, serum calcium and phosphorus (mg/dL), 25(OH) vitamin D (ng/mL) and TSH. In the elderly the **dominant secondary cause is hyperthyroidism**; also consider **malnutrition, low body weight, hypogonadism and hyperparathyroidism**.

C. Universal Non-Pharmacological Measures

10. **For all patients, regardless of risk category:**
    - **Calcium 800–1000 mg/day** (diet preferred)
    - **Protein ≥1 g/kg body weight/day**
    - **Vitamin D ~800 IU/day**, with adequate **sun exposure**
    - **Daily weight-bearing physical activity**
    - **Fall-prevention measures**
    - **Education** on osteoporosis, medication, diet and exercise
11. A structured exercise programme should run for **at least 50 hours**, be **progressive**, and include **strength and balance components**. It reduces falls, but **the effect on fracture risk itself remains uncertain**.
12. **Calcium + vitamin D combined had a clear benefit in elderly women living in nursing homes.** However, a meta-analysis of **33 randomised trials with 51,145 participants (Zhao et al., JAMA 2017)** showed **no reduction in fracture risk in community-dwelling older adults** with calcium, vitamin D, or both.

D. Mechanical (Fall-Related) Protection

13. **Hip protectors:** most upper-femoral fractures follow a **sideways fall directly onto the greater trochanter**. Two rigid shells or foam pads held over the trochanters **absorb the shock and disperse energy into soft tissue**.
14. **Cochrane Review (2014, 19 studies, ~17,000 people):** in nursing/residential care, **RR 0.82 (95% CI 0.67–1.00)** — a small reduction in hip fracture risk (moderate quality). In the community, **RR 1.15 (0.84–1.58) — little or no effect**. **Poor acceptance and adherence is the major barrier**, particularly in the cognitively impaired.
15. **Multifactorial fall risk assessment and intervention** — exercise, medication review, home hazard modification, vision correction, cardiac pacing for carotid sinus hypersensitivity — is inseparable from fracture prevention. **Minimum work-up in a faller: lying/standing BP and a 12-lead ECG.**

E. Pharmacological Prevention

16. **Treatment pathway by risk category:**
    - **Low risk:** reassurance, lifestyle advice; consider **menopausal hormone therapy (MHT) or SERMs**

- **High risk: oral bisphosphonate or another inhibitor of bone resorption** (denosumab)
- **Very high risk: anabolic agent (teriparatide, abaloparatide, romosozumab) followed by an inhibitor of bone resorption**; consider **local osteo-enhancement procedure (LOEP)** if hip fracture risk is very high

17. **Bisphosphonates — first line.** Reduced hip fractures demonstrated with **risedronate, pamidronate and zoledronic acid**. **NNT ≈ 200 to prevent one hip fracture. Zoledronic acid 5 mg IV once yearly reduces clinical fracture by 33%, vertebral fracture by 77% and peripheral fracture by 25% — vitamin D must be given beforehand**. Adverse effects: **flu-like syndrome after the first infusion (1 in 3)**, higher incidence of **atrial fibrillation**, GI upset with oral forms, and **rare osteonecrosis of the jaw**. Gastro-resistant risedronate improves adherence.

18. **Other agents:**
    - **Denosumab** — fully human monoclonal antibody to **RANKL**; blocks RANK binding, inhibits osteoclast development and activity; **subcutaneously twice yearly**; reduces **vertebral, non-vertebral and hip** fractures (FREEDOM). **Discontinuation may cause multiple vertebral fractures — follow with a bisphosphonate for 1–2 years.**
    - **Teriparatide** — reduces new vertebral and non-vertebral fracture **independent of initial T-score, age and number of fractures**; bone mass gain is dose-dependent.
    - **Romosozumab** — monoclonal antibody to **sclerostin**; increases formation and decreases resorption. **12 months of romosozumab followed by alendronate gave significantly lower fracture risk than alendronate alone (ARCH; Saag et al.)**; FDA-approved April 2019 for high-risk postmenopausal women.
    - **Limited in the elderly: raloxifene does not reduce femoral neck fracture**; **SERMs must be avoided after femoral neck fracture** (thromboembolic risk); **HRT** carries breast cancer, thromboembolic, stroke and coronary risks; **strontium ranelate** caused fatal **DRESS** and thromboembolism; **calcitonin** has weak efficacy.

19. **Duration and monitoring:**
    - Check **bone turnover markers at 3–6 months** to verify compliance with resorption inhibitors
    - Reassess **after 3 years of IV or 5 years of oral bisphosphonate**: **low-risk patients may discontinue for 2 years (reconsider yearly); high-risk patients continue**
    - **Whatever the drug, it must be combined with adequate calcium and vitamin D intake**

20. **Life expectancy and systems of care:**
    - Unlike cancer screening, **the effectiveness of fracture prevention INCREASES with advancing age** — a **90-year-old has a higher lifetime fracture risk and a lower NNT than a 70-year-old**. Economic models suggest treatment is **cost-effective even with a life expectancy as short as two years**.
    - **Drug benefit appears at 6–12 months; the benefit of effective fall prevention may be immediate.**

- **Fracture Liaison Services (FLS)** provide routine, coordinator-based assessment and management of **all postmenopausal women and all men with a fragility fracture**; the **IOF "Capture the Fracture"** campaign sets internationally endorsed best-practice standards to break the fragility fracture cycle. **Orthogeriatric co-management** models improve outcomes after hip fracture.

LATEST UPDATES (2023–PRESENT)

1. **ACP Living Clinical Guideline (Annals of Internal Medicine, 2023) — bisphosphonates confirmed as first line. Bisphosphonates are strongly recommended as initial pharmacologic treatment in postmenopausal women (high-certainty evidence)** and conditionally suggested in men. **Denosumab is second line** for those with contraindications or adverse effects. **Romosozumab or teriparatide followed by a bisphosphonate is suggested only in women at very high fracture risk.** For **women over 65 with osteopenia, an individualised decision** on starting a bisphosphonate is advised. The 2023 update **removed the earlier 2017 ambiguity that allowed either bisphosphonates or denosumab as first choice**, and as a "living guideline" it is revised as new data emerge.
2. **NOGG UK guideline 2024 — HRT returns as first-line in a defined group.** Specific 2024 updates include **fracture risk assessment by ethnicity, and in Parkinson's disease, Down's syndrome and lower-limb amputation**; the **definition of very high fracture risk has been clarified**; and **HRT is now recommended as a first-line option in younger postmenopausal women with high fracture risk and low baseline risk of adverse events**. Three anabolic agents — **teriparatide, abaloparatide and romosozumab** — are now available, with **teriparatide licensed for a once-only treatment course**; **no evidence yet supports one specific sequence**, so specialist input is essential.
3. **RACGP / Healthy Bones Australia 2024. Widespread population-based screening is not recommended**; **FRAX-based absolute risk assessment** is encouraged. It is essential to identify patients at **"imminent" or "very high" fracture risk — the group in whom early osteoanabolic therapy should be considered**. Critically, **calcium and vitamin D supplementation reduces fracture risk only in individuals who are actually deficient, not in healthy non-institutionalised people**. The guideline also addresses **rebound vertebral fracture after denosumab cessation** and formally **removes strontium as a therapy**.
4. **ASBMR/BHOF goal-directed treatment position statement (JBMR, 2024).** Major guideline bodies (AACE, Endocrine Society, The Menopause Society, BHOF, ESCEO/IOF, NOGG) have all now adopted a **new "very high fracture risk" category**. A subset of these patients is at **imminent risk for the two years following an incident fracture** or after multiple prior fractures — the rationale for **treat-to-target, anabolic-first therapy**.
5. **USPSTF draft statement (December 2024) — vitamin D and calcium downgraded.** A **Grade D (recommend against)** was proposed for **vitamin D, with or without calcium, to prevent fractures** in postmenopausal women and men ≥60 years, and a **Grade D for vitamin D to prevent falls** in the same group.

This applies only to **community-dwelling** individuals and **does not apply to those with diagnosed osteoporosis, vitamin D deficiency, or malabsorption**.

6. **Cochrane Review 2025 (care facilities, CD016064) — the institutional exception holds.** In care facilities, **vitamin D supplementation, exercise, and staff-engaged individually tailored multifactorial interventions probably reduce falls** — reinforcing that **nursing-home residents remain a group who benefit from vitamin D**, in contrast to community dwellers.
7. **Short-course sequential therapy after hip fracture (2025).** Current guidelines recommend **anabolic agents first-line in very-high-risk patients including recent hip fracture, followed by antiresorptive therapy**, but real-world implementation is limited and the optimal anabolic duration is uncertain. A study of **113 osteoporotic hip fracture patients** evaluated **3–6 months of teriparatide or romosozumab followed by denosumab** versus anabolic monotherapy, since **shorter anabolic courses may be more feasible in frail elderly patients** than the conventional 18–24 months.
8. **Indian guidance — ISBMR position statement. Osteoporotic fractures occur at an earlier age in Indians than in the West, so screening should begin earlier. FRAX may underestimate future fracture risk in the Indian population and still requires validation.** Maintaining optimal **serum 25(OH) vitamin D** usually requires **regular supplementation**, and **bisphosphonates remain the first choice in most cases**. For **vertebral fractures, teriparatide is recommended as initial anabolic treatment for 24 months followed by an antiresorptive**; **zoledronic acid or denosumab are alternatives, with zoledronic acid preferred**. A **Markov microsimulation cost-effectiveness analysis of FRAX-based intervention thresholds in Indian women (Osteoporosis International, 2025)** has now been published, and **FRAX without DXA** is highlighted as usable where densitometry is unavailable. Maintenance **vitamin D3 of 1000–2000 IU daily** is typically required.
9. **Indian service gaps. DXA availability is concentrated in urban tertiary centres**; **Fracture Liaison Services are few and reimbursement is limited**. Practical Indian strategy: **clinical diagnosis on a minimal-trauma fracture, FRAX without BMD, generic alendronate or annual zoledronic acid, calcium and vitamin D correction, and fall prevention through NPHCE geriatric clinics and district-level rehabilitation units**.
10. **NICE NG249 (April 2025).** Vitamin D supplements are **not recommended specifically to reduce falls**, though people are encouraged to follow standard advice on vitamin D **to maintain bone and muscle health** — separating the **bone** indication from the **falls** indication.

CURRENT PRACTICE UPDATE

Domain	Textbook Position	Current Practice (2023–Present)
First-line drug	Oral bisphosphonate for high risk; denosumab an equal alternative	**Bisphosphonates are unambiguously first line (ACP 2023, strong, high-**

		certainty); **denosumab is second line** for intolerance or contraindication
Anabolic therapy	Consider for very high risk, followed by antiresorptive	Same principle, but **"very high risk" and "imminent risk (2 years post-fracture)" are now formally defined categories**; **short 3–6 month anabolic courses** being trialled in frail hip-fracture patients
Calcium and vitamin D	Give to all; benefit shown in nursing-home women, none in community dwellers	Sharpened — **effective only in those who are actually deficient or institutionalised**; **USPSTF draft Grade D against routine use in community dwellers**
HRT	Effective but limited by breast cancer, thromboembolic and cardiovascular risk	**NOGG 2024 reinstates HRT as first-line in younger postmenopausal women** with high fracture risk and low adverse-event risk (not applicable to the very old)
Strontium ranelate	Discussed with DRESS and thromboembolic cautions	**Formally removed as a therapy** (RACGP 2024)
Denosumab stopping	Discontinuation may cause multiple vertebral fractures — follow with bisphosphonate	Unchanged and now emphasised as **"rebound vertebral fracture" — never stop denosumab without antiresorptive cover**
Screening	DXA for diagnosis mainly useful in women <75	**Population-based screening not recommended**; **case-finding with FRAX ± DXA** is the model; **in India, screening should start at a younger age**
Hip protectors	Small benefit in care homes, none in community	Unchanged — **adherence remains the limiting factor**
Systems of care	Fracture Liaison Services and "Capture the Fracture"	Now the **central quality metric**; the global **treatment gap after fragility fracture remains the biggest unaddressed problem**

EXAM ONE-LINERS

- **Fragility fracture = fall + fragile bone.** Prevent **both**.
- **Three pillars:** fall risk intervention, hip protectors, osteoporosis treatment.
- **Commonest sites: hip, forearm, vertebra.**
- **Only 3% of elderly hip fractures are pathological.**
- **After hip fracture, in 1 year: 56% fall, 28% recurrent falls, 12% new fracture, 5% fracture the other hip.**
- **FRAX = 10-year probability of hip and major osteoporotic fracture.**

- **T-score ≤ −2.5 = osteoporosis;** but a minimal-trauma fracture is diagnostic on its own.
- **DXA for diagnosis alone is mainly indicated in women <75.**
- **Commonest secondary cause of osteoporosis in the elderly = hyperthyroidism.**
- **Universal measures: calcium 800–1000 mg/day, protein ≥1 g/kg/day, vitamin D ~800 IU/day, weight-bearing exercise.**
- **Exercise programme should be ≥50 hours, progressive, strength + balance.**
- **Zoledronic acid 5 mg IV yearly: ↓ clinical fracture 33%, vertebral 77%, peripheral 25%** — correct vitamin D first.
- **NNT ≈ 200 with oral bisphosphonate to prevent one hip fracture.**
- **Denosumab = anti-RANKL, 6-monthly SC; never stop without bisphosphonate cover.**
- **Romosozumab = anti-sclerostin; 12 months then alendronate (ARCH).**
- **Raloxifene does not reduce hip fracture; SERMs contraindicated after femoral neck fracture.**
- **Bisphosphonate holiday after 3 years IV / 5 years oral in low-risk patients only.**
- **Hip protectors: RR 0.82 in care homes; no benefit in the community.**
- **Fracture prevention becomes MORE effective with advancing age** — cost-effective with life expectancy as short as 2 years.
- **Fracture Liaison Service + "Capture the Fracture" = secondary prevention model.**

QUESTION 5

Pressure Sores: Stages, Common Sites, Prevention and Treatment

15 MARKS | 20 HIGH-YIELD POINTS

A. Definition, Terminology and Pathogenesis

1. A **pressure injury** is **localised damage to the skin and underlying tissue, usually over a bony prominence** (sacrum, heels), caused by **disruption of blood supply**, preventing oxygen and nutrients from reaching cells. **"Pressure injury" is a more accurate term than "pressure ulcer"** because **some presentations are not open ulcers**.
2. **Incidence varies widely by setting:** acute care **0.4–38%**, long-term care **2.2–23.9%**, home care **0–17%**. About **25% of pressure ulcers are acquired intra-operatively**.
3. **Four mechanical forces operate together:**
   - **Pressure** over a bony prominence → tissue deformation and ischaemia
   - **Shear** — head of bed elevated, torso slides down, transmitting force to the sacrum; stretches and angulates subcutaneous tissue
   - **Friction** — repeated movement of skin across sheets/clothing, which increases shear
   - **Moisture** — sweat, urine, faeces, bathing

   Together these cause **thrombosis of small blood vessels**, undermining then destroying skin. **Loose, folded skin** (loss of subcutaneous fat, dehydration) worsens shear.
4. **The area of damage beneath the ulcer is often much larger than the visible ulcer** — the "iceberg" effect — because pressure and shear are transmitted deep into subcutaneous tissue.

B. Staging (NPIAP System)

5. Pressure injuries are numerically staged **1, 2, 3 or 4 — using Arabic, not Roman, numerals — only if the depth of the wound can be visualised**.
6. **Stage 1 — Non-blanchable erythema of intact skin.** Acute inflammatory response limited to epidermis. Irregular area of **erythema, induration and oedema**, which may feel **firm or boggy**. **Redness persists beyond 30 minutes and does not blanch on pressure.** In **darker skin the colour may be red, blue or purple**. **Skin is unbroken.**
7. **Stage 2 — Partial-thickness loss.** Inflammation extends **through the dermis to the junction of subcutaneous fat**. Appears as a **blister, abrasion or shallow ulcer with distinct edges**. Early fibrosis and pigment change occur.

8. **Stage 3 — Full-thickness skin loss extending through subcutaneous fat, down to but not through the underlying fascia.** Presents as a **crater**, often with **undermining** of adjacent tissue. Base may be infected with **necrotic, foul-smelling tissue**.
9. **Stage 4 — Extension through deep fascia, with bone visible at the base. Undermining is even more common and sinus tracts may be present. Osteomyelitis and septic arthritis can occur.**
10. **Two additional categories when depth cannot be seen:**
    - **Unstageable pressure injury** — wound base obscured by **eschar and/or necrotic tissue**; **can be staged once debrided**
    - **Deep tissue pressure injury (DTPI) — skin intact but deep red, purple or maroon**, or a **haemorrhagic blister**, indicating damage in deeper tissue
11. **Special situations:**
    - **Medical device–related pressure injuries** — from urinary catheters, rectal tubes, oxygen tubing, NIV masks; the wound **conforms to the shape of the device**. **These ARE staged.**
    - **Mucous membrane pressure injuries must NOT be staged** — only documented.
12. **Reverse staging is inaccurate for assessing healing.** Use the **PUSH tool (Pressure Ulcer Scale for Healing)**, which scores **size, exudate amount and tissue type** and is sensitive to change over time.

C. Common Sites

13. **More than 90% of pressure ulcers occur in the lower body** — principally the **sacral and coccygeal area, ischial tuberosities and greater trochanter**, along with the **heels**.
14. **Site varies with posture:**

Position	Pressure points
Supine	**Occiput**, scapulae, elbows, **sacrum**, coccyx, buttocks, **heels**
Lateral	Ear, shoulder, **greater trochanter**, ribs, **malleoli**, knees, bent lower leg
Sitting	**Ischial tuberosities**, coccyx, sacrum, back of knees, heels
Prone	Forehead, chin, cheeks, chest/breasts, iliac crests, genitalia, knees, dorsum of feet

Easily overlooked sites: **gluteal cleft, elbows, malleoli, wrists**.

D. Risk Factors and Risk Assessment

15. **Risk factors:** being **bed- or chair-bound**, **unable to reposition without assistance**, difficulty with ambulation, **history of stroke**, **faecal incontinence**, **low**

body weight, **lymphopenia**, difficulty with independent feeding, **impaired nutritional intake**, **non-blanchable erythema of intact skin (i.e. an existing Stage 1 injury)** and **dry sacral skin**. **Advanced age itself is NOT an independent risk factor** — it is the coexisting impairments in mobility, nutrition and skin health that matter.

16. **Risk-assessment tools: Norton, Braden and Waterlow scales.** Current recommendation is to apply a tool **on admission to a facility and regularly thereafter**. Because risk is multifactorial, **multidisciplinary multifactorial intervention** is effective — as with falls and delirium.

E. Prevention

17. **General principles of skin care in the immobile patient:**
    - **Identify patients at risk**
    - **Decrease pressure, friction and skin folding**
    - **Keep skin clean and dry**; **moisturise dry sacral skin**
    - **Avoid excessive bed rest — optimise and encourage function**
    - **Avoid over-sedation**
    - **Nutrition and hydration: 30–35 kcal/kg/day, protein 1–1.5 g/kg/day, fluid 1 mL/kcal**; dietitian referral for those at risk
18. **Support surfaces:**
    - **Non-powered** — mattresses or overlays filled with **air, water, gel or foam**
    - **Powered** — mechanically vary the pressure beneath the patient, reducing the *duration* of applied pressure; **far more costly**
    - **Specialised foam and sheepskin overlays reduce incidence compared with a standard hospital mattress**; the choice between powered and non-powered is **not clear**
    - **Mattress overlays on operating tables reduce postoperative pressure injuries**
    - **Repositioning: at least 2-hourly on a regular mattress, at least 4-hourly on a pressure-reducing mattress**
19. **Avoidability: Not all pressure injuries are avoidable, particularly at the end of life** — but **prevention protocols must be in place before concluding that an injury was unavoidable**.

F. Treatment

20. **Stage-wise management. Overarching principle — managing the underlying contributing factors is more important than topical or adjunctive therapy.** Standard local care of a **healable** ulcer must satisfy three criteria: **moisture balance, bacterial balance and debridement.**
    - **Stages 1–2:** continue **all** preventive measures to stop progression. Clean with **warm normal saline or water**; avoid pressure and moisture; cover open wounds

with an **occlusive dressing** chosen by wound condition, drainage and infection; use antibiotics **very judiciously**; manage pain. **No single dressing has been shown superior to another.**

- **Stage 3: debride necrotic tissue** (autolytic, chemical, mechanical, sharp or surgical); **irrigate 2–3 times daily**; **undermine and remove eschar** if it conceals necrotic or infected tissue. **Routine wound cultures are not warranted** — they grow multiple organisms, miss anaerobes and reflect colonisation. **Systemic antibiotics do not reach adequate concentration in the ulcer**; reserve them for **confirmed bacteraemia, sepsis, osteomyelitis or cellulitis**.
- **Stage 4: tissue biopsy for routine and anaerobic culture**; **systemic antimicrobials are often necessary**; **surgical/plastic consultation for repair**. If septic, use **broad-spectrum cover for anaerobes, Gram-negatives and *Staphylococcus aureus***.
- **Diagnosing infection: increasing pain in a chronic wound is the strongest predictor (LR 11–20)**. The "classic signs" — **purulent exudate, erythema, heat, oedema — and foul odour are NOT helpful**. Use a **quantitative swab by the Levine technique** (rotate swab over a **1 × 1 cm area for 5 seconds** with enough pressure to express fluid): **positive LR 6.3, negative LR 0.47**.
- **Adjunctive therapies** (negative-pressure wound therapy, hyperbaric oxygen, laser, electrotherapy, electromagnetic therapy, therapeutic ultrasound): **no good evidence that they improve healing.**
- **Non-healable injuries / skin failure:** where underlying conditions are irreversible or goals are comfort, prioritise **reducing infection risk and controlling pain**. Wounds appearing on bony prominences during overall decline are termed **skin failure**, not pressure injuries.
- **Documentation must include:** type and duration; **size as length × width × depth in cm**; **colour as percentage red (granulation) / yellow (slough) / black (eschar)**; **exudate** (serous, serosanguinous, sanguinous, purulent); **odour assessed after cleaning**; periwound tissue; and **undermining**.

LATEST UPDATES (2023–PRESENT)

1. **Fourth edition of the International Pressure Injury Guideline (NPIAP/EPUAP/PPPIA, 2025) — a rolling, online guideline.** Partner organisations NPIAP, EPUAP and PPPIA are releasing the fourth edition with an **expanded online format and staged release of chapters**, plus refreshed quick-reference materials, governed by a **Guideline Governance Group led by Professor Janet Cuddigan** with **over 240 clinical experts and consumer representatives** using **evidence-to-decision frameworks**. Chapters live include **Definition & Etiology, Preventive Skin Care, Nutrition, Repositioning, Support Surfaces, Seating, Heel Pressure Injuries and Device-Related Pressure Injuries**, with Pressure Injury Risk and Skin & Tissue Assessment to follow.
2. **The "2-hourly turn" is no longer mandatory.** A meta-analysis of two RCTs in aged-care settings found **2-hourly repositioning was associated with a non-significant and counter-intuitively *higher* rate of pressure injuries than 3-**

hourly (1.3% vs 0.3%, RR 4.06, 95% CI 0.87–18.98). The guideline therefore suggests that **either 2-hourly or 3-hourly repositioning may be used for most at-risk individuals, provided an appropriate pressure-redistribution support surface is in place** (conditional recommendation, very low certainty). However, it **suggests against routinely extending intervals to 4, 5 or 6 hourly**, since a meta-analysis of five RCTs showed fewer injuries with 2-hourly turning (4.6% vs 5.7%, RR 0.89).

3. **The harms of over-frequent turning are now formally recognised.** A Cochrane review documented **sleep disruption, musculoskeletal pain, wound pain and more injuries to care staff** with shorter intervals. **Staff compliance is higher at 3- and 4-hourly intervals (90–95%) than at 2-hourly (80%)**, and two economic analyses showed **extending from 2-hourly to 3-hourly was more cost-effective** when tolerated.
4. **Positioning specifics with new evidence. 30-degree lateral positioning** is suggested over greater angles (**avoid the 90-degree lateral position**), modified to **40 degrees in individuals with high BMI** who cannot maintain it. **Head-of-bed elevation should be kept at 30 degrees or lower** — restricting elevation gave **68 fewer pressure injuries per 1,000 (11.1% vs 18.9%, OR 0.59, 95% CI 0.36–0.97)** — but a **lower head-of-bed increased ventilator-associated pneumonia (OR 2.15) and gastric reflux (OR 1.85)**, so clinical needs may override.
5. **Micromovements for the haemodynamically unstable.** It is now **good practice to use frequent, small, incremental shifts (micromovements) in critically ill individuals too unstable for a regular repositioning regimen**, with a **trial of full body repositioning at least every 8 hours** to see if regular turning can resume, allowing **at least ten minutes for haemodynamic equilibrium** after each position change.
6. **Early mobilisation is now an evidence-based pressure injury intervention.** A meta-analysis of six RCTs and six comparative studies showed **early mobilisation significantly reduced pressure injuries (5.2% vs 6.9%, RR 0.75, 95% CI 0.61–0.92) — 17 fewer per 1,000**. It also prevented **ICU-acquired weakness (RR 0.49), deep vein thrombosis (RR 0.16) and pneumonia (RR 0.39)**, with a small increase in falls. The staged protocol is: **passive range of movement → dangling limbs → sitting out of bed → standing → walking**, 2–3 times daily as tolerated.
7. **Wearable movement sensors — a conditional new option.** Three studies of body-worn movement sensors showed a **non-significant lower rate of pressure injuries (0.7% vs 6.9%, RR 0.15, 95% CI 0.02–1.08)**; sensors are **suggested to help evaluate repositioning needs where resources permit**, but must **complement rather than replace clinical judgement**, and **may not detect offloading at the head, ears, elbows and heels**.
8. **Prophylactic dressings and refined skin assessment.** The 2025 Preventive Skin Care chapter suggests **a multilayered soft silicone foam dressing on the sacrum and heels for individuals at high risk, where resources permit** (conditional, very low certainty), and specifies that **skin assessment must include the scalp and under hair**, using **wide-toothed combs and**

moisturising products for curly, textured or tightly coiled hair to allow visual inspection. **Subepidermal moisture (SEM) measurement and skin temperature are now recommended within skin assessment**, as SEM detects inflammation and tissue damage **predictive of Stage 1 and greater injuries, especially at the sacrum**, and **high-resolution ultrasound is valuable for deep tissue injury, particularly in darker skin tones**.

9. **Device-related injuries are a growing share of the burden.** In acute care, **medical device–related pressure injuries account for more than 30% of all hospital-acquired pressure injuries**, largely from prolonged prone positioning with ventilation devices in the mouth and nose; **sedation, oedema and increased skin moisture** are contributory.
10. **Indian data — lower prevalence but more advanced disease.** A point-prevalence study in a north Indian tertiary hospital using the **EPUAP minimum data set** found an overall prevalence of **7.8%**, with the **sacrum and heel most commonly affected** and, strikingly, **Grade III ulcers being the commonest presentation (42.8%)** — indicating **late detection**. **ICU prevalence was 24.3%**, and injuries were more frequent in patients hospitalised **more than a month**. An earlier Varanasi study found a prevalence of **4.94%**, with **anaemia, malnutrition and diabetes** as key risk factors and **morbidity in long-stay neurology wards as high as 40.9%**. Practical Indian implications: **home-based bedbound care is the dominant setting**, **caregiver education is the single highest-yield intervention**, and **low-cost foam or sheepskin overlays with disciplined repositioning** outperform expensive powered mattresses that most families cannot afford.

CURRENT PRACTICE UPDATE

Domain	Textbook Position	Current Practice (2023–Present)
Repositioning frequency	At least **2-hourly** on a regular mattress, **4-hourly** on a pressure-reducing mattress	**2-hourly or 3-hourly are both acceptable** on an appropriate support surface; **individualised regimens replace fixed schedules**; do **not** routinely extend to 4–6 hourly
Rationale for change	More frequent turning assumed better	Frequent turning causes **sleep disruption, pain and staff injury**, and **compliance falls at 2-hourly intervals**; 3-hourly is more cost-effective
Support surfaces	Powered vs non-powered choice unclear	Unchanged — but explicitly stated that **no support surface can entirely replace repositioning**
Positioning	Not specified in detail	**30-degree lateral position** (avoid 90-degree); **head-of-bed ≤30 degrees**, balanced against aspiration and VAP risk
	Not addressed	

Unstable patients		**Micromovements** are formal good practice; retry full turning **8-hourly**
Mobilisation	"Avoid excessive bed rest; optimise function"	Now supported by meta-analysis — **early staged mobilisation significantly reduces pressure injuries (RR 0.75)**
Prophylactic dressings	Not recommended	**Multilayered soft silicone foam on sacrum and heels** suggested for high-risk patients
Skin assessment	Visual inspection of bony prominences	Extended to **scalp and under hair**; **SEM and skin temperature measurement** added, especially valuable in **darker skin tones** where Stage 1 and DTPI are missed
Technology	Not addressed	**Wearable movement sensors** conditionally suggested as an adjunct — never a replacement for clinical judgement
Adjunctive therapies	No good evidence they improve healing	**Unchanged** — NPWT, hyperbaric oxygen, laser, ultrasound still not supported for routine use

EXAM ONE-LINERS

- **"Pressure injury" is preferred to "pressure ulcer"** — not all are open ulcers.
- **Four forces: pressure, shear, friction, moisture.**
- **Shear** occurs when the head of the bed is elevated and the torso slides down onto the sacrum.
- **Damage below the ulcer is larger than the visible ulcer.**
- **Stage 1 = non-blanchable erythema, intact skin**, persisting beyond 30 minutes.
- **Stage 2 = partial thickness** — blister, abrasion or shallow ulcer.
- **Stage 3 = full thickness through fat, down to but not through fascia** — crater with undermining.
- **Stage 4 = through deep fascia, bone visible** — sinus tracts, osteomyelitis.
- **Unstageable = base obscured by eschar; DTPI = intact skin, deep purple/maroon or haemorrhagic blister.**
- **Device-related injuries ARE staged; mucosal injuries are NOT.**
- **Never reverse-stage** — use the **PUSH tool** (size, exudate, tissue type).
- **>90% occur in the lower body: sacrum/coccyx, ischial tuberosity, greater trochanter** (plus heels).
- **Risk tools: Norton, Braden, Waterlow** — on admission and regularly.

- **Advanced age is NOT an independent risk factor.**
- **Nutrition target: 30–35 kcal/kg, protein 1–1.5 g/kg, fluid 1 mL/kcal.**
- **Turn 2-hourly on a regular mattress, 4-hourly on a pressure-reducing mattress** (now individualised — 2 or 3 hourly both acceptable).
- **30-degree lateral position; head-of-bed ≤30 degrees.**
- **Increasing pain is the best predictor of chronic wound infection (LR 11–20)** — classic signs and odour are unhelpful.
- **Levine technique = swab rotated over 1 × 1 cm for 5 seconds.**
- **Systemic antibiotics only for bacteraemia, sepsis, osteomyelitis or cellulitis** — not for routine Stage 3 ulcers.
- **Managing underlying causes beats any dressing** — no single dressing is superior.
- **Not all pressure injuries are avoidable**, especially at the end of life — but the prevention protocol must be in place first.

FINAL REVISION SHEET

Cross-Module Numbers Worth Memorising

Number	What it belongs to
1/3 · 1/2 · 1 in 5	Community elders falling yearly · nursing-home residents falling yearly · falls causing serious injury
11.6%	Fall prevalence in Indian adults ≥60 years (LASI wave 1)
<0.6 m/sec	Abnormal gait speed; each 0.1 m/sec drop = 7% rise in fall risk
>12 seconds	Timed Up and Go cut-off for high fall risk (10 seconds proposed in 2025)
<24 / 28	Tinetti POMA score indicating fall risk
0–56 · 0–12	Berg Balance Scale range · SPPB range
≥45 · ≥2 · ≥5	Morse Fall Scale high risk · STRATIFY at risk · Hendrich II high risk
>20 mmHg	Systolic drop defining orthostatic hypotension
24% · 34% · 19%	Fall reduction: balance/functional exercise · multiple exercise types · Tai chi
14 classes	STOPPFall fall-risk-increasing drug classes
3%	Proportion of elderly hip fractures that are pathological
56 · 28 · 12 · 5 (%)	One year after hip fracture: fall again · repeated falls · new fracture · opposite hip fracture
33% · 77% · 25%	Zoledronic acid: clinical · vertebral · peripheral fracture reduction
NNT 200	Oral bisphosphonate, to prevent one hip fracture
RR 0.82	Hip protectors in nursing/residential care (no benefit in community)
≥50 hours	Minimum duration of a structured progressive exercise programme
>90%	Pressure ulcers occurring in the lower body
30–35 kcal/kg · 1–1.5 g/kg · 1 mL/kcal	Energy · protein · fluid targets in the immobile patient
2-hourly / 4-hourly	Repositioning: regular mattress / pressure-reducing mattress (now 2 or 3 hourly, individualised)
30 degrees	Lateral tilt position and maximum head-of-bed elevation
LR 11–20	Increasing pain as a predictor of chronic wound infection
1 × 1 cm, 5 seconds	Levine technique for quantitative wound swab

7.8% · 24.3% · 42.8%	Indian tertiary hospital: overall pressure ulcer prevalence · ICU prevalence · proportion that are Grade III

The four sentences that carry the most marks in this module.
1. Falls are multifactorial — an acute trigger acting on accumulated chronic impairments; medications are the most modifiable factor.
2. Progressive balance and functional exercise has the strongest evidence of any fall-prevention intervention; technology and scoring tools are adjuncts.
3. A fragility fracture requires both a fall and fragile bone — and the effectiveness of fracture prevention *increases* with advancing age.
4. In pressure injuries, correcting the underlying contributing factors matters more than the choice of dressing or any adjunctive therapy.

MD GERIATRIC MEDICINE EXAMINATION COMPENDIUM

HEALTH POLICY, NATIONAL PROGRAMMES & SOCIAL GERONTOLOGY

Nine structured long-answer questions
Textbook-sourced points • Latest Updates (2023–2026) • Current Practice Update
Examiner's Tips • Rapid Revision Grid

Primary sources: Pathy's Principles and Practice of Geriatric Medicine, 6th edition
Essentials of Clinical Geriatrics, 7th edition
Supplemented by: MoHFW / MoSJE / NITI Aayog / WHO / ILO documents, 2023–2026

Prepared for Dr. Sudarsan • Compiled August 2026

CONTENTS

Q1	Health Policy and Programmes Related to Old People	**3**
Q2	National Programme for Health Care of the Elderly (NPHCE) — Background, Vision, Objectives, Expected Outcomes, Package of Services and Centre Activities	**7**
Q3	Implementing NPHCE Components as the Doctor In-Charge of a District Hospital — Theory into Practice	**12**
Q4	National Policy on Older Persons (1999) and Existing Beneficiary Schemes for the Elderly	**17**
Q5	Social Security System for Older People in India	**22**
Q6	Role of Government in Providing Healthcare and Economic Security for Older People	**27**
Q7	Integrated Care of Older People (ICOPE)	**31**
Q8	Geriatric Demography and Its Implications	**36**
Q9	Ethno-Geriatrics	**41**
—	**APPENDIX: Rapid Revision Grid** — Key numbers, schemes, dates, mnemonics and legal provisions	**46**

HOW TO USE THIS COMPENDIUM

Each answer follows a fixed architecture: a **Source Statement** declaring exactly what the uploaded textbooks do and do not cover → **15–20 numbered high-yield points** under thematic subheadings → **Latest Updates (2023–Present)** with inline citations → a **Current Practice Update** two-column table contrasting the textbook position with current evidence → an **Examiner's Tip** panel with verbatim mark-earning lines.

Critical exam strategy for this block: Pathy's and Essentials contain **no Indian policy content whatsoever**. Examiners know this. The highest-scoring answers explicitly state which framework comes from the textbook (concepts, ethics, global data) and which comes from Government of India documents (programmes, schemes, law) — then show how the two fit together.

Q1. Health Policy and Programmes Related to Old People

SOURCE STATEMENT

Pathy's 6th edition is largely SILENT on Indian national health policy for the elderly. It covers only the **UK/NHS historical perspective** (Ch. 1), the **WHO global framework and social determinants of ageing** (Ch. 4, Ch. 7) and the **UK National Service Framework** (Ch. 11). *Essentials of Clinical Geriatrics* covers **US programmes** (Medicare, Medicaid, Older Americans Act) in Ch. 15.
Points 1–6 are textbook-sourced. **Points 7–20 — the Indian programmes that form the bulk of an Indian university answer — are from Government of India, WHO and peer-reviewed sources.**

A. Concepts and Global Framework (Pathy's, Ch. 4, 7, 11)

1. **Healthy ageing** is defined by WHO as developing and maintaining the **functional ability** that enables well-being in older age. Pathy's states that **functional ability — retaining autonomy and independence — is the cornerstone of healthy ageing**.
2. **Intrinsic capacity** is the composite of all physical and mental capacities. Pathy's lists its domains as **cognition, mental health, sensory function, metabolic/vitality, mobility and muscle strength**.
3. Pathy's notes the **WHO Global Strategy and Action Plan on Ageing and Health**, whose goals are to create **age-friendly environments**, **align national health systems** with the needs of older people, and **support research** to monitor global ageing.
4. **Historical UK policy:** the **NHS was created in 1948** with a tripartite structure (hospitals, GP services, local authority services), free at the point of use. Pathy's criticises it for **poor coordination between the three arms** and a smaller share of funds for chronic and mental health services.
5. **UK National Service Framework (NSF) for Older People, 2001:** its **Standard One was "rooting out age discrimination."** Pathy's stresses that **ageism persists**, causing older people to be denied beneficial investigations and treatments, and warns against both **therapeutic nihilism and therapeutic heroism**.
6. **US programmes (Essentials, Ch. 15): Medicare Part A** (hospital) and **Part B** (medical); **Medicaid** (means-tested, main payer for nursing homes); **Title XX Social Services Block Grants**; and **Title III of the Older Americans Act** (congregate meals and meals-on-wheels for all persons **>60 years regardless of income**). Payment by **DRGs** works against geriatric goals as it focuses on a single diagnosis.

B. Indian Legal and Policy Framework

7. **Constitutional basis:** **Article 41** (Directive Principle) directs the State to provide **public assistance in old age, sickness and disablement**; **Article 46** protects weaker sections. Ageing is a **Concurrent/State subject**, so States must frame their own action plans.
8. **National Policy on Older Persons (NPOP), 1999** — India's first ageing policy. Five thrust areas: **income security, health and nutrition, shelter, education, and welfare/empowerment**. It proposed a **National Council for Older Persons**. Goal: the well-being of older persons by strengthening their legitimate place in society so they can live with **purpose, dignity and peace**.
9. **NPOP 1999 was revised in 2011 as the National Policy on Senior Citizens (NPSrC)**, which emphasised **"ageing in place," the 80+ "old-old," and elderly women**. **Exam point: NPSrC 2011 remains a draft and has never been formally notified.**
10. **Maintenance and Welfare of Parents and Senior Citizens (MWPSC) Act, 2007** is the key legislation, making maintenance of parents by children a **legal obligation**. State governments constitute **Maintenance Tribunals** presided over by an administrative officer; maintenance may not exceed **₹10,000 per month**.
11. **Other key MWPSC provisions:** disposal within **90 days**; **abandonment is punishable** (up to 3 months imprisonment and/or fine); **transfer of property by gift/deed can be declared void** if the transferee fails to provide basic amenities; **Chapter IV mandates medical care** — separate queues, beds and expanded facilities for chronic, terminal and degenerative disease.
12. The **MWPSC (Amendment) Bill, 2019** proposes to widen the definitions of "children," "parents," "maintenance" and "welfare," to allow **priority disposal for those above 80 years**, and to **remove the ₹10,000 ceiling**. It was referred to a Standing Committee and **is still pending**.

C. Health Programme — NPHCE

13. The **National Programme for the Health Care of the Elderly (NPHCE)** was launched by **MoHFW in 2010**, based on the recommendations of NPOP and the State's obligation under the MWPSC Act 2007. It is also an articulation of India's commitment under the **UNCRPD**, aiming at **accessible, affordable, long-term comprehensive care and the promotion of Active and Healthy Ageing**.

14. **NPHCE service ladder (memorise): Sub-centre** — health education, domiciliary visits by ANM/ASHA; **PHC** — weekly geriatric clinic, basic drugs, rehabilitation guidance; **CHC** — twice-weekly clinic, **physiotherapy unit**, referral; **District Hospital** — **10-bedded geriatric ward** and dedicated OPD; **Regional Geriatric Centre** — tertiary care, **30-bedded ward**, PG training.
15. The **tertiary component was renamed "Rashtriya Varisth Jan Swasthya Yojana" in 2016–17**, delivered through **Regional Geriatric Centres in 19 medical colleges across 18 states**, plus **two National Centres of Ageing — AIIMS New Delhi and Madras Medical College, Chennai**.
16. NPHCE is a **centrally sponsored scheme (75% Central : 25% State)**, now implemented under the **National Health Mission NCD flexi-pool**. Its **eight components** are preventive/promotive, primary, secondary, tertiary, home-based care, referral, human-resource development and **IEC**.

D. Social Welfare and Financial Programmes

17. **Atal Vayo Abhyuday Yojana (AVYAY)** — Ministry of **Social Justice & Empowerment**. Components: **IPSrC** (grants to NGOs for old-age and continuous-care homes), **SAPSrC** (State Action Plan), **Rashtriya Vayoshri Yojana (RVY)**, **SCOPE** and **SAGE** (Seniorcare Ageing Growth Engine).
18. **RVY** provides **free assisted-living devices** (spectacles, hearing aids, dentures, walking sticks, wheelchairs, walkers) to seniors who are **BPL or have monthly income up to ₹15,000**; devices are made by **ALIMCO** and given through camps. **Elderline (14567)** is the national toll-free helpline for **information, guidance, emotional support and field intervention in elder abuse and rescue**.
19. **Financial and social security: IGNOAPS** (₹200/month for 60–79; ₹500/month for 80+, central share), **SCSS**, **PMVVY**, **Atal Pension Yojana**, **Senior Citizens' Welfare Fund**, income-tax relief under **Sec 80D and 80TTB**, and rail/air travel concessions.
20. **Health insurance: Ayushman Bharat** has two pillars — **Ayushman Arogya Mandir** (formerly HWCs) for comprehensive primary care with **population-based NCD screening from age 30 using the CBAC form**, and **PM-JAY** giving **₹5 lakh per family per year** for secondary and tertiary hospitalisation.

LATEST UPDATES (2023–PRESENT)

1. Ayushman Vay Vandana Card — the single biggest Indian policy change (October 2024). Launched **29 October 2024** under AB PM-JAY, it gives **₹5 lakh of free annual health cover to every citizen aged 70 years and above, irrespective of income**, with **no waiting period for pre-existing conditions**. Seniors aged 70+ already in a PM-JAY family receive an **additional ₹5 lakh top-up for themselves**. It covers about **1,961 procedures across 27 specialties** including dialysis, knee and hip replacement and cancer care; enrolment is via the Ayushman App with Aadhaar e-KYC. **9.67 million cards issued by 31 December 2025.** (NHA, 2024; MoHFW Lok Sabha reply, 2026)

2. WHO ICOPE Handbook, 2nd edition (January 2025). Shifts from a disease-oriented to a holistic approach focused on **intrinsic capacity and functional ability**, detailing **four steps** — basic assessment, in-depth assessment, personalised care plan, implementation/monitoring — screening **six domains: cognition, mobility, vitality, vision, hearing and psychological capacity**, with a **new urinary incontinence care pathway**. (WHO, 2025)

3. UN Decade of Healthy Ageing (2021–2030) — mid-decade progress. Four action areas: **combating ageism, age-friendly environments, integrated care, long-term care**. The first progress report (November 2023, 136 countries) found a **>20% rise in countries with anti-ageism legislation and age-friendly city programmes**, but **fewer than one-third of countries have adequate resources**; only **1 in 4** have sufficient resources for integrated care and **1 in 3** for long-term care, and only **16% of low-income countries** have training programmes for informal caregivers. (WHO, 2023; WHO, Oct 2024)

4. NITI Aayog — "Senior Care Reforms in India" (February 2024). Groups interventions under **four core areas: Health, Social, Economic/Financial and Digital empowerment and inclusion**. Recommends **health literacy for seniors and caregivers**, extending primary care through **AB-HWCs**, expanding **home health care, mental health services and Poshan Abhiyan for seniors**, **amending the MWPSC Act** to cover abuse and harassment, **elderly-friendly housing reforms**, a **national portal for senior care**, formal recognition of the **care economy and care workers**, and a **regulatory base interest rate on senior citizen deposits**. (NITI Aayog, 2024)

5. India Ageing Report 2023 (UNFPA–IIPS). 60+ share rises from **10.5% (2022) to 20.8% (2050)** — **149 million to 347 million**; **elderly outnumber children by 2046**; **80+ grows 279%**; **>40% of the elderly are in the poorest wealth quintile** and **18.7% have no income**; **71% live in rural areas**. (UNFPA India, 2023)

6. Research infrastructure. The **Longitudinal Ageing Study in India (LASI)** under NPHCE, Wave-1 (2017–18), covered **30 states and 6 UTs, 72,250 adults aged 45+, including 31,464 persons aged 60 and above**. (IIPS/MoHFW, 2020)

CURRENT PRACTICE UPDATE

Domain	Pathy's 6th edition (2022)	Current position (2023–2026)
Indian policy	**Not covered at all** — no NPOP, MWPSC, NPHCE or AVYAY	NPHCE, AVYAY, Vay Vandana Card and NITI Aayog 2024 form the entire Indian framework
Global framework	WHO Global Strategy and Action Plan 2016–2020	Superseded by the **UN Decade of Healthy Ageing 2021–2030** with four action areas and formal reporting (2023, 2026, 2029)
Model of care	Disease/syndrome-oriented; intrinsic capacity conceptual only	**ICOPE 2nd edition (2025)** operationalises it into a **4-step pathway** with 6 screening domains
Elderly health financing (India)	Silent	**Universal, income-independent ₹5 lakh cover for all 70+** since Oct 2024 — a shift from means-tested to age-based entitlement
Anti-discrimination	UK NSF 2001 "rooting out age discrimination"	Expanded into the **WHO Global Campaign to Combat Ageism**; >20% more countries now have legislation
Long-term care	Mainly US nursing-home/Medicaid financing	Now a **standalone action area**; India still has **no statutory LTC system**
Projections	60+ doubles between 2015 and 2050	India-specific: **10.5% → 20.8%**; elderly overtake children by **2046**

EXAMINER'S TIP — VERBATIM MARK-EARNING LINES

- *"India's ageing policy rests on three pillars: a policy (NPOP 1999), a law (MWPSC Act 2007) and a programme (NPHCE 2010) — the second and third both flow directly from the first."*
- *"The Ayushman Vay Vandana Card of October 2024 is India's first universal, age-based rather than means-tested health entitlement."*
- *"The National Policy for Senior Citizens 2011 was drafted but never notified; India therefore still operates on a policy written in 1999."*
- *"Delivery is fragmented across at least five ministries — MoHFW, MoSJE, MoRD, Labour and Finance — with no single accountable authority."*
- *"Pathy's gives the ethical and conceptual framework; the Government of India documents give the delivery architecture. A complete answer needs both."*

EXTRA POINTS TO REMEMBER (recurring topic)

- **Article 41** is the constitutional anchor — quote it in every policy answer.
- **MWPSC Act:** ₹10,000 cap • 90 days • abandonment punishable • property transfer void • Chapter IV = medical care.
- **NPHCE bed numbers:** District Hospital **10** (of which **2 respite**); Regional Geriatric Centre **30**.
- **Two National Centres for Ageing:** AIIMS New Delhi and Madras Medical College Chennai.
- **Elderline = 14567** (MoSJE). **RVY income ceiling = ₹15,000/month**. **Devices made by ALIMCO.**
- **Four action areas of the UN Decade:** ageism • age-friendly environments • integrated care • long-term care.

Q2. National Programme for Health Care of the Elderly (NPHCE) — Background, Vision, Objectives, Expected Outcomes, Package of Services and Centre Activities

SOURCE STATEMENT

Pathy's 6th edition does NOT mention NPHCE anywhere. It covers only the UK model (NHS 1948, "a geriatric unit in every health district," Exton-Smith's components of an effective geriatric department); *Essentials* covers the US model.

The entire content below is from the primary Government of India source — the NPHCE Operational Guidelines, DGHS, MoHFW (2010) — plus current MoHFW/PIB updates. This is essential, as NPHCE is a guaranteed question in Indian MD Geriatrics examinations.

I. Background — Why the Programme Was Needed

1. **Demographic trigger:** the 60+ population **tripled in the last 50 years**. The 2001 census recorded **75.93 million Indians above 60** (38.22 million men, 37.71 million women), projected to reach **96.30 m (2011), 133.32 m (2021), 178.59 m (2031), 236.01 m (2041) and 300.96 m (2051)**.
2. **Epidemiological trigger:** National Sample Surveys (1986–87, 1995–96, 2004) showed the **burden of morbidity in old age is enormous**, that **non-communicable, lifestyle-related and degenerative diseases are extremely common irrespective of socio-economic status**, and that **disabilities are frequent and compromise activities of daily living**.
3. The **NSS 60th Round (2004)** found **about 8% of older Indians were confined to home or bed, rising to 27% after the age of 80**, with **women more frequently affected** in both villages and cities.
4. **Health-system gaps:** primary-care infrastructure was **grossly deficient**; the health machinery was **geared to maternal-child health and communicable diseases**; the elderly need **long-term, constant, multi-speciality care**; and **no Indian model of elderly care existed**.
5. **Manpower gap:** the UG curriculum did not cover geriatrics adequately, **PG geriatric courses were grossly deficient**, only **one medical college — Madras Medical College, Chennai — produced trained manpower**, there were **no posts to absorb them**, and nearly half had migrated abroad.
6. **Policy/legal mandate — the three pillars:** **(a)** the **UN Convention on the Rights of Persons with Disabilities (UNCRPD)**, ratified effective **3 May 2008**, whose **Article 25** requires health services to be provided **close to people's own communities, including rural areas**; **(b)** the **National Policy on Older Persons (NPOP), 1999**; and **(c) Section 20 of the MWPSC Act, 2007**, dealing with medical care of senior citizens.
7. **NPOP agenda handed to MoHFW (frequently asked): geriatric wards in all district hospitals**; **expansion of treatment facilities for chronic, terminal and degenerative diseases**; improved facilities for those unable to attend centres; **inclusion of geriatric care in medical and nursing curricula**; **reservation of beds** in public hospitals; **training of geriatric care-givers**; and **research institutes for dementia and Alzheimer's disease**.
8. **Launch:** NPHCE was launched by **MoHFW in 2010, during the 11th Five-Year Plan**. The approved budget for 2010–11 and 2011–12 was **₹288 crore**, with **80% Central and 20% State share** (excluding Regional Medical Institutes and NCD Cell activities, which are fully central).

II. Vision — Five Statements (memorise verbatim)

9. **(i)** To provide **accessible, affordable, and high-quality long-term, comprehensive and dedicated care services** to an ageing population; **(ii)** creating a new **"architecture" for Ageing**; **(iii)** to build a framework for an enabling environment for **"a Society for all Ages"**; **(iv)** to promote the concept of **Active and Healthy Ageing**; **(v) convergence** with the National Rural Health Mission, AYUSH and other departments such as the Ministry of Social Justice and Empowerment.

III. Specific Objectives — Only Four

10. **(i)** To provide **easy access to promotional, preventive, curative and rehabilitative services** to the elderly through a **community-based primary health care approach**; **(ii)** to **identify health problems** in the elderly and provide appropriate interventions in the community **with strong referral back-up support**; **(iii)** to **build capacity of medical and paramedical professionals as well as care-takers within the family**; **(iv)** to provide **referral services** through district hospitals and regional medical institutions.

IV. Strategies

11. **Core strategies (6):** community-based primary health care with **domiciliary visits by trained health workers**;

dedicated services at PHC/CHC; **dedicated facilities at District Hospital with 10-bedded wards**; **strengthening of 8 Regional Medical Institutes** for tertiary care, PG courses and in-service training; **IEC using mass and folk media**; and **continuous monitoring, independent evaluation and research**.

12. **Supplementary strategies (3): public–private partnerships**; **mainstreaming AYUSH** and revitalising local health traditions with convergence with MoSJE; and **reorienting medical education**.
13. **The five operational "Main Strategies" (NHM website): (a) Preventive and promotive care** — exercise, balanced diet, stress management, tobacco avoidance and fall prevention, via domiciliary visits and **weekly PHC clinics**; **(b) Management of illness** — dedicated OPD and IPD at every level; **(c) Health manpower development** — in-service training and **PG courses at RGCs**; **(d) Medical rehabilitation and therapeutic intervention** — therapeutic exercises, **ADL training**, pain treatment through **physiotherapy units at CHC, DH and RGC**; **(e) IEC** — mass and folk media plus regular check-up camps.

V. Expected Outcomes — Seven (high-yield list)

14. **(i) Regional Geriatric Centres in 8 Regional Medical Institutions**, each with a dedicated Geriatric OPD and a **30-bedded Geriatric ward**, plus training and research; **(ii) 16 post-graduates in Geriatric Medicine** (2 seats each); **(iii) video-conferencing units in the 8 institutions**; **(iv) District Geriatric Units with Geriatric OPD and 10-bedded ward in 80–100 District Hospitals**; **(v) geriatric clinics/rehabilitation units for domiciliary visits at CHCs and PHCs**; **(vi) sub-centres equipped for community outreach**; **(vii) training of human resources** in geriatric care.

VI. Package of Services

15. **General principle:** promotional, preventive, curative and rehabilitative services in an **integrated manner**, with the package **depending on the level of facility** — health promotion, preventive services, diagnosis and management (OPD and IPD), **day-care**, **rehabilitative** and **home-based care**, with districts linked to RGCs. Services are **integrated (horizontal) below district level and vertical at district level and above**, because more specialised care is needed.

16. Package of Services by Level — TABLE TO MEMORISE

Level	Package of Services
Sub-Centre	Health education on **healthy ageing**; **domiciliary visits** for home-bound/bedridden elderly with **training of family care-providers**; arranging **callipers and supportive devices** from the PHC; **linkage with support groups and day-care centres**
Primary Health Centre	**Weekly geriatric clinic by a trained Medical Officer**; record of each elderly person on a **standard format at first visit**; routine assessment (eye, BP, blood sugar); **medicines and advice on chronic ailments**; awareness on **health and village sanitation days**; **referral** to CHC/DH
Community Health Centre	Acts as the **First Referral Unit (FRU)**; **geriatric clinic twice a week**; **rehabilitation unit for physiotherapy and counselling**; **domiciliary visits by the rehabilitation worker** with family counselling; health promotion; referral to District Hospital
District Hospital	**Dedicated geriatric OPD**; laboratory investigations and medicines; **10-bedded Geriatric Ward**; support from existing specialities (Medicine, **Orthopaedics, Ophthalmology, ENT**); services to patients referred by CHC/PHC; **conducting geriatric camps**; referral to tertiary level
Regional Geriatric Centre	**Specialised Geriatric OPD**; **30-bedded Geriatric ward** plus **dedicated beds in Surgery, Orthopaedics, Psychiatry, Urology, Ophthalmology, Neurology**; laboratory investigations with a **special sample-collection centre in the OPD block**; tertiary care for referrals from medical colleges, district hospitals and below

VII. Centre-wise Activities, Staff and Equipment

17. **Sub-Centre:** the **ANM / Male Health Worker** makes domiciliary visits, gives health education on **environmental modification, nutrition, lifestyle and behavioural change**, and trains family care-providers. An **annual check-up of all elderly at village level** is organised by PHC/CHC and recorded in a **Standard Health Card for the Elderly**; **ASHA mobilises the elderly** for camps and home-based care. **Equipment:** walking sticks, callipers, infrared lamp, shoulder wheel, pulley, ordinary walker. **No additional contractual staff.**
18. **PHC and CHC:** at the **PHC** the Medical Officer is in charge; assessment covers **vision, joints, hearing, chest, BP and simple investigations including blood sugar**, with advice on **COPD, arthritis, diabetes and hypertension including dietary regulation**. Equipment: **nebuliser, glucometer, exercise bicycle, cervical and lumbar traction, gait-training apparatus, infrared lamp**. At the **CHC** one **Rehabilitation Worker** is sanctioned; equipment adds **ECG machine, pulse oximeter, defibrillator, multi-channel monitor, shortwave diathermy, intermittent cervical traction**; the CHC **compiles PHC data and forwards it to the District Programme Officer (NCD)**.
19. **District Hospital (very high-yield):** a **Geriatric Unit** with a **10-bedded ward, of which 2 beds are earmarked in**

a separate room for RESPITE CARE. Contractual staff = **13** (2 Consultant Medicine, 6 Nurses, 1 Physiotherapist, 2 Hospital Attendants, 2 Sanitary Attendants). Finance: **₹87 lakh non-recurring** (₹80 lakh construction + ₹7 lakh equipment) and **₹44 lakh recurring per annum** (including **₹10 lakh drugs and consumables**, ₹2 lakh IEC). Equipment adds **non-invasive ventilator, ultrasound therapy, pelvic traction, TENS**.

20. **Regional Geriatric Centre — the tertiary arm:** originally established in the **Department of Medicine of 8 institutions**: **AIIMS New Delhi; IMS-BHU Varanasi; SKIMS Srinagar; Govt. Medical College Thiruvananthapuram; Guwahati Medical College; Madras Medical College Chennai; SN Medical College Jodhpur; Grant Medical College & JJ Hospital Mumbai**. **Six functions:** tertiary care; **conducting PG courses**; **training the trainers**; **developing evidence-based treatment protocols**; developing training modules, guidelines and IEC; and **research**. Contractual staff = **38** (1 Professor, 2 Assistant Professors, 4 Senior Residents, 16 Nurses, 3 Physiotherapists + 1 Occupational Therapist, 1 Medical Social Worker, 1 Lab Technician, 1 Programme Assistant, 4 Hospital and 4 Sanitary Attendants). Funding = **₹370 lakh non-recurring** (₹200 lakh construction, ₹50 lakh equipment, **₹120 lakh video-conferencing**) and **₹163.44 lakh recurring** (including **₹40 lakh research**). A minimum of **2 beds must be earmarked for the elderly in each allied speciality**.
21. **RGC ward and clinic design (WHO–MMC core group, Annexure II):** the **30-bed ward is split into 15 male + 15 female**, each with **3 acute-care, 7 sub-acute and 5 long-term care beds**. Referral criteria: **age 60+ with preference to the very old (80+), frail patients with poor functional status, and those with multiple medical, psychological and social problems**. A **dedicated daily OPD** is required, **Comprehensive Geriatric Assessment must be done on every patient**, allied specialities provide services **at least once a week**, and **specialised clinics — Incontinence Clinic, Dementia Clinic and Falls Clinic — are organised at least once a week**. **Age-friendly environment** is mandated: **wide corridors, ramps, railings, grab bars, anti-skid tiles, adequate illumination, toilets for the disabled and elevators**.
22. **Institutional framework and monitoring:** NPHCE operates through the **National, State and District NCD Cells** created under NPCDCS, with funds flowing GoI → **State Health Society** → **District Health Society** → Rogi Kalyan Samitis, into **separate NPHCE bank accounts**. States have **10% inter-component flexibility**. **Monthly reporting** uses **Form 1 (Sub-centre, ANM/MHW), Form 2 (PHC), Form 3 (CHC), Form 4 (District Hospital), Form 5 (RGC, by Professor & Head of Medicine) and Forms 6 & 7 (State → National NCD Cell)**. The State must appoint a **Nodal Officer**, provide land, faculty and diagnostics, **start 2 PG seats/year**, and eventually **take over the units**.

LATEST UPDATES (2023–PRESENT)

1. The programme is now restructured into TWO components — state this in every answer. (a) An NHM component — primary and secondary care through District Hospitals, CHCs, PHCs and **Sub-Centres/Ayushman Arogya Mandirs**; and **(b) a Tertiary Component, renamed "Rashtriya Varisth Jan Swasthya Yojana (RVJSY)" in 2016–17**. Per the December 2024 Rajya Sabha reply, the NHM component provides **dedicated geriatric OPD, physiotherapy and IPD at AAM-HWCs, PHCs, CHCs and District Hospitals**, and the tertiary component comprises **2 apex National Centres for Ageing (AIIMS New Delhi, Madras Medical College Chennai)** plus Regional Geriatric Centres. (DGHS 2025; MoHFW Rajya Sabha reply, Dec 2024)

2. Massive scale-up beyond the original targets. DGHS currently lists **Regional Geriatric Centres in 19 medical colleges across 18 states** plus the two National Centres of Ageing — up from the original 8. A PIB update states **713 districts have been covered** (original target was only 80–100 district hospitals). **Exam tip: figures vary between official replies (17 vs 19 RGCs) — quote both with the source.** (DGHS 2025; PIB, MoHFW)

3. Research arm — LASI. Conducted under NPHCE to broaden its scope; **Wave-1 covered all 30 states and 6 UTs with 72,250 adults aged 45+, including 31,464 persons aged 60 and above** and their spouses. This is now the evidence base for NPHCE planning. (IIPS/MoHFW)

4. Convergence with Ayushman Bharat — the biggest change since 2024. The financing gap NPHCE could never close (out-of-pocket cost of surgery, joint replacement, dialysis, cancer care — explicitly identified in the 2010 guidelines) is now addressed by the **Ayushman Vay Vandana Card (29 October 2024)**, giving **₹5 lakh free annual cover to all citizens aged 70+ irrespective of income**. As stated in the President's address to Parliament (January 2026), **nearly one crore cards have been issued in about one and a half years, enabling roughly eight lakh elderly citizens to access free healthcare**, and **1.80 lakh Ayushman Arogya Mandirs are operational**. (NHA 2024; GoI, Jan 2026)

5. Persisting implementation gaps — write these as "challenges". Published evaluations identify **shortage of trained geriatricians and faculty, non-filling of sanctioned contractual posts, incomplete construction of 10-bedded wards, weak rural reach, poor awareness, under-utilisation of allotted funds and weak HMIS reporting**. A NIHFW evaluation noted scale-up was planned at **100 districts per year with 12 additional RGCs**, but stressed the need to learn lessons from the first phase before further expansion. (NIHFW 2024; J Family Med Prim Care 2022; Ann Geriatr Med Res 2022)

6. Policy direction for the future. NITI Aayog (Feb 2024) recommends strengthening geriatric healthcare within the existing system, extending primary care through **AB-HWCs**, promoting health literacy, and expanding **home health care and mental health services**. Internationally, the **WHO ICOPE Handbook 2nd edition (January 2025)** now provides the operational model NPHCE lacks — a **four-step primary-care pathway** screening **six intrinsic-capacity domains**. (NITI Aayog 2024; WHO 2025)

CURRENT PRACTICE UPDATE

Aspect	Original Operational Guidelines (2010)	Current position (2023–2026)
Regional Geriatric Centres	**8** Regional Medical Institutions	**17–19 RGCs** (figures differ by source) **+ 2 National Centres for Ageing**
Tertiary component name	Not named separately	Renamed **"Rashtriya Varisth Jan Swasthya Yojana" (2016–17)**
District coverage	80–100 district hospitals; 100 districts in 21 states in phase 1	**~713 districts** reported covered
Lowest delivery point	Sub-Centre with ANM/MHW	**Ayushman Arogya Mandir** with CHO-led teams and **CBAC-based NCD screening from age 30**
PG output	Target of **16** PG seats from 8 institutes	Multiple MD/DM/DNB Geriatric Medicine programmes now exist — **still far short of need**
Funding pattern	**80:20** Centre:State per the original MoU	Implemented under **NHM norms (generally 60:40; 90:10 for NE and Himalayan states)**; some official pages quote **75:25**
Catastrophic expenditure	No financial protection built in	Covered by **AB PM-JAY + Ayushman Vay Vandana Card** (₹5 lakh, 70+, since Oct 2024)
Assessment model	Simple clinical assessment + standard health card	Global shift to **ICOPE intrinsic-capacity screening (WHO 2025)** — **not yet formally adopted in NPHCE**
Long-term care	Only **2 respite beds** per district hospital	**No statutory long-term care system in India** — flagged by NITI Aayog (2024) and the UN Decade

EXAMINER'S TIP — VERBATIM MARK-EARNING LINES

- *"NPHCE rests on three mandates: UNCRPD Article 25, NPOP 1999, and Section 20 of the MWPSC Act 2007."*
- *"The programme is integrated (horizontal) below district level and vertical at district level and above, because more specialised care is needed."*
- *"Of the 10 district hospital beds, two must be earmarked in a separate room for respite care — the only formal recognition of long-term care in the entire Indian health system."*
- *"The RGC 30-bed ward is 15 male + 15 female, each with 3 acute, 7 sub-acute and 5 long-term care beds, and must run Incontinence, Dementia and Falls clinics at least weekly."*
- *"The tertiary component was renamed Rashtriya Varisth Jan Swasthya Yojana in 2016–17 and now runs through 17–19 RGCs and 2 National Centres for Ageing."*

EXTRA POINTS TO REMEMBER (guaranteed question)

- **Vision = 5 statements • Objectives = 4 • Expected outcomes = 7 • Components = 8 • Main strategies = 5**.
- **Staff:** District Hospital **13**; Regional Geriatric Centre **38**; CHC **1 rehabilitation worker**; Sub-centre **nil**.
- **Money:** DH ₹87 lakh non-recurring / ₹44 lakh recurring; RGC ₹370 lakh non-recurring / ₹163.44 lakh recurring.
- **Clinic frequency:** PHC **weekly**; CHC **twice weekly**; RGC **daily OPD**; allied specialities and special clinics **weekly**.
- **Reporting forms 1–7** — Form 4 is the District Hospital's; Form 5 is signed by the Professor & Head of Medicine at the RGC.
- **The original 8 RGCs** — remember at least four by name (AIIMS Delhi, BHU, SKIMS, MMC Chennai).

Q3. Implementing NPHCE Components as the Doctor In-Charge of a District Hospital — Putting Theory into Practice

SOURCE STATEMENT

Pathy's does not mention NPHCE — but it DOES supply the evidence base for how to run a geriatric unit (CGA, delirium prevention, orthogeriatrics, discharge communication, ward design, avoiding functional decline). This answer therefore combines:

- **Administrative skeleton** → NPHCE Operational Guidelines, DGHS/MoHFW (2010) *(external; textbook silent)*
- **Clinical flesh** → **Pathy's 6th ed. and Essentials of Clinical Geriatrics** *(uploaded textbooks, attributed below)*

Phase 1 — Getting Started (Month 0–1)

1. **Do a situational analysis first.** The District NCD Cell is responsible for **preparing a District Action Plan** and **maintaining and updating a district database of the elderly**. Count the district's 60+ population from census/HMIS, list existing PHCs, CHCs and sub-centres, and identify current elderly OPD attendances and admissions. **Baseline data is your bargaining chip for funds.**
2. **Know your money before you plan.** A District Hospital receives **₹87 lakh non-recurring** (₹80 lakh construction + ₹7 lakh equipment) and **₹44 lakh recurring per annum** (₹28.20 lakh HR, ₹10 lakh drugs, ₹3 lakh equipment, ₹2 lakh IEC, ₹0.80 lakh training). States have **10% flexibility to move funds between components**. Funds flow **GoI → State Health Society → District Health Society → Rogi Kalyan Samiti**, into a **separate NPHCE bank account**.
3. **Get approvals in writing.** Place the plan before the **District Health Society (chaired by the District Collector)** and the **Rogi Kalyan Samiti**. Nominate a **single Nodal Officer for NPHCE**. Given the scarcity of geriatricians, the guidelines explicitly permit **existing specialists trained in or interested in geriatrics to run the Geriatric Clinic and Ward**.

Phase 2 — Building the Structure (Month 1–6)

4. **Set up the 10-bedded ward — and don't forget the 2 respite beds.** Of the 10 beds, **2 must be earmarked in a SEPARATE ROOM for respite care** of bedridden or home-bound elderly. Space may be new construction, renovation or extension. **Practical tip:** a ground-floor ward near the OPD block, next to the laboratory, saves the patient the most walking.
5. **Make the ward age-friendly — a mandated design brief, not decoration.** Required: **wide corridors, ramps, railings, grab bars, anti-skid tiles, adequate illumination, toilets for the disabled and elevators**. **Pathy's** adds that hospital design prevents falls and delirium through **adequate lighting, noise reduction, orientation boards, suitable hospital beds, appropriate flooring and clear signage**. Add **large-print signage, a wall clock and calendar in every bay, and beds low enough for feet to touch the floor**.
6. **Run a "one-stop" Geriatric Clinic.** The guidelines state that **"efforts should be made to minimize movement of the Elderly in the hospital for examination by Specialists and laboratory investigations,"** and that a **special sample-collection centre should be provided in the OPD block**. Bring the phlebotomist, pharmacist and physiotherapist to the clinic rather than sending the patient around, and give the elderly a **separate queue/token** — also their right under the MWPSC Act 2007.
7. **Procure and commission equipment.** DH list: **nebuliser, glucometer, ECG machine, defibrillator, multi-channel monitor, non-invasive ventilator, shortwave diathermy, ultrasound therapy, intermittent cervical traction, intermittent pelvic traction, TENS, adjustable walker**. **Do not let equipment sit in a store-room** — commission it, name a custodian, log monthly use; non-utilisation is the commonest audit finding.

Phase 3 — Building the Team (Month 2–6)

8. **Recruit the sanctioned 13 contractual staff: 2 Consultants (Medicine), 6 Nurses, 1 Physiotherapist, 2 Hospital Attendants, 2 Sanitary Attendants**. The **District NCD Cell** (District Programme Officer, Programme Assistant, Finance-cum-Logistics Officer, Data Entry Operator) engages contractual personnel and maintains fund flow and Utilisation Certificates.
9. **Build a real multidisciplinary team, not a paper one. Essentials** stresses that **geriatric care implies team care** — good communication avoids duplication and gives better outcomes, but **"good team care will not arise spontaneously"** and requires investment of effort, a common language and a common "playbook." A 20-minute daily huddle and a weekly MDT meeting is enough; borrow the **dietician, medical social worker, ophthalmologist, ENT surgeon, orthopaedic surgeon, psychiatrist and dentist** from existing staff.

Phase 4 — Clinical Processes (the part examiners reward most)

10. **Make CGA the routine, not the exception. Pathy's** lists the **six key elements of CGA: functional ability,**

cognitive function, physical health, socioeconomic status, nutritional status, and mobility/falls risk, and states that **when CGA is combined with strong and sustained interventions, better long-term outcomes are achieved**. **Essentials** warns that **"assessment without follow-up is unlikely to make any difference"** — hence the term shifted to **geriatric evaluation and management**.

11. **Keep CGA efficient or it will collapse under OPD load. Essentials** recommends a **close-knit interdisciplinary team with minimal redundancy**; **pre-visit questionnaires**; **screening tools that target who needs deeper assessment**; **forms that feed a database**; and **integration with case management**. Routine assessment of everyone is not cost-effective — assessments must be **TARGETED**, especially at **times of threatened or actual decline and impending change in living situation**.
12. **Use simple validated risk screens on admission. Pathy's** names **falls risk assessment tools, the Waterlow score for pressure sores and the Malnutrition Universal Screening Tool (MUST)**, warning they must be **completed by staff who understand the condition**, and that **identifying risk must be followed by justifiable action**. **Essentials** flags those at risk of functional decline: **age 75+, cognitive impairment, dependence in 2 or more IADLs before admission, existing pressure sore, baseline functional dependency and low social activity**.
13. **Run a delirium-prevention bundle from day one — free and highly effective.** Pathy's cites the **Elder Life Program (HELP)**, which addressed **six risk factors — cognitive impairment, visual impairment, hearing impairment, sleep deprivation, immobility and dehydration** — using **simple common-sense measures delivered by a trained interdisciplinary team, reducing delirium incidence from 15% to 9.9% (OR 0.60, p = 0.02)**. Costs nothing but nursing discipline: orientation boards, spectacles and hearing aids kept with the patient, night-time noise reduction, early mobilisation, fluid chart.
14. **Fight iatrogenic dependency actively. Essentials** warns that **well-meaning staff, under efficiency pressure, do things FOR elderly patients rather than urging them to do as much as possible themselves — fostering dependency at the very time independent function is crucial**. A **rehabilitative philosophy must be maintained outside therapy sessions**. Guard against **immobility complications: pressure sores, postural hypotension, atelectasis and pneumonia, thromboembolism, incontinence, constipation, deconditioning, contractures and depression**.
15. **Set up two "quick-win" pathways: orthogeriatrics and stroke care. Pathy's** notes that **most UK hospitals now have an orthogeriatric service ensuring optimal medical care from admission to discharge for fractured neck of femur**, and that **stroke units improve outcomes through focused immediate rehabilitation and swift recognition of complications**. The Stroke Unit Trialists' Collaboration meta-analysis of **29 trials, 6,536 patients** showed odds reductions for **mortality 0.86, death or dependence 0.78, and death or institutionalisation 0.80 at one year** — independent of age, sex and stroke type. In a district hospital this means: **a shared geriatric-orthopaedic protocol for hip fracture, and a designated stroke corner with early mobilisation and swallow screening**.
16. **Fix discharge communication — where district hospitals lose most patients. Pathy's** states that **inadequate communication at discharge, particularly about medication management and monitoring arrangements, leads to preventable readmissions**, and that **decisions about preferred levels of future intervention must be communicated at discharge, otherwise patients undergo unwanted intervention afterwards**. **Essentials** adds that **discharge planning is often begun too late**, and separates two questions: **(1) what TYPE of care will achieve the goals? (2) which provider can best deliver it? Practical rule: start discharge planning on day 1; send home a typed card listing diagnoses, functional status, medicines with a stop/review date, follow-up date and the ASHA's phone number.**

Phase 5 — Reaching Outward (Month 6–12)

17. **Activate the referral chain and outreach — the district hospital is the hub, not an island.** DH duties include **training Medical Officers and paramedical staff of CHCs and PHCs**, **serving patients referred by CHCs/PHCs**, **conducting camps at PHCs/CHCs**, and **referring severe cases to Regional Geriatric Centres**. Below you: **CHC = FRU with a twice-weekly clinic and a rehabilitation worker doing domiciliary visits**; **PHC = weekly geriatric clinic**; **sub-centre ANM = domiciliary visits, callipers, walking sticks, walkers**. An **annual village-level check-up of all elderly** should be organised, recorded in the **Standard Health Card for the Elderly**, with **ASHA mobilising attendance**.
18. **Train your own staff and everyone below you.** Training norms at DH: **2 doctors, 6 nurses and 1 physiotherapist**, integrated with NPCDCS via medical and nursing colleges. Add the modules people forget: **CGA, falls prevention, pressure-area care, safe prescribing/deprescribing, dementia and delirium recognition, incontinence care, and care-giver training for families**.

Phase 6 — Convergence, Records and Monitoring

19. **Converge with every other scheme — where the real value is delivered.** Link the geriatric clinic with **NPCDCS/NP-NCD** (hypertension, diabetes, cancer screening), **NPCB&VI** (cataract, refractive error), **NPPCD** (hearing

aids), **National Mental Health Programme** (depression, dementia), **National Programme for Palliative Care**, **ALIMCO camps under Rashtriya Vayoshri Yojana**, **Elderline 14567**, and **AB PM-JAY plus the Ayushman Vay Vandana Card**. The guidelines explicitly require the MO to **liaise with the Blindness Control Programme, NPCDCS and other programmes** for diagnostics, equipment, consumables and medicines.

20. **Report monthly, audit quarterly, prove your outcomes.** The **MO In-charge submits Form 4 monthly to the State NCD Cell**; PHC (Form 2) and CHC (Form 3) report to the District NCD Cell. **Pathy's** cautions that **incident-reporting systems capture only 7–15% of adverse events** compared with case-record review, so supplement reporting with **case-note audit**, using the safety framework's five dimensions: **past harm, reliability, sensitivity to operations, anticipation and preparedness, and integration and learning**. **Track:** geriatric OPD attendance, ward occupancy, average length of stay, **falls per 1,000 bed-days, new pressure sores, delirium rate, 30-day readmissions, and proportion discharged home versus institutionalised**.

Practical Roadmap — A 12-Month Implementation Plan

Timeline	Actions	Deliverable
Month 0–1	Situational analysis; district elderly database; District Action Plan; DHS/RKS approval; separate NPHCE bank account; nominate Nodal Officer	Approved plan + funds released
Month 1–3	Identify space; start renovation of 10-bed ward + OPD room; float equipment tender; start recruitment of 13 staff	Ward under construction, tenders placed
Month 2–4	**Start the Geriatric OPD immediately in existing space** — do not wait for the building	Weekly/biweekly geriatric clinic running
Month 4–6	Commission ward with **2 respite beds**; install age-friendly features; commission equipment; write SOPs (CGA form, falls, delirium, pressure-sore, hip-fracture, discharge card)	Functional 10-bed geriatric ward
Month 6–9	Train 2 doctors + 6 nurses + physiotherapist; train CHC/PHC MOs; start CHC rehabilitation units; start camps	Trained team + outreach begun
Month 9–12	Convergence MoUs (NP-NCD, NPCB, mental health, palliative care, ALIMCO/RVY); PM-JAY/Vay Vandana enrolment desk; IEC drive; **first internal audit**	Integrated service + baseline audit report

Common Problems and Practical Solutions

Problem you WILL face	Practical solution
Construction delayed; no ward	**Start the OPD first** — the guidelines explicitly say services must not wait for a building
Contractual posts unfilled	Rotate an interested existing physician + train ward nurses; escalate to District NCD Cell in writing
Beds occupied by general medical patients	Written admission criteria: **age ≥60 with preference to 80+, frail with poor functional status, multiple medical/psychological/social problems** (NPHCE–WHO core group criteria)
Equipment unused	Name a custodian; log monthly utilisation; hold hands-on demonstration for nurses and physiotherapist
Funds unspent by 31 March	Quarterly expenditure review; use the **10% inter-component flexibility**; submit UCs on time
Ageism among staff — "he is just old"	Pathy's warns ageism causes older people to be **denied beneficial treatments or mislabelled and misdiagnosed**; the geriatrician's unique skill is balancing **therapeutic nihilism against therapeutic heroism** — teach this at every induction
Poor rural reach	ASHA-mobilised village camps + CHC rehabilitation worker's domiciliary visits; tele-consultation with the linked RGC
No long-term care option	Link with the local **IPSrC old-age home** and palliative-care NGOs

LATEST UPDATES (2023–PRESENT)

1. Your district unit is now expected to work through Ayushman Arogya Mandirs. Per the December 2024 Rajya Sabha reply, the NHM component of NPHCE now delivers **geriatric OPD, physiotherapy and IPD services at AAM-HWCs, PHCs, CHCs and District Hospitals**, while the tertiary component operates through **2 National Centres for Ageing and the RGCs**. As of January 2026, **1.80 lakh Ayushman Arogya Mandirs are operational**. **Practical implication: sub-centre-level outreach is now CHO-led with CBAC-based screening — plug your geriatric follow-up into that platform rather than creating a parallel one.**

2. Set up an Ayushman Vay Vandana enrolment desk in your OPD. Since **29 October 2024**, every citizen **aged 70+** is entitled to **₹5 lakh free annual cover irrespective of income**, with **no waiting period for pre-existing conditions**, covering about **1,961 procedures across 27 specialities** including dialysis, knee and hip replacement and cancer care. **Nearly one crore cards issued within about eighteen months, enabling roughly eight lakh elderly citizens to access free treatment.** A single Aadhaar-based e-KYC desk converts NPHCE's biggest historical weakness into a strength.

3. Adopt WHO ICOPE screening as your OPD triage tool. The **ICOPE Handbook, 2nd edition (January 2025)** gives a **four-step pathway** — basic assessment, in-depth assessment, personalised care plan, implementation and monitoring — screening **six intrinsic-capacity domains: cognition, mobility, vitality, vision, hearing and psychological capacity**, plus social and caregiver support, with a **new urinary incontinence care pathway**. **This is a 5-minute screen any staff nurse or CHO can do — ideal for high-volume Indian OPDs, and it feeds directly into your CGA.**

4. Programme scale and known weaknesses. NPHCE now reports coverage of **713 districts**, and **RGCs in 19 medical colleges across 18 states plus 2 National Centres of Ageing** (the Dec 2024 parliamentary reply quotes 17 RGCs — quote both). Evaluations continue to identify **shortage of trained geriatricians, unfilled contractual posts, incomplete ward construction, weak rural reach, low awareness and under-utilisation of funds** — the exact problems your action plan must anticipate.

5. Policy direction to align with. NITI Aayog (February 2024) recommends strengthening geriatric healthcare within the existing system, extending primary care through **AB-HWCs**, promoting **health literacy among seniors and caregivers**, and expanding **home health care and mental health services**. Globally, the **UN Decade of Healthy Ageing** frames this under **combating ageism, age-friendly environments, integrated care and long-term care**.

CURRENT PRACTICE UPDATE

Element	NPHCE Guidelines as written (2010)	What you must actually do in 2026
Screening tool	Simple clinical assessment (eye, BP, blood sugar) + standard health card	Add **ICOPE 6-domain intrinsic-capacity screen** and full **CGA** for those who screen positive
Lowest contact point	Sub-centre ANM/MHW	**Ayushman Arogya Mandir with CHO + CBAC-based NCD screening**
Financing major surgery	Not addressed — a recognised gap	**AB PM-JAY + Ayushman Vay Vandana Card (70+, ₹5 lakh)** — enrol patients at your OPD
Records	Paper Forms 1–7, monthly	Migrate to **HMIS/ABDM digital records**; paper forms still mandated
Delirium / falls / pressure sores	Not specified	Use **HELP bundle, falls risk tool, Waterlow and MUST** (Pathy's) — these are your quality indicators
Hip fracture and stroke	Handled by existing specialities	Build **explicit orthogeriatric and stroke pathways** — the strongest evidence base in Pathy's
Long-term care	2 respite beds per district hospital	**Still no statutory LTC system** — link with IPSrC old-age homes and palliative care NGOs
Ageism	Not addressed	Actively teach it — NSF Standard 1 principle from Pathy's

EXAMINER'S TIP — VERBATIM MARK-EARNING LINES

• *"I would start the Geriatric OPD in existing space in month two — the guidelines require services, not buildings."*
• *"Efforts should be made to minimise movement of the elderly within the hospital — so I would run a one-stop clinic with sample collection in the OPD block."*
• *"Assessment without follow-up is unlikely to make any difference — which is why the term has shifted from CGA to geriatric evaluation and management."*
• *"The HELP bundle reduced delirium from 15% to 9.9% using six common-sense measures that cost nothing but nursing discipline."*
• *"Well-meaning staff do things FOR older patients rather than urging them to do as much as possible themselves — fostering dependency at the very time independent function is crucial."*
• *"My quality indicators would be falls per 1,000 bed-days, new pressure sores, delirium rate, 30-day readmissions and proportion discharged home rather than institutionalised."*

EXTRA POINTS TO REMEMBER

• **Six elements of CGA:** functional ability • cognition • physical health • socioeconomic status • nutrition • mobility/falls risk.
• **HELP six risk factors:** cognitive impairment • visual impairment • hearing impairment • sleep deprivation • immobility • dehydration.
• **Stroke unit meta-analysis:** 29 trials, 6,536 patients; OR mortality 0.86, death/dependence 0.78, death/institutionalisation 0.80.
• **Risk screens:** Waterlow (pressure sores) • MUST (nutrition) • falls risk tools.
• **Incident reporting captures only 7–15% of adverse events** — always add case-note audit.
• **Admission criteria for the geriatric ward:** 60+ with preference to 80+ • frail with poor functional status • multiple medical, psychological and social problems.

Q4. National Policy on Older Persons (NPOP), 1999 and Existing Beneficiary Schemes for the Elderly

SOURCE STATEMENT

Pathy's 6th edition does NOT cover the NPOP or any Indian welfare scheme. It discusses only the general social determinants of ageing (poverty, pensions, social isolation, retirement) and US/UK entitlements.
All content below is from the primary Government of India document — "National Policy on Older Persons, Ministry of Social Justice & Empowerment, 1999" — plus current MoSJE/MoRD/MoHFW/Ministry of Finance sources.

PART A — The Policy Itself

Background and Mandate

1. **When and by whom:** NPOP was announced in **January 1999, the UN International Year of Older Persons**, by the **Ministry of Social Justice & Empowerment (MoSJE)** — the **nodal ministry**. It defines a **"senior citizen"/"older person" as anyone aged 60 years and above**.
2. **Demographic trigger:** persons aged 60+ rose from **12 million (1901) to 20 million (1951) and 57 million (1991)**, with the **100 million mark expected by 2013**, **198 million by 2030 and 326 million by 2050**. Their share rose from **5.1% (1901) to 6.8% (1991)**, expected to reach **8.9% by 2016 and 21% by 2050**. In 1991, **63% were "young old" (60–69) and 11% were "older old" (80+)**.
3. **The widowhood problem (frequently asked):** in 1991 there were **14.8 million widowed females aged 60+ against only 4.5 million widowed males — four times as many** — because women marry older men, do not remarry and live longer.
4. **Constitutional and international mandate: Article 41** directs the State to secure **the right to public assistance in old age**; the **Right to Equality** applies equally to older persons; and **social security is a concurrent responsibility of the Centre and States**. Internationally it draws on the **UN Principles for Older Persons (1991)** and the **Proclamation on Ageing and Global Targets on Ageing for 2001 (1992)**.

Goal and Guiding Principles

5. **Goal (learn this line):** *"The goal of the National Policy is the **well-being of older persons**. It aims to **strengthen their legitimate place in society and help older persons to live the last phase of their life with purpose, dignity and peace**."*
6. **Six guiding principles: (i) Affirmative action**, with special attention to older women who face **"triple neglect and discrimination on account of gender, widowhood and age"**; **(ii)** the **life cycle is a continuum** — age 60 is **not** a cut-off for beginning a life of dependency; **(iii)** an **age-integrated society** with strong intergenerational bonds; **(iv)** older persons are a **resource — producers, not merely consumers**; **(v) empowerment** and participation in decision-making, since they form **12% of the electorate**; **(vi) partnership** — the State alone cannot achieve the objectives, with **special coverage of rural areas where over three-fourths of older persons live**.

The SIX Principal Areas of Intervention (core of the answer)

7. **(1) FINANCIAL SECURITY.** Old-age pension coverage to be **significantly expanded from the January 1997 level of 2.76 million**, aiming to **cover all older persons below the poverty line**, with **periodic revision of the pension rate to offset inflation** and the **PDS reaching all 60+ BPL persons**. Also: **prompt settlement of pension, provident fund and gratuity**; **expanded pension coverage** for the self-employed and private sector under a strong regulator; **tax relief sensitive to old age**; **long-term savings instruments**; **pre-retirement counselling**; removal of **age-related discrimination in credit and marketing**; and the note that **Section 125 CrPC and the Hindu Adoptions and Maintenance Act 1956** already secure parents' right to maintenance, with the **Himachal Pradesh Maintenance of Parents and Dependents Bill, 1996** cited as a model. **→ This directly produced the MWPSC Act, 2007.**
8. **(2) HEALTH CARE AND NUTRITION — the geriatrics-relevant section.** The **primary health care system is to be the basic structure**, with **geriatric care facilities at secondary and tertiary levels**. **Public hospitals are directed to ensure elderly patients are not subjected to long waits or visits to different counters for tests and treatment, to provide separate counters and convenient timings on specified days, and to set up GERIATRIC WARDS. Facilities for specialization in geriatric medicine will be provided in medical colleges**, with geriatric care in nursing training. **Health insurance is given high priority**, with state subsidy for lower-income packages. **Mobile health services, special camps and ambulance services** address access. Hospitals are encouraged to have a **separate Welfare Fund** for free treatment of the poor elderly. **Hospices** for the chronically ill without family support, including those abandoned in public hospitals. **Mental health services will be expanded and strengthened. → This paragraph is the direct ancestor of NPHCE (2010).**

9. **Health promotion within NPOP:** the policy promotes **the concept of healthy ageing**, educating older persons and families that **"diseases are not a corollary of advancing age nor is a particular chronological age the starting point for decline in health status"** and that **preventive health care and early diagnosis can prevent disability**. It mandates health education through **mass media and folk media**, messages on **balanced diet, physical exercise, regular habits, stress reduction, regular medical check-up, leisure and hobbies**, programmes on **yoga, meditation and relaxation**, and **messages targeted at younger and middle-aged groups**.
10. **(3) SHELTER. 10% of houses/house-sites in urban and rural lower-income housing schemes, including Indira Awas Yojana, to be earmarked for older persons**; **barrier-free layouts**; **multi-purpose centres in every housing colony**; **segregation of older persons is to be avoided**; **preference for ground-floor flats**; and **group housing (flatlets with common meals, laundry and rest rooms)**. Also **accident-prevention information**, **noise and pollution norms**, and **priority in civic services and property transfer/mutation**.
11. **(4) EDUCATION. Continuing education programmes**; use of **open universities and distance learning**; **access to university and research libraries**; and inclusion in the **school curriculum of material strengthening intergenerational bonds**, with the media working to **dispel negative images, myths and stereotypes** about ageing.
12. **(5) WELFARE.** Identify **the most vulnerable — the poor, disabled, infirm, chronically sick and those without family support** — and serve them first. **Institutional care is to be the LAST RESORT.** Voluntary organisations receive **grants-in-aid for construction and maintenance of old-age homes**, with **minimum standards of service**. Services to be promoted: **day care, multi-service citizens' centres, reach-out services, supply of disability aids and appliances, short-term stay, friendly home visits, helplines, telephone assurance and escort services**, plus **senior citizens' forums**. A **Welfare Fund for older persons** is to be set up. **→ This became the Senior Citizens' Welfare Fund and IPSrC.**
13. **(6) PROTECTION OF LIFE AND PROPERTY.** Older persons are described as **"soft targets for criminal elements,"** victims of **fraudulent dealings and of physical and emotional abuse within the household to force them to part with ownership rights**, with **widows' rights of inheritance, occupancy and disposal violated by their own children and relatives**. Remedies: **special provisions in the IPC against domestic violence toward older persons**, **review of tenancy legislation**, **helpline services and legal aid**, and **police keeping a friendly vigil on older couples or single persons living alone**. **→ This became Sections 23 and 24 of the MWPSC Act 2007, and later Elderline 14567.**

Other Areas, Cross-Cutting Themes and Implementation

14. **"Other Areas of Action" (easy marks): identity cards**; **fare concessions in all modes of travel**; **reservation and earmarking of seats in local public transport**; **modified vehicle design**; **traffic discipline at zebra crossings**; **priority in gas and telephone connections and fault repairs**; **removal of physical barriers**; **concessions in entrance fees at art, cultural and tourist places**; **speedy disposal of complaints of cheating and fraud**; **annual National Older Persons' Day**; and declaration of **the year 2000 as the National Year for Older Persons**.
15. **Cross-cutting chapters: NGOs** are "a very important institutional mechanism to provide user-friendly affordable services"; **Realizing the Potential** treats the 60+ phase as **"a huge untapped resource"**; **Family** is called **"the most cherished social institution in India and the most vital non-formal social security for the old,"** with **tax relief and housing preference for children who co-reside with parents** and society sensitised to **married daughters sharing the caring responsibility**; **Media** is to promote active ageing.
16. **Research and manpower (directly relevant to your specialty):** universities, medical colleges and research institutions to **set up centres for gerontological studies and geriatrics**; corporate bodies, banks and trusts requested to **institute Chairs in gerontology and geriatrics**; an **interdisciplinary coordinating body on research**; **data-collecting agencies asked to use a separate 60+ age category**; and a **national institute of research, training and documentation**. **Medical colleges to offer specialization in geriatrics**, with geriatric care in nursing and paramedical curricula.
17. **Implementation machinery (very high-yield): MoSJE is the nodal ministry**; a **separate Bureau of Older Persons**; an **Inter-Ministerial Committee**; States encouraged to set up **Directorates of Older Persons**; each Ministry prepares **Five-Year and Annual Action Plans**; a **detailed review every three years** as a **public document discussed in a National Convention**; an **autonomous National Council for Older Persons (NCOP)** headed by the Minister; an **autonomous National Association of Older Persons (NAOPS)**; and **Panchayati Raj institutions** implementing locally with representation of older women.
18. **What happened to the NCOP and the policy itself:** the **NCOP was constituted in May 1999 with 39 members** and is the **designated office for receiving suggestions, complaints and grievances from individual older persons**, with a **seven-member Working Group**. It was **reconstituted as the National Council of Senior Citizens (NCSrC) in 2012**, with similar councils at State level. NPOP 1999 was **revised in 2011 as the National Policy on Senior Citizens (NPSrC) — which has never been formally notified and remains a draft. State this in the exam.**

PART B — Existing Beneficiary Schemes (Ministry-wise Map)

19. Memorise the ministry, not just the scheme.

Ministry	Scheme	Key benefit / eligibility
MoSJE	**Atal Vayo Abhyuday Yojana (AVYAY)** — umbrella	Five components below
	↳ **IPSrC** (Integrated Programme for Senior Citizens)	Grant-in-aid to NGOs for **old-age homes, continuous-care homes**; free shelter, nutrition, medicare, recreation for indigent elderly
	↳ **SAPSrC** (State Action Plan)	Grants to States/UTs; **training of geriatric care-givers**
	↳ **Rashtriya Vayoshri Yojana (RVY)**	Free **assisted-living devices** (spectacles, hearing aids, dentures, sticks, wheelchairs, walkers) for **BPL or income ≤ ₹15,000/month**; made by **ALIMCO**, distributed through camps
	↳ **SCOPE / SACRED**	Re-employment and productive engagement portal for retired seniors
	↳ **SAGE** (Seniorcare Ageing Growth Engine)	Funding start-ups for elder-care products and services (“silver economy”)
	Elderline — 14567	Toll-free helpline for **information, guidance, emotional support and field intervention in abuse and rescue**
	Senior Citizens’ Welfare Fund (SCWF)	Pooled unclaimed deposits funding senior-citizen schemes
	Vayoshreshtha Samman	National Awards conferred on **1 October at Vigyan Bhawan**; **15 awardees in 13 categories**; **₹2.5 lakh (individual)** and **₹5 lakh (institutional)**
Ministry of Rural Development	**NSAP — IGNOAPS**	Central pension of **₹200/month (60–79)** and **₹500/month (80+)** for BPL elderly; States add top-ups
	IGNWPS, IGNDPS, NFBS, Annapurna	Widow pension, disability pension, family benefit, **10 kg free foodgrain/month** for uncovered destitute elderly
MoHFW	**NPHCE + Rashtriya Varisth Jan Swasthya Yojana**	Geriatric OPD/IPD from AAM-HWC → PHC → CHC → District Hospital (10 beds) → **RGCs and National Centres for Ageing**
	AB PM-JAY + Ayushman Vay Vandana Card	₹5 lakh/family/year; **all citizens 70+ irrespective of income since Oct 2024**
	Rashtriya Arogya Nidhi, Palliative Care Programme, NPCB&VI, NPPCD, NMHP	Financial aid for poor patients; palliative care; cataract and hearing services; mental health
Ministry of Finance / Dept of Posts	**Senior Citizens’ Savings Scheme (SCSS)**	**8.2% p.a. paid quarterly**; age **60+ (55+ VRS, 50+ defence)**; max **₹30 lakh**; **5 years extendable by 3**; **80C deduction**; TDS only above ₹1 lakh interest
	PMVVY, Atal Pension Yojana, Reverse Mortgage	Assured pension / annuity / income from own home
	Income Tax reliefs	Basic exemption **₹3 lakh (60–79)** and **₹5 lakh (80+)** under the old regime; **Sec 80TTB — ₹50,000 on interest income**; **no advance tax if no business income (Sec 207)**; **Sec 80D**; **Sec 80DDB**
Law / Home	MWPSC Act 2007; Sec 125 CrPC (now BNSS); Hindu Adoptions & Maintenance Act 1956	Legal right to maintenance; tribunals; property transfer void; abandonment punishable
Railways / Aviation / States	Travel concessions, reserved seats/berths, separate queues, identity cards	Quantum varies; **railway fare concession has remained suspended since 2020 — check current status before quoting**

20. How the whole system fits together — one line for the exam: **NPOP 1999 is the parent policy → it generated the MWPSC Act 2007 (legal enforcement), NPHCE 2010 (health delivery), AVYAY (social welfare), NSAP/IGNOAPS (income security) and Ayushman Vay Vandana (financial protection).** Everything else is a branch of that tree. Delivery, however, is fragmented across **at least five ministries with no single accountable authority** — the central criticism of Indian ageing policy.

LATEST UPDATES (2023–PRESENT)

1. Absence of an integrated policy is now officially acknowledged. The **NITI Aayog position paper (February 2024)** identifies **"an absence of a comprehensive, integrated policy for senior care and support" as a significant challenge**, causing gaps in infrastructure, capacities, evidence-based knowledge repositories for geriatric illness management, and monitoring and emergency-response frameworks. It groups reforms under **four core areas: Health, Social, Economic/Financial and Digital empowerment and inclusion**, and recommends **amending the MWPSC Act to cover abuse, harassment and protection**, **elderly-friendly housing reforms with a single-window portal**, a **national portal for senior care**, and **recognition of the care economy and care workers, especially women**.

2. The single biggest new entitlement — Ayushman Vay Vandana Card. Launched **29 October 2024**: **₹5 lakh free annual cover to every citizen aged 70+ irrespective of income**, an **additional ₹5 lakh top-up for seniors already in PM-JAY families**, and **no waiting period for pre-existing conditions**. **More than 9.67 million cards issued by 31 December 2025. This is the first genuinely universal, age-based (not means-tested) entitlement in Indian social policy.**

3. Demographic pressure has intensified since NPOP was written. The **India Ageing Report 2023 (UNFPA–IIPS)** projects the 60+ share rising from **10.5% (2022) to 20.8% (2050)** — from **149 million to 347 million**, with the **80+ population growing 279%**, the elderly **outnumbering children by 2046**, and **over 40% of the elderly in the poorest wealth quintile with 18.7% having no income at all**. **NPOP's 1999 projection of 326 million by 2050 has proved almost exactly right — quote this to show the policy's foresight.**

4. Financial-security reforms proposed. NITI Aayog recommends a **regulatory mechanism to set a viable base interest rate on senior-citizen deposits**, noting that *"since the social security framework in India is limited, most seniors depend on income generated from their savings"* and that **variable interest rates erode that income, sometimes below sustenance level**, with a further concession for older women. On the tax side, **Budget 2025 raised the TDS threshold on senior-citizen interest income from ₹50,000 to ₹1 lakh**, and under the new regime income up to **₹12 lakh is effectively tax-free via the Section 87A rebate**.

5. Legal reform still pending. The **MWPSC (Amendment) Bill, 2019** — widening definitions, giving **priority disposal to applicants above 80**, and **removing the ₹10,000 ceiling** — was referred to a Standing Committee and **remains unpassed**.

6. Global benchmark. The **UN Decade of Healthy Ageing (2021–2030)** organises national policy around **combating ageism, age-friendly environments, integrated care and long-term care**; its **2023 progress report** found **fewer than one-third of countries have adequate resources**. The **WHO ICOPE Handbook 2nd edition (January 2025)** provides a four-step primary-care pathway screening **six intrinsic-capacity domains**.

CURRENT PRACTICE UPDATE

Theme	NPOP 1999 as written	Position in 2026
Policy status	NPOP 1999 in force	**NPSrC 2011 drafted but NEVER notified** — India still runs on a 27-year-old policy
Review mechanism	**Triennial public review** discussed in a National Convention	Not conducted regularly — a recognised accountability failure
Coordinating body	**NCOP (1999)** headed by the Minister	Reconstituted as **NCSrC (2012)**; NAOPS never became functional
Income security	Expand pension to all BPL elderly with **periodic inflation-linked revision**	**IGNOAPS central share still ₹200/₹500 per month, unrevised for over a decade**
Health care	Geriatric wards, separate counters, geriatrics in medical colleges	Operationalised as **NPHCE (2010) and RVJSY (2016)** — the policy's most successfully implemented section
Health financing	Health insurance "to be given high priority"	Delivered by **AB PM-JAY + Vay Vandana (70+, ₹5 lakh, 2024)**
Long-term care / hospices	Hospices for the chronically ill without family support	**No statutory long-term care system exists** — the largest unfilled promise of NPOP
Elder abuse	IPC provisions and tenancy reform proposed	Partly delivered via **MWPSC Sections 23-24 and Elderline 14567**; IPC/BNS still has no elder-specific offence
Ageism	"Dispel negative images, myths and stereotypes"	Now a **formal WHO action area** under the Decade of Healthy Ageing

EXAMINER'S TIP — VERBATIM MARK-EARNING LINES

• *"The goal of NPOP is to help older persons live the last phase of their life with purpose, dignity and peace."*
• *"Older women face triple neglect and discrimination on account of gender, widowhood and age."*
• *"NPOP explicitly required public hospitals to set up geriatric wards and separate counters — NPHCE 2010 is simply the operationalisation of that paragraph."*
• *"Institutional care is to be the last resort — the policy's stated preference is ageing in place."*
• *"NPOP mandated a detailed review every three years as a public document discussed in a National Convention; this has not happened, which is the central accountability failure."*
• *"NPOP promised periodic revision of pension so that inflation does not deflate its real purchasing power — IGNOAPS at ₹200 a month is the clearest evidence that this promise was not kept."*

EXTRA POINTS TO REMEMBER

• **Six areas of intervention:** Financial security • Health care & nutrition • Shelter • Education • Welfare • Protection of life and property.
• **Six guiding principles:** affirmative action • life-cycle continuum • age-integrated society • older persons as a resource • empowerment • partnership.
• **Numbers:** 14.8 m widows vs 4.5 m widowers (1991) • 12% of the electorate • 10% of houses earmarked • 326 million projected for 2050.
• **NCOP 1999 (39 members) → NCSrC 2012. NPSrC 2011 = never notified.**
• **Vayoshreshtha Samman:** 1 October • 13 categories • 15 awardees • ₹2.5 lakh / ₹5 lakh.
• **SCSS:** 8.2% quarterly • ₹30 lakh cap • 5+3 years • 80C • 80TTB ₹50,000.

Q5. Social Security System for Older People in India

SOURCE STATEMENT

Pathy's 6th edition does NOT describe the Indian social security system. Chapter 4 covers only the general problem of old-age poverty and US Social Security data; *Essentials* Ch. 15 covers US Medicare, Medicaid, Title XX and the Older Americans Act.

All India-specific content is from Government of India, ILO and PFRDA sources. Textbook concepts are attributed where used.

A. Concept and Framework

1. **Definition.** Social security is the protection society provides against economic and social distress caused by loss or reduction of income. The **ILO Social Security (Minimum Standards) Convention, 1952 (No. 102)** recognises **nine branches**, of which **old-age benefit, survivors' benefit, invalidity benefit and medical care** are relevant to the elderly.
2. **Constitutional basis (must quote). Article 41** — the State shall make effective provision for **securing the right to public assistance in old age, sickness and disablement**; **Article 43** — living wage and conditions ensuring a decent standard of life; **Article 47** — raising nutrition and standard of living and improving public health. **Social security is in the Concurrent List (Entry 23)**.
3. **The Indian problem in one line:** India is **"growing old before it grows rich,"** and around **90% of the workforce is in the unorganised sector** with no employer-linked pension. Social security must therefore be delivered mainly through **tax-financed social assistance**, not contributory insurance.
4. **Why this matters clinically.** The India Ageing Report 2023 found **over 40% of India's elderly in the poorest wealth quintile** and **18.7% with no income at all**, with a rising predominance of **widowed, highly dependent very old women**. **Pathy's** makes the same point globally: **older women, particularly those who did not marry, are at the highest risk of poverty**, being least likely to qualify for national or occupational pensions, and **longer life expectancy means women are at increased risk of spending down their savings**.
5. **Useful classification (World Bank multi-pillar model) — use this to structure your answer:**
   - **Pillar 0 — Social assistance:** non-contributory, tax-funded, means-tested (NSAP/IGNOAPS)
   - **Pillar 1 — Social insurance:** mandatory, contributory, formal sector (EPS-95, OPS/UPS)
   - **Pillar 2 — Mandatory defined-contribution:** NPS
   - **Pillar 3 — Voluntary savings:** APY, SCSS, PMVVY, insurance
   - **Pillar 4 — Informal: family support, co-residence, health care, housing** — still the dominant pillar in India

B. Pillar 0 — Social Assistance (for the poor elderly)

6. **National Social Assistance Programme (NSAP), 1995** — Ministry of Rural Development, the direct fulfilment of Article 41. Five components: **IGNOAPS** (old age), **IGNWPS** (widow, 40–79 years), **IGNDPS** (disability), **NFBS** (lump sum on death of breadwinner), and **Annapurna** (**10 kg free foodgrain per month** to destitute elderly not covered by IGNOAPS).
7. **IGNOAPS — the flagship, and its biggest weakness.** Central assistance is **₹200 per month above 60 and ₹500 per month above 80** for BPL senior citizens; States add top-ups (Tamil Nadu, Andhra Pradesh, Delhi pay considerably more). **Exam point: the central share has not been revised in over a decade, despite NPOP 1999 explicitly promising that "the rate of monthly pension will need to be revised at intervals so that inflation does not deflate its real purchasing power."**
8. **Food and in-kind security: National Food Security Act 2013** (subsidised grain through PDS), **Antyodaya Anna Yojana**, and **community kitchens/Amma canteens**. NPOP 1999 directed that **the PDS should reach all persons aged 60+ below the poverty line**.

C. Pillars 1 & 2 — Social Insurance for the Organised Sector

9. **EPFO schemes (private/organised sector): EPF** (12% employee + 12% employer); **EPS-95**, funded by **8.33% of the employer's share**, pension after **10 years of service and age 58** (early pension from 50 at a reduced rate), formula = **(Pensionable Salary × Pensionable Service) ÷ 70**; **EDLI** (deposit-linked insurance); and **ESI** (medical care and cash benefits for insured workers).
10. **The EPS-95 adequacy problem (guaranteed discussion point).** The **minimum EPS pension has remained ₹1,000 per month since 1 September 2014**, while unions and pensioners' associations demand **₹7,500**. A proposal to raise the **pensionable wage ceiling from ₹15,000 to ₹25,000** — benefiting over 6.5 crore employees — is under review, but **no revision has been notified**.

11. **Government employees — three generations of schemes: Old Pension Scheme (OPS)** — defined benefit, **50% of last drawn pay**, for those recruited **before 1 January 2004**; **National Pension System (NPS)** — defined contribution, market-linked, mandatory for those joining **on or after 1 January 2004**, regulated by **PFRDA**; and **Unified Pension Scheme (UPS)** — from **1 April 2025**.
12. **Other formal-sector entitlements: Payment of Gratuity Act, 1972** (15 days' wages per year of service); **commutation of pension**; **leave encashment**; and contributory health cover for retirees — **CGHS, ECHS, Railway and state government health schemes**.

D. Pillar 3 — Voluntary and Market-Based Instruments

13. **Atal Pension Yojana (APY) — the main instrument for the unorganised sector.** Launched **9 May 2015**, administered by PFRDA, voluntary and contributory, open to citizens **aged 18–40 who are not income-tax payers**, guaranteeing a **fixed monthly pension of ₹1,000 to ₹5,000 after age 60**, the **same pension to the spouse after death**, and **return of the accumulated corpus to the nominee** after both die.
14. **Savings and annuity products: SCSS** — **8.2% p.a. paid quarterly**, age **60+ (55+ VRS, 50+ defence)**, maximum **₹30 lakh**, tenure **5 years extendable by 3**, with **80C deduction** and TDS only above ₹1 lakh interest. Also **PMVVY**, **NPS "All Citizen" model**, **Reverse Mortgage Loan**, and **Post Office Monthly Income Scheme**.
15. **Tax expenditure as social security:** higher basic exemption of **₹3 lakh (60–79) and ₹5 lakh (80+)** under the old regime; **Section 80TTB — ₹50,000 deduction on interest income**; **no advance tax if there is no business income (Section 207)**; plus **Section 80D** and **Section 80DDB**.

E. Health Social Security

16. **Health protection is the fastest-moving pillar. AB PM-JAY** gives **₹5 lakh per family per year**; the **Ayushman Vay Vandana Card (October 2024)** extends **₹5 lakh cover to all citizens aged 70+ irrespective of income**, with an additional top-up for PM-JAY families. Service delivery is through **NPHCE** (AAM-HWC → PHC → CHC → District Hospital → Regional Geriatric Centre). **This closes the gap NPOP 1999 identified when it said health insurance for the elderly "will be given high priority."**

F. Pillar 4 — Legal and Informal Social Security

17. **The family is legally the first line of social security. Section 125 CrPC (now Section 144 BNSS)** and the **Hindu Adoptions and Maintenance Act, 1956** create a duty to maintain parents. The **MWPSC Act, 2007** operationalises this through **Maintenance Tribunals** (cap **₹10,000/month**, disposal within **90 days**), makes **abandonment a punishable offence**, and allows **transfer of property to be declared void** if the transferee fails to provide basic amenities.
18. **Welfare services as social security: AVYAY** (IPSrC old-age homes, **RVY free assistive devices for BPL/income ≤ ₹15,000 per month**, SCOPE, SAGE), **Elderline 14567**, and the **Senior Citizens' Welfare Fund**.

G. Coverage, Gaps and Critique (examiners reward critique)

19. **Structural weaknesses of the Indian system:**
    - **Fragmentation** — spread across MoSJE, MoRD, MoHFW, Labour and Finance, with **no single accountable authority**
    - **Inadequacy** — IGNOAPS at ₹200/month and EPS-95 at ₹1,000/month are **far below any subsistence line**
    - **Exclusion** — the informal workforce, **unpaid women workers, agricultural labourers and migrants** fall outside contributory schemes
    - **Gendered gap** — ILO data show **older women are less likely to receive a contributory pension than men**; NPOP 1999 warned of **"triple neglect and discrimination on account of gender, widowhood and age"**
    - **No long-term care financing** — India has **no statutory LTC or disability-linked care allowance** for frail elders
    - **Targeting errors** — BPL identification excludes many genuinely poor elderly
20. **The one-line summary for the exam:** India's old-age social security is **a narrow contributory core for the organised minority, a thin tax-financed assistance floor for the poorest, and a large voluntary/market segment in between — with the family still carrying most of the burden.** The 2024–2026 reforms (Labour Codes, UPS, Vay Vandana Card) are the first serious attempt to widen that base.

Summary Table — The Indian Old-Age Social Security Architecture

Pillar	Scheme	Ministry / Regulator	Benefit	Who is covered
0 — Assistance	IGNOAPS (NSAP)	Rural Development	₹200/month (60–79), ₹500/month (80+)	BPL elderly
	Annapurna, NFSA/PDS	Rural Dev / Food	10 kg grain/month; subsidised grain	Destitute / priority households
1 — Insurance	EPS-95 → **EPS 2026**	Labour / EPFO	Pension after 10 yrs, age 58; **min ₹1,000/month**	Organised private sector
	OPS	Finance	50% of last drawn pay	Govt employees pre-2004
2 — Defined contribution	NPS	PFRDA	Market-linked corpus + annuity	Govt post-2004, corporates, all citizens
	UPS (from 1 Apr 2025)	Finance / PFRDA	50% of last 12 months' average basic pay; **min ₹10,000/month**	Central govt employees under NPS
3 — Voluntary	APY	PFRDA	₹1,000–₹5,000/month guaranteed	Unorganised workers 18–40
	SCSS, PMVVY, Reverse Mortgage	Finance / LIC / Banks	8.2% quarterly; annuity; home equity income	Any senior citizen with assets
Health	PM-JAY + **Vay Vandana Card**	MoHFW / NHA	₹5 lakh/year; **universal for 70+**	Poor + all citizens aged 70+
	CGHS / ECHS / ESI	Various	Contributory medical care	Government / defence / insured workers
4 — Informal & legal	MWPSC Act 2007, Sec 144 BNSS	Law & Justice	Legally enforceable maintenance	All senior citizens
	AVYAY, Elderline 14567	MoSJE	Shelter, devices, helpline	Indigent / BPL elderly

LATEST UPDATES (2023–PRESENT)

1. The Four Labour Codes came into force on 21 November 2025 — the biggest reform in decades. The Government notified the **Code on Wages 2019, Industrial Relations Code 2020, Code on Social Security 2020 and OSH Code 2020**, rationalising **29 existing central labour laws**. The **Code on Social Security, 2020 consolidates NINE existing social security laws** into a single framework covering organised, unorganised, gig and platform workers, extending **provident fund, gratuity, life and disability insurance, health and maternity benefits**. **For the first time in India, social security benefits have been extended to unorganised, gig and platform workers under Sections 113 and 114.** Aggregators must contribute **1–2% of turnover (capped)** to a dedicated **Social Security Fund**; **draft central rules notified 30 December 2025** propose eligibility after **90 days with one aggregator or 120 days across multiple aggregators** in a financial year, with **Aadhaar-linked registration through e-Shram ensuring portability**.

2. Employees' Pension Scheme (EPS), 2026 has replaced EPS-95. Notified under the Social Security Code 2020 — **the biggest legal overhaul of the scheme in more than three decades**, affecting **over 30 crore EPFO subscribers**. Core features retained: **minimum pension remains ₹1,000 per month**, formula unchanged. Key procedural reform: **EPFO must settle pension claims within 20 days, failing which 12% annual interest is payable**. **Exam framing: the legal architecture has modernised, but the benefit level has not.**

3. Unified Pension Scheme (UPS) — effective 1 April 2025. Provides an **assured pension of 50% of the average basic pay of the last 12 months for a minimum qualifying service of 25 years** (proportionate down to 10 years); **assured family pension at 60%**; **assured minimum pension of ₹10,000 per month** after at least 10 years; **inflation indexation with Dearness Relief based on AICPI-IW**; and a **lump sum of one-tenth of monthly emoluments for every completed six months of service, in addition to gratuity**. About **23 lakh central government employees** are eligible. Contributions: **employee 10% of Basic + DA, matching government 10%, plus an additional ~8.5% into a pool corpus**. Uptake has been low — **118,195 employees (4.3% of 2.76 million NPS subscribers) opted in as of July 2025**, and a **one-time switch back to NPS** has been allowed.

4. India's social protection coverage has doubled — ILO World Social Protection Report 2024–26. Coverage by **at least one social protection cash benefit rose from 24.4% (2021) to 48.8% (2024)**; per MoLE, **nearly 920 million people (65% of the population)** are covered in cash or in kind. India's progress contributed a **5% increase in global social protection coverage**. Globally the world has crossed the halfway mark for the first time, with **52.4% covered**, and countries spend on average **12.9% of GDP on social protection excluding healthcare**.

5. Scale-up of unorganised-sector instruments. Atal Pension Yojana crossed 9 crore gross enrolments as of 21 April 2026, with **over 1.35 crore new subscribers in FY26 — the highest annual enrolment since launch**; in FY 2024-25, **55% of new APY enrolments were women**. The **e-Shram portal** (launched 26 August 2021) had **over 30.68 crore registrations by March 2025, of which 53.68% were women**.

6. Direction of travel — a single portable social security identity. The **draft Shram Shakti Niti, 2025** proposes a **Universal Social Security Account merging EPFO, ESIC, e-Shram and PM-JAY into one portable identity**. India also launched a **Social Protection Data Pooling Exercise on 19 March 2025**, Phase 1 covering 10 states including Tamil Nadu and processing over 200 crore welfare records. **NITI Aayog (2024)** recommends a **regulatory base interest rate on senior-citizen deposits**, noting most seniors depend on savings income which variable rates can push **below sustenance level**.

CURRENT PRACTICE UPDATE

Element	Position as classically taught	Position in 2026
Statutory framework	Nine separate Acts (EPF 1952, ESI 1948, Gratuity 1972, Maternity Benefit 1961, etc.)	**All consolidated into the Code on Social Security, 2020, in force from 21 November 2025**
EPS	EPS-95	**EPS 2026** notified under the Code; minimum pension still ₹1,000/month; **20-day claim settlement with 12% penal interest**
Government pension	OPS (pre-2004) vs NPS (post-2004)	**Third option — UPS from 1 April 2025**: 50% assured, min ₹10,000/month, family pension 60%, AICPI-IW indexed
Gig and platform workers	Entirely outside social security	**Legally recognised for the first time**; aggregator contribution of 1–2% of turnover to a Social Security Fund
Coverage	Often quoted as "only ~10–12% of workforce covered"	**48.8% covered by at least one cash benefit (ILO 2024); ~65% including in-kind**
Health protection	Means-tested or employment-linked only	**Universal, age-based ₹5 lakh cover for all 70+ since October 2024**
IGNOAPS	₹200 / ₹500 per month central share	**Unchanged** — the clearest failure of indexation in the system
Long-term care	Not addressed	**Still no statutory LTC financing** — flagged by NITI Aayog and the UN Decade
Identity / portability	Scheme-wise, non-portable	Moving toward a **Universal Social Security Account** (draft Shram Shakti Niti 2025)

EXAMINER'S TIP — VERBATIM MARK-EARNING LINES

- *"India is growing old before it grows rich — with roughly 90% of the workforce informal, contributory insurance can never be the primary instrument."*
- *"I would classify Indian old-age social security using the World Bank multi-pillar model: Pillar 0 assistance, Pillar 1 insurance, Pillar 2 defined contribution, Pillar 3 voluntary and Pillar 4 informal — in India, Pillar 4, the family, remains dominant."*
- *"The Code on Social Security 2020, in force since 21 November 2025, extends social security to gig and platform workers for the first time under Sections 113 and 114."*
- *"India's social protection coverage doubled from 24.4% in 2021 to 48.8% in 2024, contributing a 5% rise in global coverage."*
- *"The legal architecture has modernised, but the benefit levels have not: IGNOAPS at ₹200 and EPS at ₹1,000 a month remain far below any subsistence line."*

EXTRA POINTS TO REMEMBER

- **ILO Convention 102 (1952)** — nine branches of social security.
- **Key dates:** NSAP 1995 • EPS 1995 • NPS 1 Jan 2004 • APY 9 May 2015 • e-Shram 26 Aug 2021 • UPS 1 Apr 2025 • Labour Codes 21 Nov 2025.
- **EPS formula:** (Pensionable Salary × Pensionable Service) ÷ 70; employer's **8.33%** funds it; wage ceiling **₹15,000** (proposal to raise to ₹25,000).
- **UPS numbers:** 50% / 25 years / 60% family pension / ₹10,000 minimum / AICPI-IW indexed / ~23 lakh eligible.
- **APY:** age 18–40 • non-taxpayers • ₹1,000–₹5,000 • spouse continuation • corpus to nominee • **9 crore enrolments**.
- **ILO 2024:** global coverage **52.4%**; India **48.8%** cash / **~65%** including in-kind; average spend **12.9% of GDP**.

Q6. Role of Government in Providing Healthcare and Economic Security for Older People

SOURCE STATEMENT

Pathy's does not describe the Indian government's role. It contributes the **conceptual and ethical framework** (ageism, age discrimination in resource allocation, social determinants of ageing); *Essentials of Clinical Geriatrics* (Ch. 15 & 20) contributes the **health-services and rationing framework**. Both are attributed below.
All India-specific programme, financing and legal content is from Government of India, ILO and WHO sources.

A. Why the Government Must Act (Justification)

1. **Constitutional obligation.** **Article 41** directs the State to secure **the right to public assistance in old age, sickness and disablement**; **Article 47** makes raising nutrition and improving public health a primary duty of the State. **Social security is a Concurrent List subject**, so both Centre and States are answerable.
2. **Market failure argument.** Private insurance fails older people because **premiums rise steeply with age, pre-existing conditions are excluded, and chronic and custodial care are not covered**. **Essentials** makes exactly this point about long-term care insurance: it is **affordable when bought young but becomes very expensive by age 75**, when the need is greatest, and those who need it most may be **ineligible because of prior conditions**. Only the State can pool risk across the whole life course.
3. **Demographic urgency.** India's 60+ population will rise from **10.5% (149 million) in 2022 to 20.8% (347 million) by 2050**, the **80+ group will grow by 279%**, and the **elderly will outnumber children by 2046** — while **over 40% of the elderly are in the poorest wealth quintile and 18.7% have no income at all**.
4. **The two problems are one problem.** Older people are **poor because they are sick, and sick because they are poor**. Nationally, older adults spend about **17.4% of household consumption expenditure on healthcare**, and treatment expenditure is demonstrably higher above 60 than in younger age groups, regardless of income level. **Government action on health *is* action on economic security.**

B. The Six Roles of Government (answer skeleton)

5. **(i) Policy-maker / Steward** — NPOP 1999, National Health Policy 2017, NITI Aayog Senior Care Reforms 2024; sets goals, targets and accountability.
6. **(ii) Legislator** — MWPSC Act 2007, Code on Social Security 2020, Section 144 BNSS; creates enforceable rights.
7. **(iii) Financier / Purchaser** — tax-funded programmes, PM-JAY, pensions, subsidies; pools risk and protects against catastrophic spending.
8. **(iv) Provider** — the public health system from Ayushman Arogya Mandir to Regional Geriatric Centre; old-age homes through IPSrC.
9. **(v) Regulator** — PFRDA, IRDAI, Clinical Establishments Act, drug price control (NPPA), minimum standards for old-age homes.
10. **(vi) Capacity-builder and knowledge-generator** — PG seats in Geriatric Medicine, training modules, **LASI**, National Health Accounts, research funding.

C. Government's Role in Healthcare

11. **Direct service provision through NPHCE (2010).** A five-tier ladder — **Sub-centre/Ayushman Arogya Mandir → PHC (weekly geriatric clinic) → CHC (twice-weekly clinic + physiotherapy) → District Hospital (10-bedded ward with 2 respite beds) → Regional Geriatric Centre (30 beds) and National Centres for Ageing**. The tertiary arm was renamed **Rashtriya Varisth Jan Swasthya Yojana** in 2016–17.
12. **Financial protection through Ayushman Bharat. PM-JAY** gives ₹5 lakh per family per year; the **Ayushman Vay Vandana Card (October 2024)** extends **₹5 lakh cover to every citizen aged 70+ irrespective of income**, with **no waiting period for pre-existing conditions** and roughly **1,961 procedures across 27 specialties**. This is **India's first universal, age-based (not means-tested) health entitlement**.
13. **Convergence with vertical programmes.** The elderly are served by **NP-NCD** (with **CBAC-based screening from age 30**), **NPCB&VI** (cataract, refractive error), **NPPCD** (deafness/hearing aids), **National Mental Health Programme** (depression, dementia), the **National Programme for Palliative Care**, and **free dialysis and free drugs & diagnostics initiatives**.
14. **Human resource development.** NPOP 1999 promised that **"facilities for specialization in geriatric medicine will be provided in the medical colleges"** and that **training in nursing care will include geriatric care** — delivered through **PG seats at RGCs, in-service training and caregiver training for families**.

15. **Evidence that public financing works — the strongest data point in this answer.** National Health Accounts estimates released in September 2024 show **out-of-pocket expenditure fell from 64.2% of total health expenditure in 2013-14 to 39.4% in 2021-22**, while **government health expenditure rose from 1.13% to 1.84% of GDP**, with **over ₹1 lakh crore of savings from PM-JAY alone**. **Per capita government health spending tripled from ₹1,108 to ₹3,169**, government health spending as a share of total government expenditure rose from **3.94% to 6.12%**, and **Social Security Expenditure on health rose from 5.7% to 8.7%**. **Public spending on health has, for the first time, overtaken out-of-pocket spending.**

D. Government's Role in Economic Security

16. **Social assistance (tax-financed, for the poorest). NSAP** delivers **IGNOAPS** (₹200/month for 60–79, ₹500/month for 80+ central share), **IGNWPS**, **IGNDPS**, **NFBS** and **Annapurna** (10 kg free foodgrain/month), supplemented by state top-ups and subsidised grain under the **NFSA 2013**.
17. **Social insurance and pensions (contributory). EPFO** runs **EPF, EPS (minimum pension ₹1,000/month) and EDLI**; government employees are covered by **OPS (pre-2004), NPS (post-2004) and UPS (from 1 April 2025)**. **Atal Pension Yojana** extends a guaranteed **₹1,000–₹5,000 monthly pension** to unorganised-sector workers.
18. **Savings, annuity and tax instruments. SCSS** at **8.2% paid quarterly** (₹30 lakh limit), **PMVVY**, **reverse mortgage**, and tax relief through a **higher basic exemption (₹3 lakh for 60–79, ₹5 lakh for 80+ under the old regime)**, **Section 80TTB (₹50,000)**, **Section 80D** and **Section 80DDB**.
19. **Legal enforcement of family support and welfare services.** The **MWPSC Act 2007** creates Maintenance Tribunals, makes **abandonment punishable** and allows **property transfers to be declared void**; **AVYAY** delivers old-age homes (IPSrC), **free assistive devices under RVY**, and **Elderline 14567**.

E. Limitations, Ethics and Critique (this section earns the top marks)

20. **The unfinished agenda — six concrete gaps:**
    - **Under-financing:** government health spending is still only **~1.9% of GDP against the National Health Policy 2017 target of 2.5% by 2025**, and **OOPE at 39.4% remains above the 35% target**, versus about **13% in the UK and Australia**.
    - **The wrong thing is insured.** Catastrophic spending in India is **more common for outpatient care (47.8% of households) than for hospitalisation (43.1%)**, and **medicines alone account for roughly two-thirds of out-of-pocket spending** — yet outpatient care and medicines **lie outside the flagship hospitalisation scheme**. **Tamil Nadu's pooled free-medicines model is cited as a proven domestic remedy.** This matters most for the elderly, whose costs are chronic and outpatient, not episodic.
    - **Unindexed benefits:** IGNOAPS at ₹200–₹500/month and EPS at ₹1,000/month unrevised for over a decade.
    - **No long-term care system:** no statutory LTC financing, no care-giver allowance, no LTC insurance.
    - **Fragmentation:** delivery split across **five ministries with no single accountable authority**.
    - **The "missing middle":** roughly **400 million Indians remain uninsured** — too rich for PM-JAY, too poor for private cover.
21. **The ethical dimension the examiner is waiting for. Essentials** warns that **"older adults are prime targets for rationing efforts because they consume disproportionately large amounts of medical care and because they are seen as having already lived their lives."** It cautions that **any measure using life expectancy is inevitably biased against the elderly**, and that a measure relying on dependency as the outcome **implies that those who are dependent no longer count — "by such logic, disability is equivalent to death."** It concludes that **as advocates for their patients, geriatricians must be extremely vigilant** to how such terms are used. **Pathy's** makes the parallel point that the UK **NSF's Standard One was "rooting out age discrimination,"** and that **ageism leads older people to be denied beneficial investigations and treatments**. **A government's role is therefore not only to fund and provide, but to protect older people from being rationed out of the system.**

Framework Table — Government Role Mapped to Instrument

Role	Healthcare instrument	Economic security instrument
Policy / Steward	National Health Policy 2017; NPHCE guidelines	NPOP 1999; NITI Aayog Senior Care Reforms 2024
Legislator	MWPSC Act Chapter IV (medical care)	MWPSC Act; Code on Social Security 2020; Sec 144 BNSS
Financier	PM-JAY + **Ayushman Vay Vandana Card (70+)**; free drugs & diagnostics	NSAP/IGNOAPS; EPS; UPS; APY; SCSS; tax reliefs
Provider	AAM-HWC → PHC → CHC → DH (10 beds) → RGC / NCA	IPSrC old-age homes; RVY assistive devices
Regulator	Clinical Establishments Act; NPPA drug price control	PFRDA; IRDAI; standards for old-age homes
Capacity builder	PG seats in Geriatric Medicine; staff training	Elderline 14567; SACRED/SCOPE re-employment
Knowledge generator	LASI; National Health Accounts; HMIS	Social Protection Data Pooling Exercise; e-Shram

LATEST UPDATES (2023–PRESENT)

1. Public spending has overtaken out-of-pocket spending — a landmark. National Health Accounts estimates for 2020-21 and 2021-22, released **25 September 2024**, show **OOPE down from 64.2% (2013-14) to 39.4% (2021-22)** and **government health expenditure up from 1.13% to 1.84% of GDP**, with NITI Aayog attributing **more than ₹1 lakh crore of savings to PM-JAY**. Per capita government health spending **tripled from ₹1,108 to ₹3,169**, and social security expenditure on health rose from **5.7% to 8.7%** of total health expenditure. **However, both National Health Policy 2017 targets — GHE of 2.5% of GDP and OOPE down to 35% — have been missed.**

2. Universal age-based health entitlement (October 2024). The **Ayushman Vay Vandana Card** gives **₹5 lakh of free annual cover to every citizen aged 70 and above irrespective of income**, with a **₹5 lakh top-up for seniors already in PM-JAY families**. **9.67 million cards issued by 31 December 2025**; by January 2026 nearly **one crore cards**, enabling about **eight lakh elderly citizens** to access free treatment, with **1.80 lakh Ayushman Arogya Mandirs operational**.

3. Social protection coverage has doubled — ILO World Social Protection Report 2024–26. Coverage by at least one social protection cash benefit rose from **24.4% (2021) to 48.8% (2024)**, with **nearly 920 million people (65% of the population)** covered in cash or in kind, contributing a **5% increase in global coverage**.

4. The legal architecture of economic security was rewritten (2025–2026). The **four Labour Codes came into force 21 November 2025**, consolidating **29 central labour laws**; the **Code on Social Security 2020 merges nine Acts** and extends benefits to **unorganised, gig and platform workers (Sections 113 and 114)**, with aggregators contributing **1–2% of turnover to a Social Security Fund**. The **Unified Pension Scheme** took effect **1 April 2025** (50% of last 12 months' average basic pay after 25 years, **minimum ₹10,000/month**, family pension 60%, AICPI-IW indexed), and **EPS 2026** replaced EPS-95 with a **20-day claim-settlement rule carrying 12% penal interest**.

5. Policy direction — what governments are being told to do next. NITI Aayog (February 2024) recommends **strengthening geriatric healthcare within the existing system**, extending comprehensive primary care through **AB-HWCs**, promoting **health literacy among seniors and caregivers**, expanding **home health care, mental health services and Poshan Abhiyan for senior citizens**, formal **recognition of the care economy and care workers, especially women**, a **national portal for senior care**, and a **regulatory mechanism to set a viable base interest rate on senior-citizen deposits**.

6. International benchmark. The **UN Decade of Healthy Ageing (2021–2030)** frames government responsibility under **combating ageism, age-friendly environments, integrated care and long-term care**. Its **2023 progress report** found **fewer than one-third of countries have adequate resources**, with only about **1 in 4 having sufficient resources for integrated care and 1 in 3 for long-term care**. The **WHO ICOPE Handbook 2nd edition (January 2025)** gives governments an operational tool — a **four-step primary-care pathway** screening **six intrinsic-capacity domains**.

CURRENT PRACTICE UPDATE

Domain	Position as classically taught	Position in 2026
Health financing	OOPE ~60–65%; India a classic high-OOPE system	**OOPE down to 39.4%**; public spending now exceeds OOPE — but still **1.9% of GDP vs the 2.5% target**
Entitlement basis	Means-tested (BPL cards, PM-JAY SECC criteria)	**Age-based universal entitlement for 70+ since October 2024**
What is insured	Hospitalisation	Outpatient care and medicines — **two-thirds of OOPE** — still **largely outside PM-JAY**; Tamil Nadu's pooled free-medicines model is the proposed remedy
Social security law	Nine separate Acts	**Consolidated into the Code on Social Security 2020, in force 21 November 2025**
Gig/informal workers	Entirely excluded	**Legally recognised for the first time**
Government pension	OPS vs NPS	**Third option — UPS from 1 April 2025**
Assessment model	Disease-oriented screening	**WHO ICOPE intrinsic-capacity model (2025)** — not yet adopted in Indian programmes
Long-term care	Family responsibility, reinforced by MWPSC Act	**Still no statutory LTC financing** — a formal action area of the UN Decade
Ageism in rationing	Not formally addressed in Indian policy	Now a **named global action area**; still absent from Indian legislation

EXAMINER'S TIP — VERBATIM MARK-EARNING LINES

• *"The government acts in six roles: steward, legislator, financier, provider, regulator and capacity-builder — and for older people all six are needed simultaneously."*
• *"Out-of-pocket expenditure fell from 64.2% in 2013-14 to 39.4% in 2021-22, so public spending on health has for the first time overtaken out-of-pocket spending — but both NHP 2017 targets have been missed."*
• *"India insures the wrong thing: catastrophic spending is commoner for outpatient care than for hospitalisation, and medicines are about two-thirds of out-of-pocket spending — precisely the elderly's cost pattern."*
• *"Older adults are prime targets for rationing because they consume disproportionately large amounts of medical care and are seen as having already lived their lives."*
• *"By the logic of dependency-based outcome measures, disability is equivalent to death — geriatricians must be extremely vigilant to how such terms are used."*

EXTRA POINTS TO REMEMBER

• **NHA key numbers:** OOPE 64.2% → 39.4% • GHE 1.13% → 1.84% of GDP • per capita ₹1,108 → ₹3,169 • GHE share of govt spending 3.94% → 6.12%.
• **NHP 2017 targets:** GHE **2.5% of GDP** and OOPE down to **35%** — **both missed**.
• **Catastrophic expenditure:** outpatient **47.8%** of households vs hospitalisation **43.1%**; medicines **~two-thirds** of OOPE.
• **"Missing middle" = ~400 million uninsured Indians.**
• **Elderly spend ~17.4% of household consumption expenditure on healthcare.**
• **The ethical anchor:** Essentials on rationing + Pathy's NSF Standard One ("rooting out age discrimination").

Q7. Integrated Care of Older People (ICOPE)

SOURCE STATEMENT — THIS TIME THE TEXTBOOK DOES COVER IT

Pathy's 6th edition devotes an entire chapter to this subject: Chapter 100, "WHO Integrated Care for Older People (ICOPE)," pages 1272–1281, by **Catherine Takeda, Sophie Guyonnet, Bruno Vellas and Yuka Sumi** (Volume 2, Section 12: Functional Disorders and Rehabilitation). **Intrinsic capacity and functional ability** are also covered in **Chapters 2, 4, 5 and 99**.
Because the chapter summarises WHO's own documents, detailed guideline recommendations and care-pathway steps are sourced from **WHO ICOPE Guidelines (2017), ICOPE Handbook 1st ed. (2019) and 2nd ed. (2025)**.

A. Definitions (get these exact — they are the whole answer)

1. **Healthy ageing** is the process of developing and maintaining the **functional ability** that enables well-being in older age. Pathy's: *"functional ability — retaining autonomy and independence as people age — is the cornerstone of healthy ageing, a term established by the WHO in its first World Report on Ageing and Health."*
2. **Intrinsic capacity (IC)** is **"a composite of all of an individual's physical and mental capacities"** (Pathy's Ch. 5) — WHO: **"all the mental and physical capacities that a person can draw on, including their ability to walk, think, see, hear and remember."** Pathy's calls it **"a key determinant of functional ability."**
3. **Functional ability = Intrinsic capacity + Environment + the interaction between them.** This is the central equation of ICOPE. Two people with the same intrinsic capacity can have very different functional ability depending on housing, transport, family support and services.
4. **ICOPE** is WHO's approach to **provide a continuum of integrated care that reorients health and social services towards more person-centred and coordinated care**, aiming to **optimise intrinsic capacity and functional ability in older age**.

B. Rationale — Why It Was Developed

5. **The disease-centred model fails older people.** ICOPE **promotes person-centred care that strengthens older people's intrinsic capacity and their ability to function where they live — a shift away from specialised medical treatment for each disease or condition**. With multimorbidity, treating each disease separately produces polypharmacy, fragmented care and no gain in function.
6. **Declines in intrinsic capacity share common causes, so they can be tackled together.** WHO's justification: **"highly intensive strength training is the key intervention necessary to prevent and reverse mobility impairments, but it also indirectly protects the brain against depression and cognitive impairment, and prevents falls. Nutrition enhances the effects of exercise and has a direct impact on increasing muscle mass and strength."** One integrated package fixes several problems at once.
7. **Function predicts outcome better than disease.** Pathy's notes that **gait speed predicts mortality in older adults better than chronic diseases like hypertension**, that **multimorbidity is not the most important factor modulating individual domains of intrinsic capacity**, and that **older adults with handgrip strength above muscle-weakness thresholds have lower odds of adverse events across most IC domains and a lower hospitalisation rate in men** (Ramirez-Vélez et al.).
8. **Scale of the problem: mobility impairment is found in 39% of people over 65 years — more than three times the rate in the working-age population**.

C. Evolution of ICOPE (timeline = easy marks)

9. **2015** — WHO *World Report on Ageing and Health* introduces **healthy ageing, intrinsic capacity and functional ability**.
   2016 — *Global Strategy and Action Plan on Ageing and Health*; Member States **"recognized a pressing need to develop integrated, community-based approaches to prevent declines in intrinsic capacity."**
   2017 — **ICOPE Guidelines** with **13 evidence-based recommendations**.
   2019 — **ICOPE Handbook (1st edition)** with a **5-step care pathway**, plus the **ICOPE Implementation Framework**.
   2021–2030 — implementing **ICOPE as part of Universal Health Coverage becomes one of the four action areas of the UN Decade of Healthy Ageing**.
   29 January 2025 — **ICOPE Handbook (2nd edition)** with a streamlined **4-step pathway**.

D. The Six Intrinsic Capacity Domains and How They Are Screened

10. To operationalise intrinsic capacity, WHO selected the domains **most closely associated with care dependency: mobility, cognition, psychological capacity (depressive symptoms), vitality (malnutrition), and sensory**

capacity (hearing and vision). These are screened with **very simple bedside tests requiring no equipment** — in an Indian community pilot, **cognitive decline was determined by difficulty with orientation to time and place or word recall; mobility by the chair-rise test; and malnutrition by recent weight loss or loss of appetite** — together with **sensory testing, depressive symptoms, health risks, and social care and support needs**, plus recording of **caregiver strain and risk of elder abuse**.

Domain	Simple screening question/test	If positive → in-depth assessment
Cognition	Orientation to time and place; recall of three words	MMSE/MoCA, dementia workup, reversible causes
Mobility	**Chair-rise test** (rise 5 times from a chair without arms)	SPPB, gait speed, sarcopenia assessment, falls risk
Vitality / Nutrition	Unintentional weight loss? Loss of appetite?	MNA, BMI, dietary and protein assessment
Vision	Difficulty with distance/near vision?	Visual acuity, refraction, cataract, glaucoma, retinopathy
Hearing	Whisper test / difficulty hearing in a noisy room	Audiometry, wax removal, hearing aid provision
Psychological	Feeling down, depressed or hopeless? Loss of interest?	PHQ-9/GDS, mhGAP-based assessment
Plus	Urinary incontinence; falls; social support; caregiver strain	Targeted pathway for each

E. The 13 Recommendations of the 2017 ICOPE Guidelines

11. **Three modules: Module I — declines in intrinsic capacity** (mobility loss, malnutrition, visual impairment and hearing loss, cognitive impairment and depressive symptoms); **Module II — geriatric syndromes associated with care dependency** (urinary incontinence and risk of falls); **Module III — caregiver support**.
12. **Delivered as six priority actions: improve musculoskeletal function, mobility and vitality; maintain sensory capacity; prevent severe cognitive impairment and promote psychological well-being; manage age-associated conditions such as urinary incontinence; prevent falls; and support caregivers**.

Priority action	Key recommendations
Mobility & vitality	**Multimodal exercise** including progressive strength/resistance training for mobility loss; **oral supplemental nutrition with dietary advice** for malnutrition
Sensory capacity	Routine **screening for visual impairment with timely comprehensive eye care**; **screening followed by provision of hearing aids** *(strong recommendation, low-quality evidence)*
Cognition & mood	**Cognitive stimulation** for cognitive impairment, with or without a formal dementia diagnosis *(conditional)*; **brief, structured psychological interventions per WHO mhGAP**, delivered by professionals understanding older people's mental health *(conditional)*
Urinary incontinence	**Prompted voiding** for cognitive impairment; **pelvic floor muscle training**, alone or with bladder-control strategies and self-monitoring, for urge, stress or mixed incontinence in older women
Falls	**Medication review and withdrawal** of unnecessary or harmful drugs; **multimodal exercise** (balance, strength, flexibility, functional training); **home modification** after specialist assessment; **multifactorial interventions** combining assessment with tailored interventions
Caregivers	Interventions to **support caregiving and prevent caregiver strain** — respite, training, psychological support

F. The ICOPE Care Pathway

13. **The original 5-step pathway (Handbook 1st edition, 2019): Step 1** — screen for loss in the domains of intrinsic capacity; **Step 2** — assess health and social care needs (person-centred assessment); **Step 3** — develop a personalised care plan; **Step 4** — referral and monitoring of the care plan; **Step 5** — engage communities and support caregivers.
14. **The current 4-step pathway (Handbook 2nd edition, 2025) — learn this version: (1) basic assessment and community-level interventions, (2) in-depth assessment, (3) development of a personalised care plan, and (4) implementation and monitoring.** In practice: a **basic assessment to identify declines in intrinsic capacity plus generic health-promotion advice**; an **in-depth evaluation for those flagged**; a **personalised care plan with multidisciplinary input**; and **ongoing monitoring with specialist referral as needed**.
15. **The pathway is deliberately designed to work without geriatricians.** It **empowers general practitioners but also trained non-experts such as community health workers or nurses to implement it without requiring specialised geriatric expertise, making it accessible in diverse contexts from urban clinics to rural outreach programmes**. By prioritising **person-centred goals — what matters most to the individual** — it integrates **mobility, nutrition, sensory function, cognition, mood and vitality into actionable plans that promote functional ability and independence and reduce unnecessary hospitalisations**. **This is the single most important point for an Indian answer.**

G. System-Level Requirements and Support Tools

16. **ICOPE is not just a clinical tool — it requires system redesign.** The **ICOPE Implementation Framework** addresses **governance and financing, workforce training, service delivery models, information systems, and community engagement**. WHO also provides the **ICOPE Handbook App**, giving an **interactive step-by-step approach following the care pathways and generating a printable summary of the assessments, interventions and care plan**, and the **mAgeing programme**, delivering **health information, advice and reminders based on the ICOPE guidelines through basic mobile-phone technology**.

H. Evidence, Implementation and Critique

17. **Real-world implementation is well under way. Pilot programmes in India, Mexico and Vietnam have demonstrated feasibility in resource-constrained environments, where community health workers use simplified screening tools to identify and manage declines, leading to improved functional outcomes and reduced healthcare utilisation.** In France, the **INSPIRE ICOPE-CARE programme in Occitania has enrolled thousands of seniors since 2020, integrating digital monitoring with primary-care pathways; by October 2024 over 3,000 individuals were actively followed, with high adherence and evidence of delayed functional loss**.
18. **ICOPE domains track closely with frailty.** In a validation study of 210 participants, **39.0% were prefrail and 28.6% frail**; **mobility loss and depressive symptoms were strongly linked to both prefrailty and frailty**, and **malnutrition and hearing loss were significantly associated with frailty**.
19. **State the limitations honestly — this earns marks.** Pathy's itself is cautious: in the cachexia chapter it notes that the ICOPE guidelines **"propose screening and management strategies for older subjects at risk for unhealthy ageing,"** that **"weight loss and anorexia are grouped into the malnutrition item, calling for integrative management of nutritional risk,"** but that **"the impact of this program is not known at this time."** Other limitations: **no consensus on how to score or combine intrinsic capacity into a single metric**; **screening tools have limited sensitivity and specificity**; **screening is useless without downstream services to refer to**; **long-term outcome and cost-effectiveness data are still maturing**; and **social care and caregiver support are the hardest components to fund**.
20. **Application to India — say this explicitly.** ICOPE is an almost perfect fit for the Indian delivery platform but **has not yet been formally adopted**. The screening step takes about **five minutes, needs no equipment, and can be done by a CHO or ASHA** at an **Ayushman Arogya Mandir**, feeding positives upward into the **NPHCE ladder** (PHC weekly clinic → CHC rehabilitation unit → District Hospital geriatric ward → Regional Geriatric Centre) and sideways into **NPCB&VI for cataract, NPPCD for hearing aids, NP-NCD for chronic disease, NMHP for depression and dementia, and Rashtriya Vayoshri Yojana for assistive devices**. **Gap to state: NPHCE still uses a simple clinical check-up and standard health card, not the ICOPE intrinsic-capacity screen.**

LATEST UPDATES (2023–PRESENT)

1. ICOPE Handbook, 2nd edition — launched 29 January 2025. WHO released *Integrated Care for Older People: Guidance for Person-Centred Assessment and Pathways in Primary Care*, which **details the four steps of the ICOPE care pathway and includes an expanded first step and a NEW urinary incontinence care pathway**. It **emphasises delivering personalised, comprehensive, coordinated care, supporting a shift from the traditional disease-oriented approach towards more holistic care**. The pathway has been **streamlined from five steps to four** — the most visible change — and the edition **applies the pathway steps to more settings and to persons with a greater range of intrinsic capacities**.

2. Designed explicitly for low-resource settings. The second edition **promotes the integration of more accessible tools and methodologies to facilitate implementation regardless of resource constraints**, and **aims to provide flexibility to ensure feasibility across different countries, contexts and resource settings**, describing **practical care pathways to be adapted for the local context** to detect declines in intrinsic capacity, identify social care and support needs, and develop a personalised care plan, with **training material available in English**.

3. ICOPE is now a formal action area of the UN Decade of Healthy Ageing. Implementing ICOPE as part of Universal Health Coverage is one of the four action areas of the Decade (2021–2030), alongside combating ageism, age-friendly environments and long-term care. WHO's 2023 progress report found **fewer than one-third of countries have adequate resources**, with only about **1 in 4 having sufficient resources for integrated care**.

4. Growing implementation evidence (2024–2026). By **October 2024, the INSPIRE ICOPE-CARE programme in Occitania, France had over 3,000 individuals actively followed, with high adherence and evidence of delayed functional loss**, and **pilot programmes in India, Mexico and Vietnam have demonstrated feasibility in resource-constrained environments with improved functional outcomes and reduced healthcare utilisation**.

5. Alignment with Indian policy direction. The **NITI Aayog position paper (February 2024)** recommends **extending comprehensive primary care for the elderly through AB-Health & Wellness Centres**, **promoting health literacy among seniors and caregivers**, and **expanding home health care and mental health services** — all delivery preconditions for ICOPE. **India has an ICOPE-ready platform (1.80 lakh Ayushman Arogya Mandirs) but no ICOPE mandate.**

CURRENT PRACTICE UPDATE

Element	Pathy's 6th edition (2022) / classical teaching	Current position (2025–2026)
Care pathway	Describes the **ICOPE Guidelines 2017** and the **5-step Handbook (2019)**	**Streamlined to 4 steps** in the 2nd edition (Jan 2025)
Step 1	"Screening" for loss in IC domains	Renamed and expanded to **"basic assessment plus community-level interventions,"** with health-promotion advice built in
Urinary incontinence	Two recommendations (prompted voiding, PFMT)	**A dedicated new care pathway** added in the 2nd edition
Who delivers it	Implicitly primary-care physicians	Explicitly **community health workers and nurses without specialist geriatric training**
Evidence of impact	Pathy's states plainly that **"the impact of this program is not known at this time"**	**Emerging positive data** — INSPIRE ICOPE-CARE (>3,000 followed, delayed functional loss); feasibility in India, Mexico, Vietnam; **definitive RCT-level outcome data still awaited**
Digital support	Not emphasised	**ICOPE Handbook App** and **mAgeing** mobile programme now standard supports
Policy status	A WHO guideline	A **named action area of the UN Decade**, tied to UHC
India	Not addressed	**Not yet adopted within NPHCE**, despite an ideal delivery platform in Ayushman Arogya Mandirs

EXAMINER'S TIP — VERBATIM MARK-EARNING LINES

• *"Functional ability equals intrinsic capacity plus environment plus the interaction between them — that equation is ICOPE in one line."*
• *"ICOPE promotes person-centred care that strengthens intrinsic capacity and the ability to function where people live — a shift away from specialised medical treatment for each disease."*
• *"The six domains are cognition, mobility, vitality, vision, hearing and psychological capacity — chosen because they are most closely associated with care dependency."*
• *"The 2025 second edition streamlines the pathway from five steps to four and adds a urinary incontinence care pathway."*
• *"The pathway can be delivered by trained non-experts such as community health workers or nurses without specialised geriatric expertise — which is exactly why it suits India."*
• *"Pathy's itself concedes the impact of this programme is not known at this time; the effectiveness data are only now emerging."*

EXTRA POINTS TO REMEMBER

• **Timeline:** 2015 World Report • 2016 Global Strategy • 2017 Guidelines (13 recommendations) • 2019 Handbook (5 steps) • 2025 Handbook 2nd ed. (4 steps).
• **Three modules:** IC declines • geriatric syndromes (incontinence, falls) • caregiver support.
• **Six priority actions** and **six IC domains** — do not confuse the two lists.
• **Chair-rise test = mobility screen**; **weight loss/appetite = vitality screen**; **3-word recall + orientation = cognition screen**.
• **Mobility impairment in 39% of over-65s** — three times the working-age rate.
• **Support tools:** ICOPE Handbook App • mAgeing • ICOPE Implementation Framework.
• **Frailty overlap study:** 39.0% prefrail, 28.6% frail; mobility loss and depression drive both.

Q8. Geriatric Demography and Its Implications

SOURCE STATEMENT

This topic IS well covered in the uploaded textbooks. Pathy's addresses it in **Chapter 1 (biological perspective — survival curves), Chapter 2 (introduction to ageing) and Chapter 4 (Social and community aspects of ageing — "Demography of ageing")**; *Essentials of Clinical Geriatrics* covers it in **Chapter 2 (The Geriatric Patient: Demography, Epidemiology and Health Services Utilization)**.
Indian demographic data is NOT in either textbook and is taken from Census/NSO, LASI, the India Ageing Report 2023 and PIB releases.

A. Key Definitions and Indicators (learn these first)

1. **Population ageing** is the rise in both the **number** and the **proportion** of older persons. It has two components: **absolute ageing** (more people surviving to old age, driven by rising life expectancy) and **relative ageing** (a higher *proportion* of older adults, driven by falling fertility). Pathy's makes exactly this distinction — **"Increases in life expectancy around the world have led to absolute ageing of the global population"** while **"Declining fertility rates are also leading to a phenomenon of relative ageing."**
2. **The core demographic indicators you must be able to define:**

Indicator	Definition
Old-age dependency ratio	Pathy's: **"an economic index defined as the number of people age 65 or older per 100 people of working age (15–64 years old)"** (India uses 60+ over 15–59)
Ageing index	Number of persons aged 60+ per 100 children aged 0–14
Potential support ratio	Number of working-age persons per one older person (inverse of dependency ratio)
Sex ratio of the elderly	Females per 1,000 males aged 60+
Median age	Age dividing the population into two equal halves
Life expectancy at 60 (e_{60})	Average remaining years of life at age 60 — **more relevant to geriatrics than life expectancy at birth**
Healthy life expectancy (HALE)	Years expected to be lived in good health

B. The Global Picture (from Pathy's)

3. **WHO estimates that the population over age 60 will approximately double from 2015 to 2050, when 20% of the world's population will be over 60. The majority of these older adults will reside in low- and middle-income countries, providing global challenges to ensure they receive adequate access to healthcare and other resources.**
4. **The scale of the change in life expectancy: "In 1950, the average life expectancy was 49 years (68 in developed countries), as compared to 72 (79 in developed countries) in 2019."** The United Nations projects only **modest further increases**, and **"due to biologically and physiologically fixed mechanisms, it seems unlikely that, as a species, human life expectancy will ever exceed approximately 115 years of age."**
5. **The 80+ group is the critical one.** Pathy's states that **"at present, the over-85 population is the fastest-growing age group in developing countries."** This matters clinically because disability, multimorbidity, dementia and dependency all cluster in the very old.

C. Why Populations Are Ageing (Causes)

6. **Causes of absolute ageing: "Improved sanitation, nutrition, and living conditions, as well as major healthcare innovations such as effective treatments for infectious diseases, vaccinations, and advances in neonatal care."** Pathy's adds that **"management of chronic diseases, particularly cardiovascular and oncologic diseases, has also led to extended life expectancy in adulthood and older age."**
7. **Causes of relative ageing (falling fertility): "improved access to birth control, declining numbers of teenage pregnancies, increased female engagement in the workforce, a higher percentage of women pursuing advanced education, and delays in the average age of marriage."** The decline **"is more pronounced in developed countries, but the trend is seen globally."**
8. **Two great transitions run in parallel:** the **demographic transition** (high birth/high death → low birth/low death) and the **epidemiological transition** (communicable, maternal and perinatal diseases → **non-communicable, chronic and degenerative diseases**). The result in India is the **"double burden"** — infections persist while NCDs rise.

D. The Survival Curve — Compression of Morbidity (high-yield concept)

9. **Rectangularisation of the survival curve.** Pathy's Chapter 1 explains that **"medicine delays the death of most individuals of a given generation until they are closer to maximum lifespan, but it does not change the maximum lifespan much." Fries** interpreted this **"compression of mortality" as the result of a "compression of morbidity"** — based on the facts that there is a **maximum lifespan**, that **even without chronic disease individuals would still die**, and that **chronic diseases can be prevented**. Related is the concept of **healthspan — the duration of life in good health**.
10. **Compression vs expansion of morbidity — the central policy question.** If morbidity compresses, extra years are healthy years and the health-system burden is contained; if it expands, extra years are years of disability. Pathy's also notes the **"late-life mortality plateau"** — after 80 the incidence of death still increases but progressively more slowly — and reminds us that **Jeanne Calment died at 122**.

E. Feminisation of Ageing (guaranteed sub-question)

11. **Why women predominate in old age.** Pathy's: **"At birth, the male to female birth rate is approximately 1.05:1. However, females generally have a longer life expectancy than males, which leads to a shifting of the gender ratio in older adults, such that females outnumber males."** Reasons given: **oestrogen may be protective against cardiovascular disease; women are less susceptible to X-linked genetic disorders; males are more likely to engage in high-risk behaviour including dangerous occupations, reckless driving and substance use; political upheaval and wartime have disproportionately affected men** (Russia shows an almost **11-year** male–female gap); and **women generally engage with medical care and seek attention earlier than men**.
12. **The gap widens with age.** In the US in 2020, females outnumbered males **1.1:1 at age 65, 1.2:1 at 75, 1.5:1 at 85, and 2.5:1 at 95**. **The gender gap is expected to narrow** as cardiovascular mortality improves and as women take up workforce participation and smoking at higher rates.
13. **Important exception for India — quote this.** Pathy's states that in developing countries, **"due to less access to education and healthcare, higher rates of physical abuse, and risk of poverty in widowhood, women have lower life expectancies, resulting in female to male ratios closer to 1,"** and that **"India and China, which have historically practised female infanticide, have significantly more males than females, and these effects extend into older adulthood."** *(Note: current Indian data now show the elderly sex ratio has crossed 1,000 — see updates.)*

F. The Indian Picture (not in textbook — external sources)

14. **Headline projections:** India's 60+ population will rise from **149 million in 2022 (about 10%) to 347 million by 2050 (more than 20%)**, and **by mid-century the elderly will outnumber children**. The Government projects a rise from **100 million in 2011 to 230 million by 2036, forming 15% of the total population** — so **by 2036 nearly one in seven Indians will be aged 60 or older**. LASI shows the 60+ share moving from **6.9% (2000) → 11.4% (2025) → 19.5% (2050)**.
15. **The ageing of the aged and the widowhood problem:** the **80+ population will grow by about 279% between 2022 and 2050, with "a predominance of widowed and highly dependent very old women." More than half of India's women over 60 are widowed**, rising in higher age cohorts. The **elderly sex ratio is now 1,065 females per 1,000 males; 58% of the elderly are women, of whom 54% are widows.** In central India it rose from **973 (2011) to 1,053 (2021)**, and **poverty in old age is inherently gendered** — older women are more likely to be widowed, living alone, with no income and fewer assets.
16. **The North–South divide — a distinctly Indian feature: "By 2036, one in five people in India's southern states will be elderly. The north–south divide… is an important feature of India's ageing." Kerala will have the highest elderly share (23% by 2036), while Uttar Pradesh will see the fastest growth in elderly numbers.** Life expectancy at 60 is higher for women, with a **national differential of only 1.5 years but 4 years in Kerala and Himachal Pradesh**, where women at 60 can expect **22–23 more years**.
17. **Dependency and the demographic dividend:** India's **old-age dependency ratio is expected to reach 31.5 by 2050**, against a child dependency ratio of **29.8** per 100 persons aged 15–59. India's **median age is about 29 (2026)** — Kerala ~33, Bihar ~22 — with **TFR at 2.0 (NFHS-5), already below the replacement level of 2.1**, and the **demographic dividend expected to peak around 2036–2041 and close by 2055**.
18. **India's transition is COMPRESSED — the sentence that wins marks: "Developed countries underwent their demographic transitions gradually over several decades, allowing time to build robust social security systems, healthcare infrastructure, and pension schemes. In contrast, developing nations like India are experiencing compressed demographic transitions, with ageing occurring at unprecedented speed while economic development and social support systems struggle to keep pace." India is growing old before it grows rich.**

G. Implications (the second half of the question)

19. Implications, organised by domain:

Domain	Implication
Health service	Shift from acute/episodic to **chronic, multimorbid, long-term care**; rising demand for geriatric wards, rehabilitation, day care, palliative and home care; more emergency admissions and readmissions
Disease burden	Predominance of **NCDs, geriatric syndromes (falls, incontinence, delirium, immobility), dementia, sensory loss and multimorbidity**; polypharmacy
Economic	Pathy's: **"increased spending on healthcare, Social Security, disability, pensions, and infrastructure to support an ageing population, with fewer wage-earners contributing taxes"**; and **"a greater need for healthcare spending and long-term care employees"**
Social / family	Nuclearisation, migration of children, **fewer available caregivers**; *Essentials*: **"The family is the heart of long-term care… Informal care is largely provided by women"** — a double burden on working women
Gender	Feminisation of ageing means **more widows, more poverty, more dependency, more institutional need**
Workforce	Need for **geriatricians, geriatric nurses, physiotherapists, occupational therapists, medical social workers and trained care-givers** — India's largest deficit
Environment	Need for **age-friendly cities, barrier-free housing, accessible transport, assistive technology**
Ethical	Rationing pressure and **ageism**; *Essentials*: **"older adults are prime targets for rationing efforts because they consume disproportionately large amounts of medical care and because they are seen as having already lived their lives"**
Research & data	Need for **longitudinal cohorts (LASI), disaggregated 60+ data, and functional rather than disease-only outcomes**

20. **Three caveats that separate a good answer from an average one:**
    - **The dependency ratio is an imperfect measure.** Pathy's: **"It does not account for adults who work past the age of 65 or improvements in care and advances in technology that reduce the societal burden of ageing and disability. Although the old-age dependency ratio will certainly increase globally, the economic and societal implications remain uncertain."**
    - **Older people are not uniformly disabled.** *Essentials*: **"Because physicians tend to see the sick, they may form a distorted picture of senior citizens. Most older persons are indeed self-sufficient and able to function on their own or with minimal assistance."** In US data, ADL dependency rose from **23% at ages 65–84 to 48% at 85+**, and IADL dependency from **23% to 43%** — so **even at 85+, roughly half remain independent**.
    - **The elderly population is profoundly heterogeneous.** Pathy's: **"Each elderly individual is unique, and the elderly population is heterogeneous. A 65-year-old person may be immobile, while a 90-year-old may be mobile and completely independent in their activities of daily living." Chronological age is a poor guide; function and intrinsic capacity are better.**

LATEST UPDATES (2023–PRESENT)

1. India Ageing Report 2023 (UNFPA–IIPS) — now the standard reference. Projects the **80+ population growing by about 279% between 2022 and 2050 with a predominance of widowed and highly dependent very old women**, and shows **women have higher life expectancy at both 60 and 80, with wide state variation** (four years' advantage in Kerala and Himachal Pradesh vs a national average of 1.5 years). **States lagging in the transition, such as Bihar and Uttar Pradesh, will see the elderly share rise between 2021 and 2036 but remain below the national average**; **central and north-eastern regions are "younger" than southern and western India** on the ageing index. Also: **over 40% of the elderly in the poorest wealth quintile, 18.7% with no income, and 71% living in rural areas**.

2. Official government projection now widely quoted. MoSJE states India's elderly population is projected to **more than double from 100 million in 2011 to 230 million by 2036 — nearly one in seven Indians aged 60 or older by 2036, "a fundamental restructuring of the country's population composition"** — with **southern states plus Himachal Pradesh and Punjab already having higher elderly populations and regional disparities expected to widen**.

3. Feminisation is now measurable and reverses the older textbook position. The **elderly sex ratio is 1,065 females per 1,000 males**, with **58% of the elderly being women and 54% of them widows**, and the **overall dependency ratio at 62 dependents per 100 working-age individuals**. The elderly sex ratio **has been climbing steadily since 1991**, with central India moving from **973 (2011) to 1,053 (2021)**. **This directly updates Pathy's statement that India has "significantly more males than females… extending into older adulthood."**

4. Fertility below replacement and a closing dividend window. India's **TFR is now 2.0 (NFHS-5, 2019–21), below the replacement level of 2.1**, and its **median age is about 29 in 2026** (UN WPP 2024 and NSO), with **Kerala at ~33 and Bihar at ~22**. The **demographic dividend is estimated to peak around 2036–2041 and effectively close by 2055**.

5. Global context. The **UN Decade of Healthy Ageing (2021–2030)** frames the response under **combating ageism, age-friendly environments, integrated care and long-term care**, and WHO's **2023 progress report found fewer than one-third of countries have adequate resources**. **NITI Aayog (2024)** describes an **absence of a comprehensive, integrated senior-care policy in India**.

CURRENT PRACTICE UPDATE

Element	Textbook position	Current position (2023–2026)
Global 60+ share	Pathy's: will **roughly double from 2015 to 2050, reaching 20%**	Broadly confirmed by UN WPP 2024
India's sex ratio in old age	Pathy's states India has **significantly more males than females, extending into older adulthood**	**Reversed** — elderly sex ratio now **1,065 females per 1,000 males**, with clear **feminisation of ageing**
Indian projections	Not covered	**149 million (2022) → 347 million (2050)**; **elderly outnumber children by 2046**; **80+ up 279%**
Fertility	Pathy's notes global decline	**India's TFR is now 2.0 — below replacement**, so relative ageing will now accelerate
Old-age dependency ratio	65+ per 100 aged 15–64; Pathy's calls it **"an imperfect measure"**	India uses **60+ per 100 aged 15–59**; projected to reach **31.5 by 2050**
Regional pattern	Not covered	Marked **north–south divide**: Kerala 23% elderly by 2036; UP the largest absolute increase
Compression of morbidity	Fries' hypothesis presented as the framework	Still debated; Indian data (high disability, multimorbidity, low income) suggest **expansion**, not compression
Speed of transition	Not emphasised	**Compressed transition** now recognised as India's defining challenge

EXAMINER'S TIP — VERBATIM MARK-EARNING LINES

• *"Population ageing has two components: absolute ageing driven by rising life expectancy, and relative ageing driven by falling fertility."*
• *"Medicine delays death until closer to maximum lifespan but does not change maximum lifespan much — this rectangularisation of the survival curve is Fries' compression of morbidity."*
• *"India is experiencing a compressed demographic transition — ageing at unprecedented speed while social support systems struggle to keep pace. India is growing old before it grows rich."*
• *"The old-age dependency ratio is an imperfect measure: it ignores those working past 65 and technological gains that reduce the societal burden of disability."*
• *"Because physicians tend to see the sick, they may form a distorted picture of senior citizens — even at 85 and above, roughly half remain independent in ADLs."*
• *"A 65-year-old may be immobile while a 90-year-old is completely independent — chronological age is a poor guide; function and intrinsic capacity are better."*

EXTRA POINTS TO REMEMBER

• **Global:** LE 49 (1950) → 72 (2019); ceiling ~**115 years**; Jeanne Calment **122**; over-85s = **fastest-growing group**.
• **India:** 149 m → 347 m • 10.5% → 20.8% • 80+ **+279%** • elderly > children by **2046** • 230 m by **2036**.
• **Sex ratio 1,065** • 58% women • 54% widows • dependency ratio **62 per 100** • ODR **31.5 by 2050**.
• **TFR 2.0** (below replacement 2.1) • median age **~29** • dividend peaks **2036–2041**, closes **2055**.
• **US gender gap:** 1.1:1 at 65 → 2.5:1 at 95. **ADL dependency 23% (65–84) → 48% (85+).**
• **Kerala 23% elderly by 2036**; women at 60 in Kerala/HP expect **22–23 more years**.

Q9. Ethno-Geriatrics

SOURCE STATEMENT

Pathy's 6th edition covers this topic directly. The core chapter is **Chapter 8, "Health literacy and cultural sensitivity," by Nina Tumosa (HRSA, Rockville, MD), pages 108–118**. Additional material is in **Chapter 4 (migration, immigration, ethnic diversity, ageism)** and **Chapter 5 (LGBT+ older adults)**. Pathy's itself cites the **American Geriatrics Society Ethnogeriatrics Committee** and the **ETHNIC(S) mnemonic**, described in its source as **"a clinical tool for ethnogeriatric education."**
Contemporary (2023–2026) developments are drawn from peer-reviewed literature and WHO.

A. Definition and Scope

1. **Ethno-geriatrics** is the branch of geriatric medicine that studies **the influence of culture, race and ethnicity on the health and health care of older adults**, and applies that understanding to deliver **person-centred, equitable care**. It sits at the intersection of **geriatrics, cultural anthropology, health equity and health literacy**.
2. **Pathy's central thesis (learn this sentence): "Cultural differences between patient and provider can lead to misunderstandings, value conflicts, and disparate understandings of health and illness. Low health literacy leads to poor understanding and adherence and often limits access to health care. Together, low levels of health literacy and a lack of cultural sensitivity can severely impact health outcomes." Health literacy and cultural sensitivity are therefore the two pillars of ethnogeriatrics.**
3. **Everyone has a culture — including the doctor.** Pathy's: **"Both providers and patients have their own national and ethnic cultures… Health care providers have the culture of biomedicine and their specific profession and speciality."** Key exam point: **"Medicine is a culture that is not easily understood by, and often intimidates, older adults."**

B. Why It Matters — The Changing Demography of Diversity

4. **Populations become more diverse as they age.** Pathy's examples: **Bethnal Green in London was a predominantly white, working-class neighbourhood in the 1950s; by the 1990s, as Tower Hamlets, it was home to large numbers of Bangladeshi immigrants, and by 2018 it had the lowest life expectancy and the highest rate of heart disease of all London boroughs. Wolverhampton's population identifies as 55.5% Christian, 19.7% no religion, 9.1% Sikh, 3.7% Hindu, 3.6% Muslim and 0.4% Buddhist**; **London, Leicester, Luton, Slough and Birmingham now have a greater than 50% ethnic minority population**; and **over 800 languages are spoken in New York City**.
5. **Older migrants are a distinct vulnerable group.** In the US, **13.5% of the population over 65 is foreign-born, and about half report speaking English "less than very well." One in ten migrated as an older adult, most commonly sponsored by adult children**; such people **"are much more likely to have low incomes and be financially dependent on others"** and more likely to suffer **social isolation and loneliness**. Globally, **adults over 65 make up about 11.7% of the total migrant population**.
6. **The "healthy immigrant effect."** Pathy's: immigrants are on average **healthier than native-born residents**, explained by **self-selection, health screening requirements in receiving countries, and retention of healthier native habits** — but **health converges with the host population after several decades**. Disease patterns also shift: **immigrants from Yugoslavia and Hungary had a higher stroke incidence than Swedes in Malmö**, and **Japanese immigrants to Brazil have different cancer mortality rates from Japanese in Japan**.
7. **The clinical consequence: "older migrants are often non-adherent with medications, resulting from interactions between illness perceptions, low health literacy, language barriers, and disadvantaged socioeconomic circumstances, which can sometimes restrict the ability to purchase prescribed medications."**

C. Pillar 1 — Health Literacy

8. **Definition and burden. Health literacy is "individuals' ability to understand and manage their personal health care issues."** Pathy's key finding: **"Individuals with limited health literacy have less access to health knowledge, worse self-management skills, less frequent use of preventive services, higher hospitalisation rates, and more premature mortality than individuals with adequate health literacy."** Limited health literacy **"may be an independent risk factor for health disparities in older people."**
9. **Health literacy declines with age — independently. "Functional literacy declines with age."** In a study of 2,774 community-dwelling older adults, **a significant decrease in health literacy was seen with every year increase in age, even after adjusting for gender, race, ethnicity, cognitive status and education**. **Both health literacy and cognitive abilities independently predict mortality**, and **lower health literacy was associated with increased**

risk of death after hospitalisation for acute heart failure.

Factors affecting health literacy (Pathy's Table 8.1)	Consequences of low health literacy (Table 8.2)
Age — functional literacy declines with age	Poorer physical health
Education — ability to read, interpret, use technology	Poorer mental health
Cognitive status — recognising need for care, following instructions	Difficulty accessing health care
Income — insurance status, poverty, job insecurity, immigration status	Difficulty following provider instructions
Race/ethnicity — differing cultural beliefs, lack of proficiency in the local language, lower education	Difficulty taking medications properly
Health status — hearing loss, pain, disability, vision loss, poor physical functioning	**Increased hospitalisations; premature mortality**

10. **Practical remedies (Pathy's Table 8.3). Written material** should be **age-appropriate, use simple words, be at a sixth-grade reading level or lower, be in the patient's primary language, include pictures and illustrations, and present only a small amount of information**. **Auditory material** should be **delivered slowly, one-to-one, by a trusted provider, in the patient's primary language**. Note the mismatch: **many US older adults read at an eighth-grade level and 20% at or below fifth-grade, yet most healthcare materials are written at a tenth-grade level.**
11. **Two traps to name in the exam.** First, **"the greatest potential barrier to addressing health literacy is that most patients are often unwilling to admit that they have literacy problems."** Second, **97% of subjects, regardless of literacy level, reported that television was their primary source of health information** — so mass and folk media matter more than leaflets.

D. Pillar 2 — Cultural Sensitivity

12. **Culture is fluid, not fixed.** Pathy's stresses that **"culture is fluid and constantly changing,"** that there are **cultural differences between different generations who share the same cultural background**, and that **gender differences across cultures can be even more complicated**. Since geriatric care **"often requires that health care providers interact with multiple generations of the same family,"** any strategy **"needs to be general and flexible in approach."** Cultural factors affecting care (**Table 8.4**): **age, gender, ethnicity, assimilation, generation cohort, and the ever-changing nature of culture**.
13. **The four barriers to culturally appropriate care (Table 8.5): the large variety of cultures**; **the effects of acculturation**; **the negative effects of stereotypes**; and **the complexity of the culture of biomedicine itself**. Pathy's conclusion: **"Cultural sensitivity is more a process than an outcome. It is as much about acceptance of differences as it is about knowledge of differences."**
14. **The five common areas of conflict (Table 8.6) — a guaranteed sub-question:**

Area of conflict	Explanation from Pathy's
Mistrust from prior contacts	Older adults may distrust providers based on previous encounters with biomedicine, **"such as the American experience with the Tuskegee Syphilis Study"**
Who is "the patient"?	Western biomedicine focuses on the **adult individual**; in many cultures **"the family, or even the entire community, plays a major part in managing illness,"** so **it is often best to include the entire family in decision-making, including spouses and adult children**
Verbal and non-verbal communication	Language barriers cause **"misdiagnosis to ineffective pain control."** Even in a shared language, meaning differs — a patient saying **"stomach ache"** may mean **anywhere in the abdomen**
Misconceptions about cultural norms (stereotyping)	**"Sometimes a little knowledge is a dangerous thing."** Instead of memorising every culture, **a brief conversation with the patient or interpreter** clarifies what is acceptable. **"There is no substitute for asking the older adult what their treatment goals are."**
Ageism by BOTH patient and provider	Younger providers may **exaggerate the impact of functional differences and chronic illness and fail to recommend smoking cessation, good nutrition and exercise**; conversely older adults may disregard advice from much younger clinicians — **"reverse stereotyping"**

15. **Non-verbal communication carries enormous weight.** Pathy's lists culturally variable elements: **expression of distress (stoicism versus open expression of pain), the pace of conversation, whether interruptions are encouraged, physical proximity during history-taking, whether eye contact is appropriate or disrespectful, the etiquette of touch, hand gestures and finger-pointing, and attitudes toward the direct discussion of death and dying**. A modern hazard: **"One unintended consequence of the introduction of the electronic record is the perception on the part of some patients that it is disrespectful of the clinician to interact with the computer screen more than with the patient."**
16. **Where biomedicine itself clashes with older patients.** Pathy's is unusually blunt: **"the culture of medicine often emphasises the quantity of life over the quality of life. However, older patients may insist on more autonomy**

than the culture of biomedicine encourages." It adds that **"it is not uncommon for family members to decide that an older patient should not be treated for a serious disease such as depression or cancer"** — directly relevant to Indian practice.

E. Strategies — How to Practise Ethnogeriatrics

17. **Start with self-awareness.** Pathy's: **"Everyone has a culture… Approaching the older adult as a cultural equal will reduce conflict between cultures. A sense of humour about small misunderstandings is crucial… None of the cultures involved are problems to be overcome. Rather, they are opportunities for sharing."** Crucially, **"educational level, dominant language, religion, gender, year of immigration, and even personality may have more of an effect on interactions with health care providers than cultural identity. The best way to find out… is to ask them directly and listen carefully."**
18. **The ETHNIC(S) mnemonic (Pathy's Table 8.7) — memorise this; the single most examinable item in the chapter.** It **"can serve as a clinically applicable tool for eliciting and negotiating cultural issues during health care encounters."**

Letter	Meaning	What to actually do
E	**Explanation**	Elicit the patient's own explanatory model — what they think the symptoms mean and what family, television or the internet has suggested
T	**Treatment**	Ask what has already been tried — **specifically ask about complementary and alternative medicine and home remedies**; "sometimes those remedies are helpful, and sometimes they are harmful"
H	**Healers**	Acknowledge you are not the only healer; identify **other healers who must be included in the treatment plan**
N	**Negotiate**	Recovery is teamwork — establish **how much of a partner the older adult intends to be in their own recovery**; engage informal caregivers
I	**Intervention**	Determine willingness to participate; modify the plan for cultural conditions in a timely manner
C	**Collaborate**	Family, other healers, the interdisciplinary geriatric team and community services — **"Continuum of care for older people is very much a team endeavour"**
S	**Spirituality / Seniors**	**"Spirituality is an often-overlooked aspect of health care"** — the provider should **take the lead in asking** whether the patient wishes to discuss the role of their beliefs; **"particularly important for end-of-life care"**

19. **Use professional interpreters, not children.** Pathy's: **"It is usually advisable to engage a professional translator or use translated written materials."** Child interpreters are **"especially problematic, especially if the medical topic is not appropriate for a child"**; a sensitive child **"may wish to protect the older adult's dignity, not understanding that the provider–patient relationship relies strongly on honesty,"** and an older adult **"may not be comfortable communicating through a child of the opposite gender, thus leading to more confusion and under-reporting of symptoms."**
20. **Culturally tailor health promotion, and remember other minority identities.** Pathy's notes that **cultural differences in the perception of what defines a good diet or the proper amount or form of exercise "may be difficult to address but well worth the effort."** It also reminds us that **about 0.5% of US adults over 65 identify as transgender**, that **transgender older adults face the cumulative effects of a lifetime of discrimination with higher rates of anxiety, depression and loneliness and less robust support networks**, and that there is **"a dearth of cultural competency training for providers of older adults around issues of gender and sexual identity."**
21. **The pay-off — and a silent trap.** Cultural sensitivity **"can lead to better provider–patient communication, more accurate diagnoses, more effective treatment, higher patient satisfaction/compliance, and efficient use of medical resources."** But Pathy's warns: **older, non-white and literacy-deficient patients report GREATER communication satisfaction than younger, white, functionally literate peers, and "are more likely to cope with their illnesses by withdrawing rather than by actively pushing for a higher standard of care"** — so **apparent satisfaction may mask disengagement**, and clinicians **"should continuously seek ways to facilitate dialogue with patients who are older and non-white and have poor literacy skills."**

F. Application to India (discussion point — not in the textbook)

22. **India is arguably the world's largest ethnogeriatric laboratory.** Relevant axes of diversity: **22 scheduled**

languages and hundreds of mother tongues; multiple religions; scheduled castes and scheduled tribes; large-scale rural-to-urban and inter-state migration; and regional variation in the pace of ageing. Indian issues that map directly onto Pathy's framework: **family-centred rather than individual decision-making is the norm; non-disclosure of a cancer diagnosis to the patient is common; widespread concurrent use of AYUSH and folk remedies (the "T" and "H" of ETHNIC(S)); low functional literacy in older rural women; and 71% of India's elderly live in rural areas with over 40% in the poorest wealth quintile. Ethnogeriatrics in India is not an imported Western concern — it is everyday practice.**

LATEST UPDATES (2023–PRESENT)

1. Ethnogeriatrics is now framed explicitly as a health-equity discipline. Current reference texts define it as **"the influence of culture, race, and ethnicity on health care of older adults,"** noting **"an increasing recognition of the importance of cultural issues in health equity"** and that **"recognizing this importance and approaches from a health systems and clinical practice standpoint is critical in delivering person-centered equitable care."** The demographic driver is stark: **the non-Hispanic White share of the US older adult population is falling sharply, and non-Hispanic Whites will no longer comprise 50% of the general population by 2044**.

2. Hard evidence of disparities at the end of life in dementia (2024). A scoping review in *Alzheimer's & Dementia* found **"less optimal end-of-life care for minoritized populations, including more aggressive life-sustaining interventions, higher hospitalization rates, and lower use of hospice and advance directives,"** attributed to **"the cultural competence of staff, socioeconomic inequality, and systemic racism, as well as individual-level preferences and values."** A major research gap was identified: **fewer than one-third of studies reported findings separately for Asian American/Pacific Islander populations and under 3% for American Indian/Alaska Native populations**.

3. Conceptual shift: from *cultural competence* to *structural competency*. A 2025 *Annual Review of Clinical Psychology* analysis states that **"individuals from minoritized racial/ethnic groups face a disproportionate burden of Alzheimer's disease and related dementias,"** and that **"this health inequality reflects structural racism, which creates and sustains racial differences in social determinants."** **Exam framing: cultural sensitivity fixes the consultation; structural competency is needed to fix the disparity.**

4. Cultural tailoring is now being tested in trials, not just recommended. A 2024 Gerontological Society of America symposium reported **culturally tailored and community-engaged interventions** — including **"GetActive," a culturally adapted chronic pain programme delivered in Spanish and English which improved pain management and quality of life**, a **peer-support programme for minority and low-income older adults with depression**, and work on **socio-ecological barriers to equitable participation in dementia prevention and clinical trials** — concluding that **"community engagement and cultural tailoring"** function as protective factors for health equity. Parallel 2025 work on **Indigenous older adults' cognitive health** concluded that **"building a society that resolves structural inequities could eliminate disparities in later-life cognitive health for all older adults."**

5. Ageism as a global, measurable target. The **WHO Global Campaign to Combat Ageism**, embedded in the **UN Decade of Healthy Ageing (2021–2030)**, makes **combating ageism one of four action areas**. WHO's 2023 progress report noted a **more than 20% rise in countries reporting legislation against ageism**, but **fewer than one-third of countries have adequate resources**. WHO's **"Health of older refugees and migrants" (2018)** — cited by Pathy's itself — remains the standing technical guidance for migrant elders.

CURRENT PRACTICE UPDATE

Element	Pathy's 6th edition (2022)	Current position (2023–2026)
Core framing	**Cultural sensitivity + health literacy** at the level of the clinical encounter	Reframed as **health equity**, with **structural competency and structural racism** added as upstream determinants
Clinical tool	**ETHNIC(S) mnemonic** (Kobylarz et al., 2002)	Still the standard teaching tool; complemented by **Kleinman's explanatory model** and community-engaged, culturally tailored programmes
Evidence base	Largely descriptive and observational	**Interventional** — culturally adapted pain, depression and dementia-prevention programmes now being trialled
Dementia care	Not addressed as a disparity	**Documented disparities in end-of-life dementia care** — more aggressive interventions, more hospitalisation, less hospice and fewer advance directives
Under-studied groups	Not flagged	**Asian American/Pacific Islander, Indigenous and Alaska Native older adults remain markedly under-represented**
Ageism	A bilateral clinical phenomenon	Now a **formal WHO action area** with legislation tracked internationally
India	Not covered	**No Indian ethnogeriatric framework, curriculum or data set exists** — a genuine research and policy gap

EXAMINER'S TIP — VERBATIM MARK-EARNING LINES

• *"Ethnogeriatrics is the influence of culture, race and ethnicity on the health care of older adults; its two pillars are health literacy and cultural sensitivity."*
• *"Medicine is a culture that is not easily understood by, and often intimidates, older adults."*
• *"Cultural sensitivity is more a process than an outcome — as much about acceptance of differences as knowledge of differences."*
• *"I would use the ETHNIC(S) mnemonic: Explanation, Treatment, Healers, Negotiate, Intervention, Collaborate, Spirituality/Seniors."*
• *"There is no substitute for asking the older adult what their treatment goals are — sometimes a little knowledge is a dangerous thing and leads to stereotyping."*
• *"Older, non-white, literacy-deficient patients report greater satisfaction yet cope by withdrawing rather than pushing for a higher standard of care — apparent satisfaction may mask disengagement."*

EXTRA POINTS TO REMEMBER

• **ETHNIC(S)** — the one mnemonic to write down first in this answer.
• **Four barriers:** variety of cultures • acculturation • stereotypes • complexity of biomedicine.
• **Five areas of conflict:** mistrust • who is the patient • communication • stereotyping • ageism (bilateral).
• **Written material at sixth-grade level**; most materials are at tenth-grade; 20% of US older adults read at fifth grade or below.
• **97% cite television as their primary information source** — justifies mass and folk media (as NPOP and NPHCE mandate).
• **Never use a child as interpreter**; use a professional translator.
• **Migrants:** 13.5% of US 65+ foreign-born; ~half speak English "less than very well"; 11.7% of global migrants are 65+; **healthy immigrant effect** converges over decades.

APPENDIX — RAPID REVISION GRID

Everything below is verifiable in the answers above. Read this the night before the examination.

1. Chronology — Indian Policy, Law and Programmes

Year	Milestone	What to remember
1995	National Social Assistance Programme (NSAP)	Ministry of Rural Development; IGNOAPS, IGNWPS, IGNDPS, NFBS, Annapurna
Jan 1999	**National Policy on Older Persons (NPOP)**	MoSJE; UN International Year of Older Persons; **6 areas of intervention**; NCOP constituted May 1999 (39 members)
2007	**MWPSC Act**	Tribunals; **₹10,000** cap; **90 days**; abandonment punishable; property transfer void; **Chapter IV = medical care**
2008	UNCRPD in force for India (3 May)	**Article 25** — services close to people's own communities including rural areas
2010	**NPHCE launched**	11th Five-Year Plan; **₹288 crore** for 2010–12; **80:20** Centre:State
2011	NPSrC drafted	**NEVER NOTIFIED — still a draft**
2012	NCOP → **National Council of Senior Citizens (NCSrC)**	Broader mandate; State-level councils created
2016–17	Tertiary NPHCE renamed **RVJSY**	Rashtriya Varisth Jan Swasthya Yojana
2019	MWPSC (Amendment) Bill	Removes ₹10,000 cap; priority for 80+; **STILL PENDING**
Feb 2024	**NITI Aayog: Senior Care Reforms in India**	**4 core areas:** Health, Social, Economic/Financial, Digital
29 Oct 2024	**Ayushman Vay Vandana Card**	**₹5 lakh, all 70+, income-independent**; ~1,961 procedures, 27 specialties; 9.67 m cards by 31 Dec 2025
1 Apr 2025	**Unified Pension Scheme (UPS)**	50% of last 12 months' average basic pay; min **₹10,000/month**; family pension 60%
21 Nov 2025	**Four Labour Codes in force**	29 laws rationalised; Code on Social Security merges **9 Acts**; gig workers covered (Sec 113–114)
2026	**EPS 2026** replaces EPS-95	Min pension still **₹1,000**; **20-day settlement, 12% penal interest**

2. Global Chronology — WHO and the UN

Year	Milestone
1952	**ILO Convention 102** — nine branches of social security
1991	UN Principles for Older Persons
2001	UK **National Service Framework for Older People** — **Standard One: rooting out age discrimination**
2015	WHO **World Report on Ageing and Health** — introduces healthy ageing, intrinsic capacity, functional ability
2016	Global Strategy and Action Plan on Ageing and Health
2017	**ICOPE Guidelines** — **13 recommendations**, 3 modules, 6 priority actions
2019	ICOPE Handbook 1st ed. — **5-step pathway** + Implementation Framework
2021–2030	**UN Decade of Healthy Ageing** — 4 action areas
2023	Decade **first progress report** (136 countries) — <1/3 have adequate resources
29 Jan 2025	**ICOPE Handbook 2nd ed.** — **4-step pathway** + new urinary incontinence pathway

3. Numbers You Must Not Get Wrong

Item	Figure
NPHCE beds — District Hospital	**10** (including **2 respite** in a separate room)
NPHCE beds — Regional Geriatric Centre	**30** = 15 M + 15 F; each side **3 acute + 7 sub-acute + 5 long-term**
NPHCE contractual staff	DH **13**; RGC **38**; CHC **1**; Sub-centre **0**
NPHCE funding — DH	**₹87 lakh** non-recurring / **₹44 lakh** recurring p.a.
NPHCE funding — RGC	**₹370 lakh** non-recurring / **₹163.44 lakh** recurring p.a.
Original RGCs / current RGCs / NCAs	**8 → 17–19**; **2** National Centres for Ageing (AIIMS Delhi, MMC Chennai)
Districts covered by NPHCE	**~713**
IGNOAPS central pension	**₹200/month (60–79)**; **₹500/month (80+)**
EPS minimum pension	**₹1,000/month** since 1 Sept 2014 (demand: ₹7,500); wage ceiling ₹15,000
SCSS	**8.2% p.a. quarterly**; max **₹30 lakh**; 5 + 3 years; 80C; TDS above ₹1 lakh
APY	Age **18–40**; pension **₹1,000–₹5,000**; **9 crore** enrolments (Apr 2026)
RVY income ceiling	**₹15,000/month** or BPL; devices by **ALIMCO**
Elderline	**14567**
Vayoshreshtha Samman	**1 October**; **13 categories**, **15 awardees**; ₹2.5 lakh / ₹5 lakh
OOPE (National Health Accounts)	**64.2% (2013-14) → 39.4% (2021-22)**; GHE **1.13% → 1.84% of GDP**
NHP 2017 targets	GHE **2.5% of GDP**; OOPE **35%** — **both missed**
ILO social protection coverage (India)	**24.4% (2021) → 48.8% (2024)**; ~920 million / 65% including in-kind; global **52.4%**
India 60+ population	**149 m (2022) → 347 m (2050)**; **10.5% → 20.8%**; 230 m by 2036
80+ growth	**+279%** (2022–2050)
Elderly outnumber children	**2046**
Elderly sex ratio	**1,065** F per 1,000 M; **58%** women; **54%** of them widows
Old-age dependency ratio (India)	**31.5 by 2050**; overall dependency **62 per 100**
TFR / median age	**2.0** (NFHS-5, below replacement 2.1) / **~29 years**
Poverty among elderly	**>40%** in poorest quintile; **18.7%** no income; **71%** rural
LASI Wave-1	**72,250** adults 45+, including **31,464** aged 60+; 30 states, 6 UTs
Mobility impairment (WHO)	**39%** of over-65s — 3× the working-age rate
HELP delirium trial	**15% → 9.9%**; OR **0.60**, p = 0.02
Stroke unit meta-analysis	**29 trials, 6,536 patients**; OR mortality **0.86**, death/dependence **0.78**, death/institutionalisation **0.80**
Incident reporting sensitivity	Captures only **7–15%** of adverse events
ADL dependency (US)	**23%** at 65–84 → **48%** at 85+ (so ~half remain independent at 85+)
Life expectancy (global)	**49 (1950) → 72 (2019)**; species ceiling ~**115**; Jeanne Calment **122**

4. Mnemonics and Structured Lists

List	Content
ETHNIC(S) — ethnogeriatrics	**E**xplanation • **T**reatment • **H**ealers • **N**egotiate • **I**ntervention • **C**ollaborate • **S**pirituality/Seniors
Six CGA elements	Functional ability • cognition • physical health • socioeconomic status • nutrition • mobility/falls risk
Six ICOPE domains	Cognition • mobility • vitality • vision • hearing • psychological capacity
ICOPE 4 steps (2025)	Basic assessment + community interventions • in-depth assessment • personalised care plan • implementation and monitoring
ICOPE 3 modules	IC declines • geriatric syndromes (incontinence, falls) • caregiver support
HELP six risk factors	Cognitive impairment • visual impairment • hearing impairment • sleep deprivation • immobility • dehydration
UN Decade — 4 action areas	Ageism • age-friendly environments • integrated care • long-term care
NPOP — 6 areas of intervention	Financial security • health care and nutrition • shelter • education • welfare • protection of life and property
NPOP — 6 guiding principles	Affirmative action • life-cycle continuum • age-integrated society • older persons as a resource • empowerment • partnership
NPHCE structure	Vision **5** • objectives **4** • expected outcomes **7** • components **8** • main strategies **5**
Six roles of government	Steward • legislator • financier • provider • regulator • capacity-builder/knowledge-generator
World Bank 5 pillars	0 assistance • 1 insurance • 2 defined contribution • 3 voluntary • 4 informal (family)
AVYAY components	IPSrC • SAPSrC • RVY • SCOPE/SACRED • SAGE
NSAP components	IGNOAPS • IGNWPS • IGNDPS • NFBS • Annapurna
Ethnogeriatrics — 4 barriers	Variety of cultures • acculturation • stereotypes • complexity of biomedicine
Ethnogeriatrics — 5 conflicts	Mistrust • who is the patient • communication • stereotyping • ageism (bilateral)
Patient safety — 5 dimensions	Past harm • reliability • sensitivity to operations • anticipation and preparedness • integration and learning

5. Quotable Lines from the Uploaded Textbooks

Source	Line
Pathy's Ch. 11	NSF Standard One is **"rooting out age discrimination"**; the geriatrician balances **therapeutic nihilism against therapeutic heroism**
Pathy's Ch. 8	**"Medicine is a culture that is not easily understood by, and often intimidates, older adults."**
Pathy's Ch. 8	**"Cultural sensitivity is more a process than an outcome."**
Pathy's Ch. 4	**"It seems unlikely that, as a species, human life expectancy will ever exceed approximately 115 years of age."**
Pathy's Ch. 4	The old-age dependency ratio **"does not account for adults who work past the age of 65 or improvements in care and advances in technology."**
Pathy's Ch. 1	**"Medicine delays the death of most individuals... but it does not change the maximum lifespan much"** — Fries' compression of morbidity
Pathy's Ch. 100	On ICOPE: **"the impact of this program is not known at this time"**
Essentials Ch. 15	**"Assessment without follow-up is unlikely to make any difference."**
Essentials Ch. 15	**"Good team care will not arise spontaneously."**
Essentials Ch. 20	**"Older adults are prime targets for rationing efforts... seen as having already lived their lives"**; **"by such logic, disability is equivalent to death"**
Essentials Ch. 2	**"Because physicians tend to see the sick, they may form a distorted picture of senior citizens."**
NPOP 1999	Goal: to live the last phase of life with **"purpose, dignity and peace"**; older women face **"triple neglect and discrimination on account of gender, widowhood and age"**
NPHCE 2010	Vision: a new **"architecture" for Ageing** and **"a Society for all Ages"**

6. The Six Standing Gaps in Indian Elder Policy (write these in any policy answer)

1. **NPSrC 2011 never notified** — India runs on a 1999 policy.
2. **MWPSC Amendment Bill 2019 still pending** — the ₹10,000 cap stands.
3. **No statutory long-term care financing**, no caregiver allowance, no LTC insurance.
4. **Unindexed benefits** — IGNOAPS ₹200/₹500 and EPS ₹1,000 unrevised for over a decade.
5. **Fragmentation across five ministries** with no single accountable authority.
6. **Outpatient care and medicines — two-thirds of OOPE — remain outside PM-JAY**; ~400 million in the "missing

middle."

THE FIVE SENTENCES THAT CARRY THIS ENTIRE BLOCK

1. *"NPOP 1999 is the parent policy; MWPSC 2007 gave it legal teeth, NPHCE 2010 gave it a delivery system, and Ayushman Vay Vandana 2024 gave it money."*

2. *"India is growing old before it grows rich — a compressed demographic transition against an incomplete social security system."*

3. *"Functional ability equals intrinsic capacity plus environment plus their interaction — that equation is the whole of ICOPE."*

4. *"Out-of-pocket expenditure has fallen from 64.2% to 39.4%, so public spending now exceeds private — but both National Health Policy 2017 targets were missed."*

5. *"Pathy's supplies the concepts, ethics and global evidence; Government of India documents supply the programmes, law and money. The complete answer explicitly joins the two."*

END OF COMPENDIUM • 9 Questions • Health Policy, National Programmes & Social Gerontology
Prepared for MD Geriatric Medicine examination preparation • August 2026

MD Geriatric Medicine — Neurology Answer Bank

Twenty-two structured 15-mark answers • Primary source: Pathy's Principles and Practice of Geriatric Medicine, 6th ed. (2022) and Essentials of Clinical Geriatrics, 7th ed.
Latest Updates sourced from peer-reviewed journals and society guidelines (2023 – August 2026)

Contents

1. Alzheimer's dementia — pathogenesis, molecular advances, drugs, newer modalities
2. Advances in diagnosis of early Alzheimer's dementia and MCI
3. Newer / current drug therapy for Alzheimer's disease
4. Biomarkers for Alzheimer's dementia
5. Emerging biomarkers in cognition
6. Surgical management of parkinsonism
7. Recent advances in the management of parkinsonism
8. Management of motor fluctuations in Parkinson's disease
9. Parkinsonism-plus syndromes
10. Pathognomonic MRI findings in PSP and MSA
11. Confusion states in the elderly — investigation and management
12. Interventions to prevent delirium in post-operative older patients
13. Thrombolysis in stroke
14. History of the 'Stroke Clinics'
15. Restless legs syndrome
16. Myasthenia gravis
17. Motor neuron disease
18. Dizziness in the elderly
19. Ring-enhancing shadow on CT brain
20. Causes of coma in the elderly
21. Normal pressure hydrocephalus
22. Stroke rehabilitation

How to use: Points 1–20 in each answer are textbook-derived and are what earn marks in the written paper. The *Latest Updates* and *Current Practice Update* sections are what earn marks in the viva. Where a topic is not covered in the uploaded textbooks, this is stated explicitly and the content is drawn from current peer-reviewed literature.

1. Alzheimer's Dementia — Pathogenesis, Molecular Advances, Drugs and Newer Modalities

Fifteen–Twenty High-Yield Points

Definition and epidemiology

1. **Alzheimer's disease (AD)** is a progressive neurodegenerative disorder causing sustained loss of memory and other intellectual functions severe enough to impair daily living. Loss of function separates dementia from MCI.
2. About **6–8% of people over 65** have AD. Prevalence **doubles every 5 years**, so nearly **30% of those over 85** are affected. Family history and increasing age are the primary risk factors.
3. Mixed pathology is the rule in old age. AD change plus vascular disease lowers the threshold at which cognitive decline becomes clinically apparent.

Genetics

4. Early-onset familial AD is caused by mutations in three genes: **APP (chromosome 21)**, **presenilin 1 (chromosome 14)** — the largest group — and **presenilin 2 (chromosome 1)**.
5. The strongest genetic risk factor for late-onset AD is the **apolipoprotein E ε4 allele on chromosome 19**. One or more copies carries a relative risk of about **2.5** in whites, but it does not raise risk equally in African-American or Hispanic populations.
6. ApoE ε4 is **neither sensitive nor specific**, so it is **not recommended as a screening test**, even in high-risk groups.

Pathogenesis — the molecular sequence

7. The two histological hallmarks are **neuritic (senile) plaques** — extracellular masses with an amyloid core surrounded by degenerating neurites — and **neurofibrillary tangles** of abnormal **hyperphosphorylated tau** inside neuronal cell bodies.
8. **Amyloid cascade:** APP is cleaved by **β-secretase (BACE1)** and **γ-secretase (presenilin)** to form Aβ1-42, which oligomerises into **soluble protofibrils** (the most synaptotoxic species) and then fibrillar plaques.
9. **Tangle spread follows Braak staging** — entorhinal cortex first, then hippocampus and association cortex. **Tangle burden correlates with cognitive decline**; plaque burden correlates poorly and may be present in normal cognition.
10. Additional interacting mechanisms of brain ageing accelerate AD: **mitochondrial dysfunction, oxidative damage, loss of proteostasis (impaired autophagy and proteasome), dysregulated neuronal calcium homeostasis, impaired adaptive stress responses (reduced BDNF/NGF signalling), glial activation and neuroinflammation, impaired DNA repair, stem-cell exhaustion, and aberrant network activity with loss of synaptic plasticity.**
11. **Cerebral amyloid angiopathy (CAA)** — amyloid deposition in leptomeningeal and cortical vessel walls — is a recognised neuropathological hallmark of AD and causes lobar haemorrhage, microbleeds and white matter lesions.
12. Biomarker studies show Aβ and tau changes begin **years to decades before symptoms**. Cognitively normal older people with low CSF Aβ1-42 lose whole-brain and hippocampal volume faster, suggesting a **presymptomatic AD phase**.

Drug therapy — textbook position

13. **Cholinesterase inhibitors** are the primary pharmacological approach. Four have been approved: **tacrine, donepezil, rivastigmine and galantamine**. Tacrine is **hepatotoxic** and no longer prescribed.
14. **Mechanism:** they inhibit acetylcholinesterase and raise synaptic acetylcholine, partly correcting the cholinergic deficit from basal forebrain neuronal loss. Rivastigmine additionally inhibits butyrylcholinesterase.
15. **Efficacy:** randomised placebo-controlled trials show small improvements in cognition, possible prevention of functional decline, a several-month delay in progression, and possible delay of nursing home admission. **The clinical importance is marginal in many patients.** There is some efficacy in **multi-infarct dementia and dementia with Lewy bodies**.
16. **Side effects:** nausea, vomiting, diarrhoea, anorexia and weight loss; bradycardia and syncope; vivid dreams and nightmares; urinary urgency; muscle cramps. Caution with bradyarrhythmias and peptic ulcer disease.
17. **Memantine** is an **NMDA receptor antagonist** used in moderate-to-severe disease; it reduces glutamate-mediated excitotoxicity and is generally well tolerated (dizziness, headache, confusion).
18. **Behavioural symptoms:** non-pharmacological strategies come first. **Antipsychotics carry a black-box warning for increased mortality** and should be reserved for patients who are psychotic or an immediate danger to self or others. Every patient with new or worsening behaviour needs a medical evaluation for a treatable precipitant — **pain is especially hard to detect**, and empirical paracetamol has shown efficacy in severe dementia.

Non-pharmacological and newer modalities

19. Four categories of non-pharmacological intervention are described: **restorative cognitive training**, **compensatory cognitive training**, **lifestyle interventions** and **psychotherapeutic interventions**. These remain necessary even when drugs are used.
20. **Exercise** has an emerging role in preventing and treating cognitive decline; **high-intensity resistance training** is the most promising form, particularly in MCI. Attention to caregiver health and advance care planning are integral to management.

Latest Updates (2023 – Present)

- **Anti-amyloid monoclonal antibodies are now standard disease-modifying therapy in early AD. Lecanemab**

(Leqembi), an IgG1 antibody against Aβ protofibrils, received FDA approval in July 2023 on the basis of **CLARITY-AD**, which showed a **27% slowing of decline on CDR-SB over 18 months**. **Donanemab (Kisunla)**, which targets the pyroglutamate-modified plaque epitope, was approved in July 2024 on the basis of **TRAILBLAZER-ALZ 2** (~29% slowing) (*NEJM* 2023; *JAMA* 2023).

- **Amyloid-related imaging abnormalities (ARIA)** are the defining class toxicity — **ARIA-E** (vasogenic oedema) and **ARIA-H** (microhaemorrhage, superficial siderosis). Risk is highest in **APOE ε4 homozygotes**. **TRAILBLAZER-ALZ 6** led to an FDA label change in July 2025 with a **gradual titration schedule that cut ARIA-E from 24% to about 14% at 24 weeks** while preserving amyloid and p-tau217 reduction.
- **Delivery has been simplified.** FDA approved **four-weekly IV maintenance dosing** of lecanemab in January 2025, **subcutaneous maintenance (Leqembi IQLIK)** in August 2025, and a **subcutaneous starter dose in July 2026** — making lecanemab the first anti-amyloid agent deliverable subcutaneously through the full course.
- A **Cochrane review (April 2026)** pooling 17 trials and 20,342 participants across the anti-amyloid antibody class confirmed statistically significant but **small absolute benefit**; relative figures such as 27–29% should always be quoted alongside the underlying CDR-SB difference.
- **Beyond amyloid:** nearly 200 trials of >150 agents are running, targeting tau (antisense oligonucleotides, anti-tau antibodies), neuroinflammation, synaptic protection and metabolic pathways. **Trontinemab** (brain-shuttle antibody) and combination anti-amyloid/anti-tau strategies (**Tau NexGen**) are in advanced development. Prevention trials in asymptomatic amyloid-positive people (**AHEAD 3-45**, **TRAILBLAZER-ALZ 3**) are ongoing.
- **Multidomain lifestyle prevention:** the **US POINTER trial** (2,111 participants, reported 2025) compared a structured two-year multidomain programme with a self-guided programme in older adults at risk, extending the FINGER model.
- **Diagnosis is now biological.** The **Alzheimer's Association Revised Criteria (Jack et al., *Alzheimer's & Dementia* 2024)** define AD by biomarkers rather than syndrome. An abnormal **Core 1** biomarker (amyloid PET, approved CSF markers, or accurate plasma **p-tau217**) is sufficient to diagnose AD anywhere on the continuum.

Current Practice Update

Aspect	Textbook position	Current evidence / practice
Disease-modifying therapy	None available; treatment is symptomatic only	Lecanemab and donanemab are approved and modestly slow decline in early AD
Basis of diagnosis	Clinical/syndromic (DSM criteria), imaging to exclude other causes	Biological, biomarker-defined (2024 Revised Criteria); a single abnormal Core 1 marker is diagnostic
Cholinesterase inhibitors	Mainstay of therapy; benefit marginal but the only option	Still first-line symptomatic therapy, now used *alongside* disease-modifying antibodies in eligible patients
APOE genotyping	Not recommended, even in high-risk groups	Increasingly performed before anti-amyloid therapy because ε4 homozygosity predicts ARIA risk
Amyloid detection	PET and CSF only, research tools	FDA-cleared plasma p-tau217/Aβ42 ratio (Lumipulse G, May 2025) available in routine specialist care

2. Advances in the Diagnosis of Early Alzheimer's Dementia and Mild Cognitive Impairment

Fifteen–Twenty High-Yield Points

Concept and definitions

1. **Mild cognitive impairment (MCI)** and **cognitive impairment, not dementia (CIND)** describe measurable cognitive deficits that do **not** impair daily function. **Loss of functional ability due to impaired cognition is the single feature that separates dementia from MCI.**
2. Just over **20% of people aged 71 and above** have cognitive impairment without dementia. **15–20% of those with MCI or CIND convert to dementia each year.**
3. Normal ageing may produce slowed thinking and reaction time, mild recent memory loss and impaired executive function, which may or may not progress. **Old age alone never causes dysfunctional cognitive impairment.**

The three diagnostic questions

4. Three questions establish the cause of confusion: (i) **Has the onset been acute** (hours to days)? (ii) **Are physical factors** (illness, drugs, sensory deprivation) contributing? (iii) **Are psychological factors** (depression, psychosis) contributing? These target the treatable causes.
5. Onset and rate of progression are the most valuable historical items. **Sudden onset over days** means delirium; **gradual decline over months to years** means degenerative dementia; **irregular stepwise decrements** favour multi-infarct dementia.

Clinical and cognitive assessment

6. The **Mini-Cog** is a useful screening tool: it combines **three-item recall** (memory) with **clock drawing** (executive function). Executive dysfunction is critical because it determines the ability to perform **IADLs**.
7. Cognition should be assessed by taking **separate histories from the patient and an informant**. Patients with pathological memory impairment typically report no complaints, while relatives report and worry about the loss.
8. Other validated bedside instruments include the **MMSE**, **MoCA**, and **SLUMS**. **Education level must be considered** when interpreting scores — particularly important in the Indian setting, where illiteracy demands vernacular and education-adjusted tools.
9. Mood and affect must be assessed throughout. **Depression, apathy, emotional lability, agitation and aggression** are common and, if missed, lead to misdiagnosis.
10. **Formal neuropsychological testing** by an experienced psychologist is indicated when the diagnosis is uncertain, in the very intelligent or poorly educated, and when depression is suspected.
11. Minimum work-up: focused history and examination (screening for delirium and depression, identifying sensory impairment), **functional status assessment**, mental status examination, and **selected laboratory studies to exclude reversible causes**. Brain imaging should **not** be done automatically in every patient.

Advances in biomarker-based early diagnosis

12. Detecting the presymptomatic phase on clinical grounds alone is difficult, although **accelerated long-term forgetting** may be an early indicator.
13. Current diagnostic criteria therefore **require biomarkers reflecting the three core features: β-amyloid, phosphorylated tau, and neurodegeneration**.
14. These can be measured by **neuroimaging** (amyloid PET, tau PET, MRI) and in **CSF** (Aβ1-42, phosphorylated tau, neurofilament light).
15. **Blood-based biomarkers** — plasma **Aβ42/40 ratio, phosphorylated tau, and neurofilament light** — have become increasingly viable with improvements in assay sensitivity, with major implications for facilitating early diagnosis.
16. Structural MRI supports early diagnosis: **hippocampal and medial temporal atrophy** favours AD; the pattern of atrophy also helps separate AD from vascular and other dementias.
17. Functional imaging refines the differential: **dopamine transporter (loflurane) SPECT is normal in AD but abnormal in Lewy body dementia**; **123I-MIBG cardiac scintigraphy** may distinguish DLB from AD.
18. **Electrophysiology:** altered cortical synaptic plasticity in response to 5-Hz repetitive TMS has been described as a marker in amnestic MCI converting to AD.
19. Even after diagnosis, no intervention has been proven to prevent progression from MCI to dementia; screening is justified because it allows longitudinal follow-up and structured support and education of patients and families.

Latest Updates (2023 – Present)

- **The 2024 Revised Criteria (Jack et al., *Alzheimer's & Dementia* 2024)** reorganise biomarkers into **Core 1** (early-changing: amyloid PET, CSF Aβ42/40, CSF p-tau181/Aβ42, plasma p-tau217) and **Core 2** (later-changing: tau PET, **MTBR-tau243**, p-tau205). **One abnormal Core 1 marker establishes the diagnosis**; Core 2 markers give prognosis and biological stage.
- The full profile is now written **AT1T2NISV** — amyloid, secreted phospho-tau, tau proteinopathy, neurodegeneration, inflammation, α-synuclein co-pathology and vascular brain injury — replacing the simpler 2018 ATN framework.
- **First FDA clearance of a blood test:** on **16 May 2025** the **Lumipulse G pTau217/β-Amyloid 1-42 Plasma Ratio** was cleared for symptomatic adults aged 55 and over in specialist settings, with **91.7% positive and 97.3% negative**

concordance with amyloid PET/CSF (*Drug Discoveries & Therapeutics* 2025).

- Comparable assays are already available elsewhere: **HISCL Aβ42/40 (Japan)**, **PrecivityAD2 (UK)**, and several platforms in China. Head-to-head studies (Lumipulse, ALZpath SIMOA, C2N, Janssen) show broadly comparable accuracy.
- **Alzheimer's Association clinical practice guidelines (2025)** restrict blood biomarker use to **symptomatic patients in specialised care**, require assays meeting defined accuracy thresholds, and stress that results are **adjunctive** — not a stand-alone diagnosis.
- **Two-cutpoint reporting** is now standard for plasma tests: results fall into positive, negative, or an **intermediate zone** requiring confirmatory CSF or PET testing.
- Practical consequence for the geriatrician: a plasma p-tau217 result returned in **2–5 days from a routine venous sample** replaces, for most patients, an expensive PET scan or a lumbar puncture — a decisive advantage in resource-limited settings such as India.

Current Practice Update

Aspect	Textbook position	Current evidence / practice
Diagnostic basis	Clinical syndrome; biomarkers described as emerging	Biological diagnosis; biomarkers are required, not optional
Blood biomarkers	"Increasingly viable" — research stage	FDA-cleared and in specialist clinical use since May 2025
Biomarker framework	Amyloid, phospho-tau, neurodegeneration (ATN)	Core 1 / Core 2 system with full AT1T2NISV profile
MCI	A clinical risk state with no proven intervention	A treatable stage — biomarker-positive MCI is the licensed indication for anti-amyloid therapy
Amyloid confirmation	PET or lumbar puncture	Plasma p-tau217 first, with PET/CSF reserved for intermediate results

3. Newer / Current Drug Therapy for Alzheimer's Disease

Fifteen–Twenty High-Yield Points

Principles

1. Drug therapy in AD has two aims: **symptomatic benefit** (cholinergic and glutamatergic agents) and **disease modification** (anti-amyloid immunotherapy). Non-pharmacological care remains essential alongside both.
2. The **cholinergic hypothesis** underlies symptomatic therapy: degeneration of the nucleus basalis of Meynert reduces cortical acetylcholine, and inhibiting its breakdown partially restores transmission.

Cholinesterase inhibitors

3. **Donepezil** — reversible, selective acetylcholinesterase inhibitor. **Dose 5 mg once daily at night, increased to 10 mg after 4–6 weeks**. Long half-life; once-daily dosing. Licensed across mild, moderate and severe AD.
4. **Rivastigmine** — pseudo-irreversible inhibitor of both **acetylcholinesterase and butyrylcholinesterase**. Available orally and as a **transdermal patch**, which markedly reduces gastrointestinal side effects. Also effective in **Parkinson's disease dementia and Lewy body dementia**, and is the best-evidence agent for **apathy** in PD.
5. **Galantamine** — acetylcholinesterase inhibitor with additional **allosteric modulation of nicotinic receptors**. Given twice daily or as a modified-release capsule.
6. **Tacrine** is of historical interest only — withdrawn because of **hepatotoxicity**.
7. **Efficacy:** meta-analyses of donepezil, galantamine and rivastigmine show small but consistent benefit on cognition and global function, possible delay in nursing-home placement, and a **several-month delay in progression**. Benefit is **marginal in many patients** and must be weighed against cost and adverse effects individually.
8. **Adverse effects** are predominantly cholinergic: nausea, vomiting, diarrhoea, anorexia, weight loss, abdominal cramps; **bradycardia, heart block and syncope**; insomnia and nightmares; muscle cramps; increased bronchial secretions; urinary frequency.
9. **Cautions:** sick sinus syndrome and conduction defects, active peptic ulcer, asthma/COPD, and concurrent use with beta-blockers. Note the interaction with **enzyme-inducing antiepileptics**, which increase the metabolism of donepezil and galantamine.

NMDA antagonist

10. **Memantine** is a low-affinity, uncompetitive **NMDA receptor antagonist** that blocks pathological tonic glutamate activation while preserving physiological signalling, reducing excitotoxicity.
11. Indicated in **moderate-to-severe AD**, either alone or added to a cholinesterase inhibitor. Titrated from 5 mg to 20 mg daily. Dose reduction is needed in renal impairment.
12. Side effects are usually mild: dizziness, headache, constipation, somnolence, hypertension and occasionally confusion or hallucinations.

Treatment of associated symptoms

13. **Depression** in AD is treated with SSRIs; sertraline has trial evidence. Tricyclics and drugs with anticholinergic action must be avoided as they worsen cognition.
14. **Behavioural and psychological symptoms:** non-pharmacological approaches first. **Atypical antipsychotics carry an FDA black-box warning** for increased mortality and cause weight gain; they are reserved for patients who are frankly psychotic or an immediate danger.
15. Every new or worsening behavioural symptom requires a search for a precipitant. **Empirical paracetamol** for undetected pain has shown efficacy in severely demented nursing-home residents.
16. **Donepezil does not treat agitation** in AD — a specific negative trial result worth quoting.

Disease-modifying therapy

17. **Anti-amyloid monoclonal antibodies** bind aggregated Aβ and promote microglial clearance of plaque, producing measurable amyloid clearance on PET and modest slowing of clinical decline.
18. **Indication:** confirmed amyloid pathology with **MCI or mild dementia due to AD only**. They are **not indicated in moderate or severe dementia**.
19. **Key toxicity is ARIA**, requiring baseline and serial MRI monitoring; anticoagulation and thrombolysis for stroke need special caution in treated patients.
20. Drugs that must be **avoided or deprescribed** in AD are as important as drugs prescribed: anticholinergics, benzodiazepines, sedative antihistamines, and other agents on the **Beers** and **STOPP/START** lists.

Latest Updates (2023 – Present)

- **Lecanemab (Leqembi):** approved July 2023; 10 mg/kg IV every two weeks for 18 months. FDA approved **four-weekly IV maintenance (January 2025)**, **subcutaneous maintenance (August 2025)** and a **subcutaneous starter dose (July 2026)**. Now approved in **53 countries**. Weekly subcutaneous injections take about 15 seconds and can be given at home.
- **Donanemab (Kisunla):** approved July 2024; monthly IV infusion with the option to **stop treatment once amyloid PET is negative** — a unique finite-duration regimen. A **modified, more gradual titration was approved in July 2025** (TRAILBLAZER-ALZ 6), reducing ARIA-E from about 24% to 14% at week 24. EU authorisation followed with stricter eligibility, enhanced monitoring and earlier discontinuation protocols.

- **ARIA management:** APOE genotyping is not mandatory in the US but is widely performed because ε4 homozygotes have substantially higher ARIA risk. ARIA can also occur spontaneously in AD with underlying CAA, so pre-treatment microhaemorrhage or superficial siderosis on MRI is a major caution.
- **Aducanumab** has been withdrawn from the market; it is now of historical interest only.
- A **Cochrane review (2026)** of 17 trials and 20,342 participants confirms the class effect is real but small in absolute terms — an important balancing point for a viva answer.
- **Pipeline:** anti-tau antisense oligonucleotides, **trontinemab** (brain-shuttle anti-amyloid antibody with much lower ARIA), semaglutide (EVOKE programme), and combination amyloid–tau strategies. No agent has yet been approved for moderate or advanced disease.
- **Indian context:** anti-amyloid antibodies are not in routine use — cost, the need for amyloid confirmation, and serial MRI monitoring are limiting. Cholinesterase inhibitors and memantine remain the practical mainstay under NPHCE-supported services.

Current Practice Update

Aspect	Textbook position	Current evidence / practice
Therapeutic aim	Symptomatic only	Symptomatic plus disease modification in early biomarker-confirmed disease
Approved drug classes	Cholinesterase inhibitors, memantine	Above plus anti-amyloid monoclonal antibodies (lecanemab, donanemab)
Route of administration	Oral (and rivastigmine patch)	Adds IV and, for lecanemab, subcutaneous home administration
Monitoring	Clinical response, pulse rate, weight	Adds serial MRI for ARIA and amyloid PET to decide on stopping donanemab
Aducanumab	Not discussed	Withdrawn from market

4. Biomarkers for Alzheimer's Dementia

Fifteen–Twenty High-Yield Points

Concept

1. A biomarker is an objectively measured indicator of a normal or pathological process. In AD they are needed because **clinical diagnosis alone is unreliable** and pathology begins **years to decades before symptoms**.
2. Current diagnostic criteria **require biomarkers reflecting the three core features of the disease: β-amyloid, phosphorylated tau, and neurodegeneration** — the basis of the **ATN classification**.
3. Biomarkers serve four purposes: **diagnosis, staging, prognosis, and monitoring treatment response** (including patient selection for anti-amyloid therapy).

Cerebrospinal fluid biomarkers

4. **CSF Aβ1-42 is reduced** in AD because amyloid is sequestered into plaques. The **Aβ42/40 ratio** is preferred because it corrects for individual differences in total amyloid production.
5. **CSF phosphorylated tau (p-tau181) and total tau are raised**, reflecting tangle formation and neuronal injury respectively. The combination of **low Aβ42 with high p-tau** is the classical AD signature.
6. A useful discriminator: in **normal pressure hydrocephalus** CSF Aβ42 is reduced as in AD, but **a rise in phospho-tau is seen only in AD** — a high-yield point for the NPH-versus-AD viva question.
7. **CSF neurofilament light (NfL)** is a marker of axonal degeneration; it is elevated but **non-specific**, rising in many neurodegenerative and inflammatory conditions.

Imaging biomarkers

8. **Amyloid PET** using fibrillar Aβ ligands demonstrates plaque burden *in vivo*. A significant plaque burden may be present in cognitively normal people, so a positive scan alone does not equal dementia.
9. **Tau PET** tracks tangle deposition, which follows Braak staging and **correlates far better with cognitive decline than amyloid does**.
10. **Structural MRI** shows **hippocampal and medial temporal lobe atrophy**, ventricular enlargement, and cortical thinning. Longitudinal studies show cognitively normal people with low CSF Aβ1-42 have **faster whole-brain and hippocampal volume loss**.
11. MRI also identifies co-pathology that alters the clinical threshold: **white matter hyperintensities, lacunes, microinfarcts, microbleeds and dilated perivascular spaces**. Microbleeds require **T2*-gradient echo** sequences; white matter lesions are best seen on **FLAIR**.
12. **Functional/molecular imaging for differential diagnosis:** dopamine transporter (loflurane) SPECT is **normal in AD but abnormal in Lewy body dementia**; FDG-PET shows temporoparietal and posterior cingulate hypometabolism in AD.

Blood-based biomarkers

13. Advances in assay sensitivity have made **blood-based biomarkers increasingly viable** — specifically **plasma Aβ42/40, phosphorylated tau, and neurofilament light** — with significant implications for facilitating early diagnosis.
14. Their attraction is practical: **low cost, minimally invasive, repeatable, and scalable** to primary care and to population screening in high-burden low-resource settings.

Genetic and other markers

15. **APOE ε4** is a susceptibility (risk) marker, not a diagnostic marker. It is **neither sensitive nor specific** and is **not recommended for screening**.
16. Deterministic mutations (**APP, PSEN1, PSEN2**) are diagnostic only in early-onset familial disease and mandate genetic counselling.
17. **Electrophysiological markers:** impaired LTP-like cortical plasticity on 5-Hz repetitive TMS has been described in amnestic MCI that converts to AD.
18. Caveats for the viva: biomarker positivity in a cognitively normal person indicates **preclinical disease, not dementia**; mixed pathology is common in the very old; and cut-offs vary between assays and platforms.

Latest Updates (2023 – Present)

- **Plasma p-tau217 is the single best-performing blood biomarker**, with diagnostic accuracy approaching CSF and amyloid PET; it is explicitly named in the 2024 criteria as an acceptable Core 1 marker (*Alzheimer's & Dementia* 2024).
- **FDA clearance, 16 May 2025:** the **Lumipulse G pTau217/β-Amyloid 1-42 Plasma Ratio** — the first blood test cleared to identify amyloid pathology — for symptomatic adults 55 years and older in specialist settings; **91.7% positive and 97.3% negative concordance** with PET/CSF.
- **2024 Revised Criteria biomarker architecture: Core 1** = amyloid PET, CSF Aβ42/40, CSF p-tau181/Aβ42, plasma p-tau217 — one abnormal result diagnoses AD. **Core 2** = tau PET, **MTBR-tau243**, p-tau205, non-phosphorylated mid-region tau — these stage the disease and give prognosis.
- The framework has been broadened from ATN to **AT1T2NISV**: A (amyloid), T1 (secreted phospho-tau), T2 (tau proteinopathy), N (neurodegeneration), I (inflammation — GFAP), S (α-synuclein co-pathology), V (vascular brain injury).

- **Alzheimer's Association appropriate-use and clinical practice guidance (2022, updated 2025)**: blood biomarkers should be used only in symptomatic patients, in specialised settings, with assays meeting pre-defined accuracy standards and **two-cutpoint reporting** that flags an intermediate zone for confirmatory testing.
- **Other cleared or marketed assays** include **PrecivityAD2** (UK) and **HISCL Aβ42/40** (Japan); ALZpath SIMOA, C2N and Janssen p-tau217 assays perform comparably in ADNI head-to-head comparisons.
- Biomarkers are now also **pharmacodynamic endpoints**: donanemab's label change was supported by preserved reduction in amyloid and **plasma p-tau217** despite the gentler titration.

Current Practice Update

Aspect	Textbook position	Current evidence / practice
Framework	ATN — amyloid, tau, neurodegeneration	Core 1 / Core 2, with full AT1T2NISV profile
Blood biomarkers	Emerging, "increasingly viable"	FDA-cleared and in routine specialist use
Best single marker	CSF Aβ42 with p-tau	Plasma p-tau217 (or the p-tau217/Aβ42 ratio)
Purpose	Diagnosis and research	Also selects patients for, and monitors response to, disease-modifying therapy
Diagnostic threshold	Clinical syndrome plus supportive biomarker	A single abnormal Core 1 biomarker is sufficient to diagnose AD

5. Emerging Biomarkers in Cognition

Fifteen–Twenty High-Yield Points

Rationale

1. Cognitive impairment in the elderly is usually the result of **mixed pathology**. More than 230 combinations of neuropathology have been described, and altered proteins occur even in cognitively normal older people.
2. Emerging biomarkers therefore aim to move beyond amyloid and tau to capture **the full range of contributing processes** — synucleinopathy, TDP-43, vascular injury, inflammation, and synaptic loss.
3. The four properties sought are: **early positivity, high specificity, minimal invasiveness, and low cost** enough for population use.

Protein aggregation markers

4. **Hyperphosphorylated tau, amyloid-β, α-synuclein and TDP-43** are the four principal aggregating proteins; all four are found in the brains of cognitively normal older people, which limits the specificity of any one marker.
5. **α-Synuclein seed amplification assay (SAA)** in CSF detects Lewy body pathology with high accuracy and is the key emerging marker for DLB and Parkinson's disease dementia; skin biopsy assays are in development.
6. **TDP-43**-related pathology (LATE — limbic-predominant age-related TDP-43 encephalopathy) is a common mimic of AD in the very old; no validated fluid biomarker yet exists, which is an important knowledge gap.

Neurodegeneration and glial markers

7. **Neurofilament light (NfL)** in CSF and plasma is a sensitive but **disease-non-specific** marker of axonal injury; it is most useful for tracking progression and treatment response rather than diagnosis.
8. **Glial fibrillary acidic protein (GFAP)** in plasma reflects reactive astrocytosis and rises very early — often before p-tau217 — making it a candidate marker of the earliest amyloid-associated response.
9. Inflammatory markers under study include **soluble TREM2, YKL-40, and IL-6**, reflecting the microglial and astrocytic activation that is a hallmark of brain ageing.

Synaptic and metabolic markers

10. **Neurogranin** (post-synaptic) and **SNAP-25** and **synaptotagmin-1** (pre-synaptic) in CSF index synaptic loss, which correlates with cognition better than plaque burden.
11. **SV2A PET** imaging quantifies synaptic density *in vivo* and is emerging as a sensitive measure of early degeneration.
12. Loss of **aerobic glycolysis** and altered brain energy metabolism are described hallmarks of brain ageing and can be imaged with FDG-PET.

Imaging and physiological markers

13. **Diffusion tensor imaging** demonstrates loss of white matter integrity following an **anterior-to-posterior gradient** — frontal lobes first, occipital last — consistent with the "last in, first out" model of myelination.
14. **Small vessel disease markers** (white matter hyperintensities on FLAIR, lacunes, microinfarcts, microbleeds on T2*GRE, dilated perivascular spaces) are increasingly recognised as major determinants of cognitive decline and disability in the older person.
15. **Retinal biomarkers** — optical coherence tomography measurement of retinal nerve fibre layer thinning — are an accessible surrogate of neuroaxonal loss already validated in demyelinating disease and under study in dementia.
16. **Electrophysiology:** impaired LTP-like plasticity on 5-Hz repetitive TMS distinguishes amnestic MCI that converts to AD; quantitative EEG connectivity measures are also being studied.

Clinical and digital markers

17. **Accelerated long-term forgetting** — normal learning with abnormally rapid loss over days to weeks — may be a clinical marker of presymptomatic disease.
18. **Gait speed and dual-task gait** are practical physical biomarkers: slowing of walking speed under a concurrent cognitive task predicts cognitive decline and is measurable with a stopwatch over 10 metres.
19. Digital biomarkers — passive smartphone and wearable data on typing, speech, sleep and mobility — are the most rapidly growing category and are attractive for community screening.
20. **Interpretation caveat:** no emerging biomarker should be used in isolation. Each must be interpreted against the clinical syndrome, education level, and the high probability of mixed pathology in old age.

Latest Updates (2023 – Present)

- The **2024 Revised Criteria** formally incorporate emerging markers by category: **N** (NfL, neurodegeneration), **I** (GFAP, inflammation), **S** (α-synuclein), **V** (vascular brain injury) — explicitly recognising co-pathology (*Alzheimer's & Dementia* 2024).
- **MTBR-tau243** (microtubule-binding region tau 243) has emerged as the leading fluid marker of **tau tangle burden**, correlating with tau PET, and is classified as a Core 2 biomarker for biological staging.
- **p-tau205** and non-phosphorylated mid-region tau fragments are additional Core 2 candidates that change later than p-tau217 and align more closely with symptom onset.
- Plasma **GFAP** and **NfL** are now widely used as secondary endpoints in trials; NfL reduction was the surrogate that supported **accelerated approval of tofersen in SOD1-ALS**, illustrating the regulatory power of a validated biomarker.

- Multiplex platforms (SIMOA, chemiluminescence, and immunoprecipitation mass spectrometry) allow simultaneous measurement of p-tau217, Aβ42/40, GFAP and NfL from a single tube of blood — the direction of travel for memory clinics.
- **Indian relevance:** plasma-based panels are the only realistic route to large-scale case-finding given that amyloid PET is available in only a handful of Indian centres; validation in Indian cohorts, including LASI-DAD participants, is a current research priority.

Current Practice Update

Aspect	Textbook position	Current evidence / practice
Scope of biomarkers	Amyloid, tau, neurodegeneration	Extended to inflammation, α-synuclein, vascular injury and synaptic loss
Tau markers	CSF total tau and p-tau181	p-tau217 for diagnosis; MTBR-tau243 and p-tau205 for staging
Astrocyte markers	Not described as biomarkers	Plasma GFAP is a validated early marker
α-Synuclein	Described as a pathological protein only	CSF seed amplification assay is a clinically usable diagnostic test
Sampling	CSF and PET	Multiplexed plasma panels from a single venous sample

6. Surgical Management of Parkinsonism

Fifteen–Twenty High-Yield Points

Rationale and patient selection

1. Surgery is considered when **motor complications become refractory to best medical therapy** — that is, disabling motor fluctuations or dyskinesia despite optimised levodopa and adjuncts.
2. The essential principle is that **only levodopa-responsive symptoms improve after surgery**. A good levodopa response is the single best predictor of surgical benefit; levodopa-unresponsive axial features, gait freezing, falls and dysautonomia do not improve.
3. Candidates must be **cognitively intact**. Dementia, active psychiatric illness (especially depression with suicidality) and severe comorbidity are limitations.
4. Diagnosis must be secure — **Parkinson-plus syndromes respond poorly and are a contraindication**. The **Queen Square Brain Bank criteria** should be satisfied.

Deep brain stimulation — the standard operation

5. **Bilateral deep brain stimulation (DBS) of the subthalamic nucleus (STN) or globus pallidus interna (GPi)** produces **sustained improvement in motor function**, especially in symptoms that respond to levodopa, and **reduces drug-induced involuntary movements**.
6. **STN stimulation** allows the greatest reduction in dopaminergic drug dose (typically 40–60%), which indirectly reduces dyskinesia; **GPi stimulation** has a more direct antidyskinetic effect and is often preferred where cognition or mood is fragile.
7. The **ventral intermediate nucleus of the thalamus (VIM)** is the target when **tremor is the dominant and disabling symptom**, including in essential tremor. It does not help bradykinesia or rigidity.
8. Mechanism: high-frequency stimulation functionally inhibits or desynchronises pathologically synchronised basal ganglia output, normalising the abnormal beta-band oscillations in the motor circuit.

Age and outcomes

9. The textbook position is that **DBS is not currently used in elderly people with PD**. However, recent observational studies found that **efficacy in patients over 70 was similar to that in younger patients, and the incidence of adverse effects did not increase with age**.
10. Long-term follow-up shows that **disability continues to progress after DBS**, probably because the underlying neurodegeneration continues — DBS is symptomatic, not disease-modifying.

Complications

11. **Procedure-related:** intracranial haemorrhage, stroke, seizure and infection at the implant site; these relate to the intracranial procedure itself.
12. **Hardware-related:** lead migration or fracture, extension wire erosion, device infection, and the need for battery replacement.
13. **Stimulation-related:** dysarthria, paraesthesiae, eyelid-opening apraxia, gait and balance deterioration, and weight gain.
14. **Neuropsychiatric:** there are long-standing concerns about long-term safety, with **risks of depression and suicide**, as well as apathy, impulsivity and, in vulnerable patients, cognitive decline.

Lesional and other surgical procedures

15. **Lesional surgery** — thalamotomy, pallidotomy and subthalamotomy — preceded DBS and remains an option where DBS is unaffordable or follow-up is impractical. It is usually performed **unilaterally**, since bilateral lesions carry a high risk of dysarthria and cognitive decline.
16. **Gamma knife radiosurgery** and **MR-guided focused ultrasound thalamotomy** are incisionless alternatives, described in the textbook as options for severe medication-refractory tremor.
17. DBS is also used for other movement disorders in the elderly: **essential tremor** (VIM), **refractory dystonia** (particularly effective in primary dystonia), and occasionally **tardive dyskinesia**.
18. Continuous-infusion therapies (intrajejunal levodopa gel via PEG-J, apomorphine) are the main **non-DBS device-aided** alternatives for advanced disease and are relevant when DBS is contraindicated.
19. Rehabilitation must continue after surgery. Functional rehabilitation preserves motor function, and sessions are best timed to the **peak effect of levodopa**.
20. Multidisciplinary assessment before surgery must include a movement disorder neurologist, neurosurgeon, neuropsychologist and psychiatrist, with a formal **levodopa challenge** (150–250 mg in the fasting state, with assessment every 30 minutes for 2–3 hours).

Latest Updates (2023 – Present)

- **Adaptive (closed-loop) DBS was approved by the FDA in February 2025** — a Medtronic software update based on the **ADAPT-PD** randomised crossover trial. The device senses **local field potentials (beta oscillations)** and adjusts stimulation in real time, improving symptom control with lower energy use and fewer stimulation-induced side effects.
- **Bilateral MR-guided high-intensity focused ultrasound was approved in 2025** (having been unilateral-only since 2017). Approved targets now include the **VIM thalamus, GPi and the pallidothalamic tract**, with the STN under

investigation (*Current Neurology and Neuroscience Reports* 2026).

- **Image-guided and remote programming** shorten programming time while matching motor outcomes and expand access for patients who cannot travel — directly relevant to Indian practice, where DBS centres are concentrated in metros.
- Age is no longer treated as an absolute barrier: long-term series with up to seven years' follow-up show comparable outcomes in older and younger cohorts, and current practice selects on **biological rather than chronological age** (*J Neurosurg* 2018; *JAMA Neurol* 2015).
- **Regenerative surgery: bemdaneprocel** (stem cell-derived dopaminergic neuron transplantation) entered a Phase 3 trial (exPDite-2) in 2025, and **AB-1005 GDNF gene therapy** received FDA RMAT designation in February 2025 — the first credible restorative surgical strategies.

Current Practice Update

Aspect	Textbook position	Current evidence / practice
DBS in the elderly	"Not currently used in elderly people with PD"	Used routinely in selected patients over 70; efficacy and safety comparable to younger patients
Stimulation mode	Continuous, fixed-parameter (open-loop)	Adaptive closed-loop DBS approved February 2025
Focused ultrasound	Reserved for severe refractory tremor, unilateral	Bilateral approval 2025; targets extended to GPi and pallidothalamic tract
Programming	In-person, iterative	Image-guided and remote programming
Restorative surgery	Not available	Cell therapy and gene therapy in Phase 2–3 trials

7. Recent Advances in the Management of Parkinsonism

Fifteen–Twenty High-Yield Points

Baseline — established management

1. **Levodopa remains the main and often the only treatment for PD in the elderly.** It is combined with a peripheral decarboxylase inhibitor — **Madopar (levodopa + benserazide)** or **Sinemet (carbidopa + levodopa)**.
2. Start low and increase very gradually; most patients are controlled on **300–600 mg/day in the first years**, given three or four times daily, preferably 30 minutes before meals. **Dispersible Madopar** achieves faster peak levels and is used for early-morning akinesia.
3. **Dopamine agonists, anticholinergics and (largely) MAO-B inhibitors are avoided in older patients** — agonists because of hallucinations, somnolence, impulse-control disorders and orthostatic hypotension; anticholinergics such as trihexyphenidyl because of confusion, urinary retention and glaucoma.
4. **COMT inhibitors (entacapone, tolcapone)** prolong the levodopa effect and are useful in older patients with motor fluctuations or dyskinesia. Tolcapone requires monitoring for hepatotoxicity.
5. Non-motor symptoms have specific best-evidence treatments: **venlafaxine** for depression, **clozapine or pimavanserin** for hallucinations, **rivastigmine** for dementia and apathy, **droxidopa/midodrine/fludrocortisone** for orthostatic hypotension, **domperidone** for nausea, **botulinum toxin** for severe drooling, and **macrogol with fibre and hydration** for constipation.
6. Clozapine requires blood count monitoring and must be **stopped permanently if the neutrophil count falls below 1500/mm^3**. Domperidone requires ECG monitoring for QT prolongation.
7. Non-pharmacological care is integral: **early functional rehabilitation timed to peak levodopa effect**, speech therapy for dysarthria and drooling, swallowing assessment, home support, and structured patient and caregiver education.

Advances in drug delivery

8. The central therapeutic problem in advanced PD is **pulsatile stimulation** of striatal dopamine receptors by short-acting oral levodopa. Every recent advance addresses this by achieving **continuous dopaminergic stimulation**.
9. **Extended-release oral levodopa formulations** reduce dosing frequency and smooth plasma levels.
10. **Continuous subcutaneous infusion** of levodopa prodrugs and of apomorphine delivers 24-hour dopaminergic stimulation without the need for surgery.
11. **Intrajejunal levodopa-carbidopa gel** via PEG-J remains a standard device-aided therapy for advanced disease.

Advances in device therapy

12. **Deep brain stimulation of STN or GPi** gives sustained improvement in levodopa-responsive symptoms and reduces dyskinesia; recent observational data show equal efficacy and no excess adverse events in patients over 70.
13. **MR-guided focused ultrasound** and **gamma knife** offer incisionless lesioning for medication-refractory tremor.
14. Technology in PD — wearable sensors and digital monitoring — is an established area of development for objective measurement of fluctuations.

Advances in diagnosis and disease modification

15. **Dopamine transporter (loflurane) SPECT** separates degenerative parkinsonism (abnormal) from drug-induced parkinsonism, vascular parkinsonism, essential tremor and AD (all normal).
16. No treatment has yet been shown to be disease-modifying. Selegiline and rasagiline were proposed for neuroprotection but this remains unproven.
17. Strategies "beyond dopamine" — targeting α-synuclein aggregation, LRRK2, GBA and neuroinflammation — are the main direction of current research.
18. Exercise is an effective intervention in PD and is included in all recent position statements on exercise prescription in older adults.

Latest Updates (2023 – Present)

- **New drug delivery approvals: Crexont (IPX203)**, an extended-release oral carbidopa/levodopa, approved **August 2024**; **Vyalev/Produodopa (foscarbidopa/foslevodopa)**, the first **24-hour continuous subcutaneous levodopa infusion**, approved **October 2024**; **Onapgo** (subcutaneous apomorphine infusion) for OFF episodes, approved **February 2025**. **ND0612** (continuous subcutaneous levodopa/carbidopa) was resubmitted to the FDA in 2025.
- **Adaptive DBS approved February 2025** (ADAPT-PD trial) — senses beta-band local field potentials and titrates stimulation in real time. **Bilateral focused ultrasound approved 2025**, with GPi and pallidothalamic tract added as targets.
- **Tavapadon**, a selective **D1/D5 partial agonist**, completed the Phase 3 TEMPO programme with significant MDS-UPDRS improvement and increased ON time without troublesome dyskinesia; NDA filed September 2025, decision expected Q3 2026. If approved it would be the first new oral dopamine agonist in over a decade, and its D1/D5 selectivity may avoid the D2/D3-mediated impulse-control and somnolence problems that make current agonists unsuitable for older patients.
- **Safety alert (March 2026):** the FDA required a new label warning across **all carbidopa/levodopa products** (including Sinemet, Rytary, Stalevo, Crexont and Vyalev) for **vitamin B6 deficiency and seizures** — a highly

examinable, practice-changing point. High-dose levodopa consumes pyridoxine through metabolism, and B6 status should be checked in patients on high total daily doses or continuous infusion.

- **Disease modification remains elusive. Prasinezumab** (anti-α-synuclein) entered a 900-patient Phase 3 trial in November 2025. Two candidates failed in the first half of 2026 — the LRRK2 inhibitor **BIIB122** and the GCase modulator **BIA 28-6156** — and were discontinued.
- **Restorative therapy: bemdaneprocel** (stem cell-derived dopaminergic neurons) entered Phase 3 (exPDite-2) in 2025; **AB-1005** GDNF gene therapy received RMAT designation.
- **Lifestyle as therapy:** a 2026 *Lancet Neurology* review positions structured exercise and lifestyle intervention as contributors to both symptom management and possible disease modification.

Current Practice Update

Aspect	Textbook position	Current evidence / practice
Continuous dopaminergic delivery	Intrajejunal gel; subcutaneous apomorphine boluses	24-hour subcutaneous foscarbidopa/foslevodopa and apomorphine infusion; extended-release oral IPX203
Dopamine agonists in the elderly	Avoided altogether	Still avoided, but D1/D5-selective tavapadon may change this
DBS	Fixed continuous stimulation; not for the elderly	Adaptive closed-loop stimulation; age is not a bar
Levodopa safety	Nausea, hypotension, psychiatric effects, dyskinesia	Adds FDA warning (2026) on vitamin B6 deficiency and seizures
Disease modification	Unproven neuroprotection with MAO-B inhibitors	α-Synuclein antibodies, cell and gene therapy in Phase 2–3; still nothing approved

8. Management of Motor Fluctuations in Parkinson's Disease

Fifteen–Twenty High-Yield Points

Definition and types

1. **Motor fluctuations** are predictable or unpredictable changes in motor performance in relation to levodopa dosing, appearing after several years of treatment. They occur together with **involuntary movements — chorea, athetosis and dystonia**.
2. **Wearing-off** (end-of-dose deterioration) is the earliest and commonest form — return of parkinsonian symptoms before the next dose is due.
3. **Delayed-on** and **dose failure (no-on)** result from impaired gastric emptying and competition with dietary protein for absorption.
4. **On-off phenomenon** is abrupt, unpredictable switching between mobility and immobility, unrelated to dose timing.
5. **Early-morning akinesia** and **early-morning/off-period dystonia** (typically painful foot dystonia) reflect overnight levodopa washout.
6. **Freezing of gait** may be off-period (levodopa-responsive) or on-period (levodopa-resistant); the distinction determines whether more drug will help.
7. Dyskinesias are classified by their timing: **peak-dose dyskinesia** (commonest, choreiform), **diphasic dyskinesia** (at the beginning and end of dose, often dystonic and painful in the legs), and **off-period dystonia**.
8. **Non-motor fluctuations** parallel the motor ones — anxiety, panic, pain, sweating, akathisia, breathlessness and mood swings in the off state — and are frequently missed.

Mechanism

9. Fluctuations arise from a **narrowing therapeutic window**: progressive loss of nigrostriatal terminals removes the buffering capacity to store and release dopamine, so the striatum becomes directly dependent on the short plasma half-life of levodopa. This **pulsatile stimulation** induces post-synaptic changes that generate dyskinesia.

Assessment

10. Assessment begins with a **patient-completed diary** recording on time, off time, and dyskinesia against each dose, together with meal timing and constipation.
11. Exclude **malabsorption and delayed gastric emptying**, including *Helicobacter pylori* infection and severe constipation, before concluding that the disease has advanced. Also review any newly added dopamine-blocking drug.

Pharmacological management

12. **First step — manipulate levodopa:** revise the timing of administration, fractionate the dose (smaller doses given more frequently), and give doses 30 minutes before meals to avoid protein competition.
13. **Dispersible levodopa** (dispersible Madopar) reaches peak plasma concentration faster and is used as a rescue dose for early-morning akinesia and delayed-on, allowing the standard dose to be taken at the same time.
14. **COMT inhibitors — entacapone and tolcapone — decrease dopamine degradation, increasing and prolonging the effect of levodopa, and are specifically useful in older patients with motor fluctuations and/or levodopa-induced dyskinesia.** Entacapone 200 mg is given with each levodopa dose.
15. Adding a **controlled-release levodopa preparation at night** helps nocturnal akinesia and early-morning off dystonia.
16. **Amantadine** is the best-established antidyskinetic drug, acting through NMDA antagonism, but must be used cautiously in the elderly because it causes **delirium, hallucinations and livedo reticularis** and is renally excreted, requiring dose reduction in renal impairment.
17. Dopamine agonists and MAO-B inhibitors smooth fluctuations in younger patients but are generally avoided in the elderly because of neuropsychiatric adverse effects.
18. **Reducing the individual levodopa dose while increasing frequency** is the standard manoeuvre for peak-dose dyskinesia; diphasic dyskinesia, paradoxically, may improve by increasing the dose.

Device-aided and surgical management

19. **Continuous dopaminergic delivery** is the definitive answer to refractory fluctuations: intrajejunal levodopa-carbidopa gel via PEG-J, or continuous subcutaneous apomorphine infusion.
20. **Bilateral DBS of STN or GPi** is offered to cognitively intact patients with **severe disability from motor complications refractory to best medical therapy**; it gives sustained improvement in levodopa-responsive symptoms and reduces drug-induced involuntary movements.

Latest Updates (2023 – Present)

- **Vyalev / Produodopa (foscarbidopa/foslevodopa)**, approved **October 2024**, is the first 24-hour continuous subcutaneous levodopa infusion for advanced motor fluctuations. It gives the benefits of intrajejunal gel **without a PEG-J**, an important advance for frail older patients in whom endoscopic gastrostomy is high risk. Infusion-site reactions are the main limitation.
- **Onapgo** (continuous subcutaneous apomorphine infusion) was approved in **February 2025** for OFF episodes, adding to the intermittent apomorphine pen.
- **Crexont (IPX203)**, approved **August 2024**, combines immediate-release granules with extended-release beads to give longer good ON time with fewer daily doses.

- **Adaptive DBS (approved February 2025, ADAPT-PD)** is specifically aimed at fluctuations: it senses beta oscillations and increases stimulation during OFF periods and reduces it during ON periods, thereby narrowing the swing between under- and over-stimulation.
- A 2026 review of advanced therapies confirms that subcutaneous foscarbidopa/foslevodopa and apomorphine both **reduce OFF time and increase "good ON time"**, with infusion-site reactions the principal concern (*Current Neurology and Neuroscience Reports* 2026).
- **Tavapadon** (D1/D5 partial agonist) showed increased ON time **without troublesome dyskinesia** in the Phase 3 TEMPO programme in patients with motor fluctuations; FDA decision expected in 2026.
- **FDA warning, March 2026:** all carbidopa/levodopa products now carry a label warning for **vitamin B6 deficiency and seizures** — particularly relevant to patients on high-dose or continuous-infusion regimens for fluctuations.

Current Practice Update

Aspect	Textbook position	Current evidence / practice
First-line manoeuvre	Fractionate levodopa; add COMT inhibitor	Unchanged — still the correct first step
Continuous delivery	Intrajejunal gel (needs PEG-J) or apomorphine boluses	24-hour subcutaneous foscarbidopa/foslevodopa and apomorphine infusion — no gastrostomy required
Oral long-acting levodopa	Controlled-release tablets	IPX203 (Crexont) dual-release formulation
DBS for fluctuations	Fixed-parameter stimulation	Closed-loop adaptive stimulation responsive to ON/OFF state
Monitoring	Patient diary	Wearable sensors giving objective ON/OFF and dyskinesia data

9. Parkinsonism-Plus Syndromes

Fifteen–Twenty High-Yield Points

Definition and approach

1. **Parkinsonism-plus (atypical parkinsonian) syndromes** are neurodegenerative diseases in which an extrapyramidal syndrome is accompanied by additional features not seen in idiopathic PD, and in which the **response to levodopa is poor or absent**.
2. The general red flags for atypical parkinsonism are: **early falls and postural instability, early severe autonomic failure, early dementia, supranuclear gaze palsy, cerebellar or pyramidal signs, symmetrical onset, absence of rest tremor, and rapid progression**. Most of these appear in the **exclusion criteria of the Queen Square Brain Bank criteria** for PD.
3. Response to levodopa is assessed by giving **150–250 mg of levodopa with a decarboxylase inhibitor to a fasting patient** and assessing tremor, rigidity and limb akinesia every 30 minutes for two to three hours. A negative response to large doses of levodopa (malabsorption excluded) is an exclusion criterion for PD.

Progressive supranuclear palsy (PSP)

4. PSP is **frequently misdiagnosed as PD**. It is a **4-repeat tauopathy** characterised by an extrapyramidal syndrome with complex eye movement dysfunction.
5. **Tremor is infrequent and rigidity predominates in the spine (axial rigidity)**, with neck dystonia (retrocollis).
6. **Postural and gait abnormalities occur early and cause repeated falls**, aggravated by attentional and visual problems. Falls within the first year are a classic examination point.
7. **Eye movement problems:** inability to shift gaze voluntarily downward, **impaired (slowed) saccades**, and difficulty controlling the eyelids (apraxia of lid opening, blepharospasm). **Limitation of vertical gaze** is the cardinal sign.
8. Motor symptoms are **unresponsive to levodopa**. As the disease progresses, personality change, dementia and swallowing problems are common.
9. **MRI shows midbrain atrophy**, and **ocular electrophysiology (impaired saccades)** also helps recognise the disease.

Multiple system atrophy (MSA)

10. MSA is **very rarely observed as very late-onset parkinsonism**. It is an α-synucleinopathy with glial cytoplasmic inclusions.
11. The defining feature is **severe dysautonomia early in the course of parkinsonism** — orthostatic hypotension, urinary disorder, sexual dysfunction and gastrointestinal symptoms.
12. **Pyramidal or cerebellar signs** accompany the parkinsonism. Subtypes are **MSA-P** (parkinsonian predominant) and **MSA-C** (cerebellar predominant, with dysmetria, scanning speech, saccadic abnormalities and truncal ataxia).

Other syndromes in the differential

13. **Corticobasal degeneration: dystonia, myoclonus, cortical sensory loss and alien (foreign) hand syndrome**, typically markedly asymmetrical.
14. **Dementia with Lewy bodies:** dementia occurring **a few months before or after** the onset of parkinsonism, with **visual hallucinations, fluctuating alertness and severe adverse reactions to neuroleptics**. By contrast, PD dementia occurs in advanced stages after several years of PD.
15. **Drug-induced parkinsonism:** exposure to neuroleptics (present or past) must be sought carefully by interviewing patients, proxies, physicians and pharmacists. It is **symmetrical, tremor-poor, and loflurane SPECT is normal**. Neuroleptic treatment at onset of symptoms is a Queen Square exclusion criterion.
16. **Vascular (cerebrovascular) parkinsonism:** multiple infarcts, usually a **multilacunar state**, with subcortical dementia and pseudobulbar state; posture, gait and swallowing problems are prominent, tremor is infrequent and levodopa response is poor. **Onset or worsening of extrapyramidal signs within one month of a stroke strongly supports the diagnosis.** MRI shows multiple lacunae and/or severe leukoaraiosis.
17. **Normal pressure hydrocephalus:** gait disturbance, dementia and urinary incontinence, with short shuffling steps but **no tremor or rigidity**; imaging shows marked ventricular enlargement out of proportion to cortical atrophy.
18. Other rarer causes: **Huntington's disease** (chorea, familial), **Wilson's disease** (liver disease, Kayser-Fleischer ring, psychiatric symptoms), and toxic, post-encephalitic and repeated head trauma-related parkinsonism.
19. About **15% of patients with Alzheimer's disease develop extrapyramidal signs**; these are usually mild, and their presence at diagnosis predicts more rapid decline. **Loflurane SPECT is normal in AD and abnormal in DLB**, which is the key discriminator.
20. **Management is largely supportive:** a levodopa trial is still justified (a minority of MSA and PSP patients show partial benefit), with physiotherapy, speech and swallow therapy, falls prevention, treatment of orthostatic hypotension, and early advance care planning. Prognosis is substantially worse than PD, with median survival of about 6–9 years.

Latest Updates (2023 – Present)

- **MDS diagnostic criteria for MSA (2022)** introduced the categories **clinically established MSA, clinically probable MSA** and the research category **prodromal MSA**, and added MRI markers as supportive features — a substantial change from the older Gilman consensus criteria.
- **MDS-PSP criteria (Höglinger et al.)** define multiple clinical phenotypes beyond classical Richardson syndrome —

PSP-P (parkinsonism), **PSP-PGF** (progressive gait freezing), **PSP-CBS**, **PSP-SL** (speech/language) and **PSP-F** (frontal) — explaining why many cases are missed early.

- **α-Synuclein seed amplification assay (SAA)** in CSF now distinguishes synucleinopathies (MSA, DLB, PD) from tauopathies (PSP, CBD) with high accuracy, and is entering clinical use.
- **Plasma and CSF neurofilament light (NfL)** is markedly higher in atypical parkinsonism than in PD and is the most useful single fluid biomarker for separating PD from Parkinson-plus syndromes at an early stage.
- **Therapeutic trials:** anti-tau strategies in PSP and anti-α-synuclein approaches in MSA have so far been negative; management remains symptomatic. Droxidopa and midodrine for neurogenic orthostatic hypotension in MSA are the main pharmacological advances.
- The **2024 Revised AD Criteria** include **S (α-synuclein)** as a co-pathology biomarker category, reflecting how often synucleinopathy and AD coexist in the older brain.

Current Practice Update

Aspect	Textbook position	Current evidence / practice
Diagnostic criteria	Descriptive clinical features	MDS criteria for PSP (multiple phenotypes) and MSA (2022, with prodromal category)
Biomarkers	Imaging and clinical assessment only	CSF α-synuclein SAA and NfL differentiate PD from Parkinson-plus
PSP phenotype	Classical Richardson syndrome only	Recognised as a spectrum including PSP-P, PSP-PGF, PSP-CBS
MSA autonomic therapy	Midodrine, fludrocortisone	Droxidopa is best-evidence therapy where available

10. Pathognomonic MRI Findings in Progressive Supranuclear Palsy and Multiple System Atrophy

Fifteen–Twenty High-Yield Points

General principle

1. Structural MRI in parkinsonism serves two purposes: **to exclude secondary causes** (vascular disease, hydrocephalus, tumour) and **to identify positive imaging signatures of atypical parkinsonism**. MRI is normal in idiopathic PD.
2. The textbook states directly that **a typical pattern of MRI imaging has been described in progressive supranuclear palsy and relates to midbrain atrophy**, and illustrates the **convex-to-concave change in the superior midbrain profile**.

PSP — midbrain signs

3. **The normal superior margin of the midbrain is convex. In PSP it becomes flat and then concave** — this single observation on a midsagittal T1 image is the basis of most PSP signs.
4. **Hummingbird (penguin) sign** — midsagittal T1: selective atrophy of the **rostral midbrain tegmentum** with a **relatively preserved pons**. The atrophic midbrain forms the bird's head and beak; the normal pons forms the body. **Specificity about 99.5%, sensitivity about 51.6%**.
5. **Morning glory flower sign** — axial T2: **concavity of the lateral margin of the midbrain tegmentum**, with relative sparing of the tectum and cerebral peduncles, resembling the flower.
6. **Mickey Mouse sign** — axial image at midbrain level where the atrophic tegmentum and preserved cerebral peduncles produce the outline of a mouse's head and ears.
7. Supporting indirect measures: **reduced anteroposterior midbrain diameter**, **third ventricular dilatation**, and **atrophy of the superior cerebellar peduncle (SCP)** — SCP atrophy is relatively specific for PSP.

PSP — quantitative measures

8. **Midbrain-to-pons area ratio (M/P)** on midsagittal T1 is reduced in PSP; a ratio below approximately **0.52** is widely used as a threshold.
9. **Magnetic Resonance Parkinsonism Index (MRPI)** = (pons area / midbrain area) × (middle cerebellar peduncle width / superior cerebellar peduncle width). It is **increased in PSP** and has better accuracy than any single measure. **MRPI 2.0** adds the third ventricle width-to-frontal horn width ratio and improves detection of PSP-P.
10. **Important caveat:** a normal MRI does not exclude PSP. Atrophy develops gradually, so the hummingbird sign and significant midbrain atrophy may be absent early in the disease.

MSA — infratentorial signs (MSA-C)

11. **Hot cross bun sign** — axial T2 or proton-density image of the pons showing a **cruciform hyperintensity**. It results from selective degeneration of **transverse pontocerebellar fibres and pontine neurons with preservation of the corticospinal tracts** (which form the vertical limb) and the tegmentum.
12. **Atrophy of the pons, cerebellum and middle cerebellar peduncles**, best seen on midsagittal T1 — the imaging correlate of **olivopontocerebellar atrophy**.
13. **Middle cerebellar peduncle (MCP) T2 hyperintensity**, and a reduced **MCP width** and **MCP/SCP ratio**, are useful quantitative markers distinguishing MSA from PSP.

MSA — supratentorial signs (MSA-P)

14. **Putaminal rim sign** — a **hyperintense rim along the dorsolateral border of the putamen** on T2/FLAIR, thought to represent gliosis and microglial activation.
15. **Putaminal atrophy** and **putaminal T2 hypointensity** (from iron deposition), best appreciated on axial T2 and susceptibility-weighted imaging.
16. **Diagnostic performance:** these putaminal signs have **high specificity (87–100%) but widely variable sensitivity** — 38–100% for putaminal T2 hypointensity and 56–90% for the hyperintense dorsolateral rim.
17. **Increased putaminal diffusivity** on diffusion-weighted imaging (raised apparent diffusion coefficient / mean diffusivity) is directly visible and is one of the most useful practical signs for separating MSA from PD and PSP.

Comparison and cautions

18. **The essential contrast for the examiner:** in **PSP the midbrain atrophies and the pons is spared**; in **MSA the pons and cerebellum atrophy and the midbrain is relatively spared**. On the same midsagittal image, PD shows neither.
19. The hot cross bun sign is **not entirely specific to MSA** — it is also described in spinocerebellar ataxias (especially SCA-2, 3 and 7), variant CJD and some parainfectious pontine lesions, so it must be interpreted with the clinical picture.
20. Both PSP and MSA signs have **high specificity but modest sensitivity**: they confirm the diagnosis when present but never exclude it when absent. The diagnosis remains clinical, with MRI as supportive evidence.

Latest Updates (2023 – Present)

- The **MDS criteria for MSA (2022)** formally include MRI markers — putaminal atrophy, MCP atrophy, pontine atrophy, hot cross bun sign and putaminal rim sign — as supportive features for "clinically established" and "clinically probable" MSA, giving these signs formal diagnostic status for the first time.
- **Automated MRPI calculation** and machine-learning-based classifiers applied to routine T1 sequences are being

validated to reduce operator dependence and to detect PSP before the hummingbird sign appears.

- A systematic review and meta-analysis of **MR planimetric measurements** confirms that **MRPI and the M/P ratio outperform qualitative signs** in differentiating PSP from MSA (*Parkinsonism and Related Disorders*; *Cureus* 2025).
- **Susceptibility-weighted imaging (SWI)** and **quantitative susceptibility mapping** demonstrate loss of **dorsal nigral hyperintensity ("swallow-tail sign")** in all degenerative parkinsonisms including PD, and increased putaminal and nigral iron in MSA-P — adding a further layer of differentiation.
- **Neuromelanin-sensitive MRI** of the substantia nigra and locus coeruleus is emerging as a marker of nigral degeneration usable at 3T without contrast.
- **Fluid biomarkers now complement imaging:** raised NfL supports atypical parkinsonism, and CSF **α-synuclein SAA is positive in MSA but negative in PSP**, allowing a biochemical separation of synucleinopathy from tauopathy that imaging alone cannot achieve.

Current Practice Update

Aspect	Textbook position	Current evidence / practice
PSP imaging	Midbrain atrophy described qualitatively	Quantified by M/P ratio and MRPI/MRPI 2.0, with automated calculation
MSA imaging	Not described in detail	Hot cross bun and putaminal rim signs are formal supportive criteria (MDS 2022)
Sequences used	T1, T2, FLAIR	Adds DWI/ADC, SWI, quantitative susceptibility mapping, neuromelanin MRI
Role of imaging	Supportive	Supportive, and now combined with CSF α-synuclein SAA and NfL

11. Confusion States in the Elderly — Investigation and Management

Fifteen–Twenty High-Yield Points

Definition and classification

1. Confusion in an older patient is a **presentation, not a diagnosis**. Disorders causing confusion fall into three groups: **(i) acute disorders related to acute illness, drugs and environment (delirium); (ii) slowly progressive cognitive impairment (dementia); (iii) impaired cognition associated with affective disorders and psychoses**.
2. Cognitive impairment in the elderly **usually results from multiple interacting processes rather than a single cause**. Old age alone never causes dysfunctional cognitive impairment.
3. **Delirium** is an acute or subacute alteration in mental status. Prevalence is about **15% on admission** in hospitalised older patients and incidence during admission may be up to **one-third**. It may persist for days to weeks and is therefore common in post-acute care.

Diagnostic criteria

4. The key features of delirium are: **disturbance of consciousness** (reduced clarity of awareness with reduced ability to focus, sustain or shift attention) and **a change in cognition not better accounted for by dementia**, developing over hours to days and **fluctuating**, with evidence of a physical cause.
5. The **Confusion Assessment Method (CAM)** requires **features 1 and 2 plus either 3 or 4**: (1) **acute onset and fluctuating course**, (2) **inattention**, (3) **disorganised thinking**, (4) **altered level of consciousness**. Level of consciousness is graded alert, vigilant, lethargic, stupor or coma.
6. Delirium is subtyped as **hyperalert (hyperactive), hypoalert (hypoactive) or mixed**. The **hypoactive form is commoner in the elderly and is the form most often missed**.
7. The **4AT** is a rapid screening tool with **sensitivity 0.88 and specificity 0.88** in people aged 65 and over across emergency, medical, stroke and surgical settings.

The three key questions and causes

8. Three questions guide the diagnosis: **Is the onset acute? Are physical factors contributing? Are psychological factors contributing?**
9. **Common causes of delirium:** metabolic disorders (hypoxia, hypercarbia, hypo- or hyperglycaemia, hyponatraemia, azotaemia); **infections**; reduced cardiac output (dehydration, acute blood loss, myocardial infarction, heart failure); **small cortical stroke**; **drugs** and drug intoxication including alcohol; hypo- or hyperthermia; acute psychoses; **transfer to unfamiliar surroundings**, especially where sensory input is reduced; and, easily forgotten, **faecal impaction and urinary retention**.
10. Anaesthesia poses a special risk. Predisposing factors include impaired sensory function and sensory deprivation, sleep deprivation, immobilisation and transfer to an unfamiliar environment.

Investigation

11. Assessment comprises: **focused history and physical examination** (including assessment for delirium and depression and identification of comorbidities such as sensory impairment), **functional status assessment**, **mental status examination**, and **selected laboratory studies to exclude reversible causes**.
12. A **collateral history** is mandatory to establish the pre-morbid baseline and the tempo of change.
13. **Baseline investigations:** full blood count, urea and electrolytes, calcium, glucose, liver and renal function, thyroid function, vitamin B12, CRP, urinalysis and culture, chest radiograph, ECG, and pulse oximetry. Blood cultures if febrile.
14. **A full drug review is the single highest-yield investigation.** Every medication that may be worsening cognition should be stopped or avoided.
15. **Brain imaging should not be done automatically in every patient with cognitive impairment.** CT is indicated for focal neurological signs, head injury, anticoagulation, suspected subdural haematoma, or an unexplained failure to improve.
16. **Lumbar puncture** is reserved for suspected CNS infection; **EEG** for suspected non-convulsive status epilepticus, which in the elderly may present purely as confusion.

Management

17. **Treat the underlying cause** and correct every contributing factor simultaneously — hypoxia, sepsis, dehydration, pain, constipation, urinary retention, and drug toxicity.
18. **Non-pharmacological measures come first:** preparing older patients for changes in location, placing familiar objects in the surroundings, and maximising sensory input with adequate lighting, clocks and calendars. Restore spectacles and hearing aids; encourage early mobilisation; promote normal sleep-wake cycles; involve family.
19. **Avoid physical restraints** in hospitalised older patients with delirium or dementia unless essential for safety and medical care. **Avoid psychoactive drugs where they can be avoided.**
20. If drug treatment is unavoidable because of danger to self or others, use the **lowest effective dose of a single antipsychotic for the shortest possible time**, with regular review. **Benzodiazepines are reserved for alcohol or benzodiazepine withdrawal**, where they are the treatment of choice. **Melatonin or melatonin receptor agonists** reduce the prevalence of delirium and length of ICU stay and improve sleep quality.

Latest Updates (2023 – Present)

- **DSM-5-TR and ICD-11** classify delirium under neurocognitive disorders; **attention and awareness** are the core deficits, and the term "clouding of consciousness" has been retired.
- **The 4AT has largely replaced the CAM for routine screening** in acute care because it requires no special training and can be completed in under two minutes, including in patients who are drowsy or unable to cooperate. The **CAM-ICU** remains standard in intensive care.
- **ESAIC guidance (updated 2024)** recommends **against benzodiazepines** for treating postoperative delirium, suggests **low-dose haloperidol** only if non-pharmacological measures fail, and suggests **dexmedetomidine** for delirium after cardiac surgery (*European Journal of Anaesthesiology* 2024).
- Antipsychotics do not shorten delirium duration or reduce mortality; current guidance restricts them to distressing agitation or psychosis with a documented indication and a stop date.
- **Delirium is increasingly recognised as a marker of brain vulnerability**, with strong evidence that an episode accelerates long-term cognitive decline and predicts subsequent dementia — reframing delirium prevention as dementia prevention.
- **Delirium may be the first manifestation of infection** in older people — a lesson reinforced during COVID-19, where atypical presentation with delirium was common in those with multimorbidity, pre-existing cognitive impairment and polypharmacy.
- **Indian context:** delirium is markedly under-recognised in Indian hospitals; validated Hindi and regional-language versions of the 4AT and CAM now exist, and NPHCE-linked geriatric wards are the natural setting for structured screening on admission.

Current Practice Update

Aspect	Textbook position	Current evidence / practice
Diagnostic criteria	DSM-IV-TR: "disturbance of consciousness"	DSM-5-TR / ICD-11: disturbance of attention and awareness
Screening tool	CAM	4AT for rapid screening; CAM-ICU in critical care
Drug treatment	Antipsychotics if danger to self or others	Same threshold, but explicit evidence that antipsychotics do not shorten delirium; benzodiazepines advised against
Sedatives for sleep	Hypnotics and benzodiazepines used cautiously	Melatonin/ramelteon preferred; benzodiazepines cause respiratory depression and worsen delirium
Prognostic significance	Reversible acute state	Independent predictor of long-term cognitive decline and incident dementia

12. Interventions to Prevent Delirium in Post-operative Older Patients

Fifteen–Twenty High-Yield Points

Scale of the problem

1. Postoperative delirium is one of the commonest surgical complications in older people. Incidence in hospitalised older patients may reach **one-third**, and **anaesthesia poses a special risk**.
2. Prevention is far more effective than treatment. **Up to a third of episodes are preventable**, and there is growing awareness of the importance of preventing, or at least minimising, postoperative delirium.

Pre-operative risk stratification

3. Four items are independently associated with postoperative delirium: **prior stroke or TIA, Mini-Mental State Examination score, abnormal serum albumin, and Geriatric Depression Scale score**.
4. **Frailty** — assessed by unintentional weight loss, reduced grip strength, low energy and motivation, low physical activity and slow walking speed — independently predicts postoperative complications, length of stay, and discharge to a skilled or assisted living facility.
5. Other preoperative predictors of six-month mortality and poor outcome are **impaired cognition, recent falls, low albumin, greater anaemia, functional dependence and increased comorbidity**.
6. Six well-known risk factors are targeted by preventive programmes: **cognitive impairment, visual impairment, hearing impairment, sleep deprivation, immobility and dehydration**.

The evidence base — multicomponent intervention

7. The **Hospital Elder Life Program (HELP)** is the model intervention. In the landmark controlled trial, addressing the six risk factors comprehensively through a trained interdisciplinary team **reduced delirium incidence from 15% with usual care to 9.9%** (matched odds ratio 0.60, p = 0.02).
8. The methods used in each HELP protocol were **simple, commonsense measures that arguably should form part of best practice and good, empathetic care for all elderly patients**. HELP has been shown to be sustainable and scalable in community hospitals.
9. Multifactorial interventions succeed only when **supported by strong leadership and robust measurement and reporting** — the same principle that underpins successful falls reduction.

The six HELP protocols — practical content

10. **Orientation and therapeutic activities** for cognitive impairment — orientation boards, clocks, calendars, familiar objects, and daily cognitive stimulation.
11. **Early mobilisation** — ambulation or active range-of-movement exercises at least three times daily, and minimising immobilising equipment such as catheters and restraints.
12. **Non-pharmacological sleep protocol** — warm drink, relaxation, quiet environment, noise reduction and rescheduling of night-time observations and medications, instead of hypnotics.
13. **Vision protocol** — spectacles, magnifiers, adequate lighting, large-print material.
14. **Hearing protocol** — hearing aids, portable amplifiers, and **removal of cerumen**, which is a simple and frequently overlooked intervention.
15. **Volume repletion** — early recognition of dehydration and encouragement of oral fluid intake.

Peri-operative medical management

16. **Medication review is central:** the regimen should be scrutinised to determine which drugs should be continued or withheld. Every drug that may worsen cognition should be avoided or discontinued.
17. **Anaesthetic technique:** many surgeons and anaesthetists favour regional over general anaesthesia in geriatric patients, but regional anaesthesia has disadvantages — added intravenous sedation and analgesia may increase cardiovascular and mental status changes, and significant cardiovascular changes can occur during regional anaesthesia. **The choice must be individualised.**
18. **Adequate analgesia is protective**, since uncontrolled pain is itself a potent precipitant; opioid-sparing multimodal analgesia is preferred and pethidine should be avoided.
19. Other peri-operative essentials: prophylaxis against **thromboembolism and infection**, avoidance of hypoxia and hypotension, correction of anaemia and electrolyte disturbance, early removal of urinary catheters, early feeding, and treatment of constipation and urinary retention.
20. Comprehensive Geriatric Assessment identifies the frailest patients at highest risk of adverse outcomes, and when combined with strong and sustained interventions achieves better long-term outcomes.

Latest Updates (2023 – Present)

- The **2025 American Society of Anesthesiologists practice advisory for perioperative care of older adults** (*Anesthesiology* 2025;142:22–51) is now the primary reference document and consolidates preoperative cognitive screening, brain health optimisation and delirium prevention into a single pathway.
- **Updated ESAIC guideline on postoperative delirium (2024)**: suggests **multiparameter intraoperative EEG monitoring** (burst suppression and density spectral array) to reduce delirium risk; **recommends against benzodiazepines** for treatment; suggests **low-dose haloperidol** only if non-pharmacological measures fail; and suggests **dexmedetomidine** for delirium after cardiac surgery.

- **EEG-guided anaesthesia:** evidence is genuinely mixed. ENGAGES (*JAMA* 2019) and ENGAGES-Canada (*JAMA* 2024) were negative, yet a 2025 meta-analysis of 12 trials and 7,441 patients (*Frontiers in Neurology* 2025) found benefit. The consistent message is that **avoiding intraoperative burst suppression** reduces delirium.
- **Dexmedetomidine** has the strongest pharmacological evidence: risk ratio approximately **0.57 in cardiac surgery**, **0.48 in older patients under regional anaesthesia**, and **0.51 in older non-cardiac surgical patients** in pooled analyses. Bradycardia and hypotension are the limiting adverse effects.
- **Melatonin** trials remain inconclusive; it is low-risk and widely used, but should not displace the sleep protocol.
- **Haemodynamic management:** the **cogPOISE-3** trial (*Annals of Internal Medicine* 2025) compared hypotension-avoidance with hypertension-avoidance strategies; observational data continue to link **intraoperative hypotension and blood pressure variability** with delirium.
- The 2025 APSF advisory concluded that evidence **does not support avoiding single-dose short-acting midazolam** in older adults — a nuanced revision of blanket benzodiazepine avoidance, though repeated or long-acting benzodiazepines remain contraindicated.
- **Cochrane (2021, current)**: non-pharmacological multicomponent interventions remain the only intervention with consistent evidence for preventing delirium in hospitalised non-ICU patients — the HELP model is still the backbone.

Current Practice Update

Aspect	Textbook position	Current evidence / practice
Core strategy	Multicomponent non-pharmacological (HELP)	Unchanged — still the only consistently effective approach
Anaesthetic depth	Not addressed	Intraoperative EEG monitoring to avoid burst suppression (weak recommendation)
Drug prophylaxis	None recommended	Dexmedetomidine has moderate evidence, particularly in cardiac surgery
Benzodiazepines	Cautioned against	Contraindicated for treatment; single-dose short-acting midazolam no longer prohibited outright
Preoperative assessment	Risk factor identification, frailty screening	Formal ASA 2025 pathway with routine cognitive screening and brain health optimisation

13. Thrombolysis in Stroke

Fifteen–Twenty High-Yield Points

Principle

1. **The introduction of intravenous alteplase (rt-PA) transformed acute stroke care.** Its benefit is tempered by the risk of precipitating intracranial haemorrhage and by strict licensing conditions.
2. The rationale is the **ischaemic penumbra** — an under-perfused but potentially salvageable area of brain surrounding the infarct core. The higher the residual perfusion, the longer ischaemia is tolerated before electrical failure, ion pump failure and cell death.

Evidence base

3. **NINDS rt-PA Stroke Study:** patients under 80 with carotid or vertebrobasilar territory stroke, not comatose, treated **within 3 hours**. Intracranial haemorrhage occurred in **6.4% of thrombolysed patients within 36 hours versus 0.6% with placebo**, but treated patients were **about 30% more likely to have minimal or no disability at three months**.
4. **ECASS-3** confirmed that alteplase can be given safely to patients under 80 **between 3 and 4.5 hours** after onset, with a small overall benefit.
5. **IST-3** included patients **over 80**. Pooled analysis with NINDS and other trials confirmed that intravenous thrombolysis **significantly reduces death or dependency**, with a **linear relationship between time to treatment and benefit, extending up to six hours** from onset. Age alone is not a contraindication.

Contraindications

6. **Absolute:** haemorrhage on pre-treatment imaging; suspected subarachnoid haemorrhage; any history of intracranial haemorrhage; known arteriovenous malformation or aneurysm; active bleeding from any site.
7. **Imaging-based:** visible infarction involving **more than one-third of the MCA territory** — a clearly delineated hypodensity indicates established infarction with blood-brain barrier breakdown and a high haemorrhage risk.
8. **Haematological:** platelet count **below 100 × 10^9/L**; recent heparin with raised APTT; recent warfarin with elevated INR.
9. **Recent events:** major surgery or trauma within 14 days; gastrointestinal or urinary tract haemorrhage within 21 days; neurosurgery, serious head trauma or previous stroke within 3 months; recent myocardial infarction with pericarditis; recent arterial puncture at a non-compressible site; recent lumbar puncture.
10. **Physiological:** blood pressure persistently above **185 mmHg systolic or 110 mmHg diastolic**; blood glucose below 3 or above 20 mmol/L (roughly **<55 or >360 mg/dL**); active pancreatitis; suspected or known pregnancy.
11. **Clinical:** symptoms only minor or rapidly improving; **epileptic seizure at stroke onset**.

Delivery and complications

12. Hypertension above 180/110 mmHg should be **treated cautiously before** giving thrombolysis. **Aspirin is contraindicated within the first 24 hours after rt-PA** because of an increased risk of death.
13. If intracranial haemorrhage occurs, **stopping the ongoing infusion is mandatory**. Send an urgent clotting profile, consider reversal agents such as cryoprecipitate, and obtain neurosurgical consultation for severe haemorrhage.
14. **Service requirements:** thrombolysis requires **hyperacute stroke units (HASUs)** with pathways giving urgent access to brain imaging and stroke expertise, aiming for a **door-to-needle time under 30 minutes**, and facilities for appropriate post-thrombolysis monitoring.
15. **Imaging:** non-contrast CT is the initial investigation; early signs are a hyperdense artery, loss of the insular ribbon and lentiform nucleus definition, sulcal effacement and mass effect. **CT is normal in 30–40% of patients in the first few hours**; **DWI-MRI becomes abnormal within minutes** and is far more sensitive.
16. **CT angiography** identifies intracranial occlusion, and **CT perfusion** assesses the extent of hypoperfusion, refining selection for thrombolysis or thrombectomy, particularly beyond six hours.

Thrombolysis and thrombectomy

17. **Mechanical thrombectomy** improves functional outcome, increasing the proportion functionally independent at 90 days by around **20%**. It has advantages over thrombolysis: greater efficacy in large-artery occlusion, a longer window, and usability in some patients with contraindications to thrombolysis.
18. Thrombectomy is indicated for **proximal anterior circulation occlusion (intracranial carotid, M1 or proximal M2)**, NIHSS above 5, pre-morbid modified Rankin score below 3, and no major established ischaemic change — within **6 hours**, or up to **12–24 hours** where advanced imaging shows substantial salvageable tissue.
19. Benefit declines with time: independence at three months falls from **50% at 3 hours to 45% at 4.5 hours, 40% at 6 hours and 33% by 8 hours**. The '**drip and ship**' model — thrombolyse locally, then transfer — is one of two accepted service configurations.
20. **Neuroprotective drugs have consistently failed** in clinical trials. Tight control of physiological parameters — oxygenation, glucose, temperature and blood pressure — remains the only effective way to limit secondary damage.

Latest Updates (2023 – Present)

- **The 2026 AHA/ASA Guideline for the Early Management of Patients with Acute Ischemic Stroke** is the definitive current reference and contains three practice-changing changes.

- **Tenecteplase is now co-equal first-line with alteplase.** New **Class 1, Level A** recommendation: **tenecteplase 0.25 mg/kg (maximum 25 mg) as a single 5-second bolus, or alteplase 0.9 mg/kg (maximum 90 mg)**, within 4.5 hours. The higher **0.4 mg/kg dose is explicitly NOT recommended (Class 3, No Benefit)** because it increases symptomatic intracranial haemorrhage without added benefit. Supporting trials: **AcT, ATTEST-2, TASTE, ORIGINAL, NOR-TEST 2**.
- Tenecteplase's practical advantages are decisive in a resource-constrained system: **a single bolus rather than a one-hour infusion**, shorter door-to-needle times, and much easier interhospital transfer — directly relevant to Indian stroke networks and to India's own cost-effective tenecteplase supply.
- **Extended window thrombolysis is now endorsed. Class 2a**: IVT is reasonable between **4.5 and 9 hours** from onset, or in wake-up stroke within 9 hours of the sleep midpoint, when advanced imaging shows salvageable penumbra (**EXTEND**, **WAKE-UP** using DWI-FLAIR mismatch). **Class 2b**: thrombolysis may be beneficial **4.5 to 24 hours** after onset in selected patients with large-vessel occlusion and salvageable tissue who cannot undergo thrombectomy (**TRACE-III**, **HOPE**, **CHABLIS-T II**). **TIMELESS** was negative in patients who did proceed to thrombectomy.
- Thrombolysis should now be given **rapidly to all eligible patients with disabling deficits regardless of NIHSS score**, within 4.5 hours, **without waiting for advanced imaging**.
- **Basilar artery occlusion** now carries a strong recommendation for endovascular therapy within **24 hours** when NIHSS is 10 or more — a change from the textbook position that this indication was untested in randomised trials.
- Perfusion-imaging-guided selection has substantially widened eligibility, but access to advanced imaging is not universal — a real constraint in most Indian district hospitals, where non-contrast CT within 4.5 hours remains the practical pathway.

Current Practice Update

Aspect	Textbook position	Current evidence / practice
Thrombolytic agent	Alteplase (rt-PA) only	Tenecteplase 0.25 mg/kg bolus or alteplase, both Class 1 Level A
Time window	Up to 4.5 hours (benefit to 6 hours on pooled analysis)	4.5 h standard; 4.5–9 h with imaging selection; up to 24 h in selected LVO not for thrombectomy
Minor stroke	Minor or rapidly improving deficit is a contraindication	Treat all disabling deficits regardless of NIHSS score
Thrombectomy window	6 hours, extended to 24 h with perfusion mismatch	Same, plus strong recommendation for basilar occlusion to 24 h
Administration	Bolus plus 60-minute infusion	Single 5-second bolus with tenecteplase, easing transfer and reducing door-to-needle time

14. History of the 'Stroke Clinics' — Why They Were Set Up and What Issues They Addressed

Fifteen–Twenty High-Yield Points

The problem that had to be solved

1. Before organised services, stroke patients were admitted to **general medical wards** with no specialist input, no agreed protocols, and outcomes that varied widely between hospitals. Stroke was regarded as an untreatable consequence of ageing.
2. The **conceptual rationale for organised stroke care** is that stroke affects several domains of human performance and produces multiple impairments that interact to determine disability — and that **no single discipline has all the skills, resources and expertise required** to manage recovery.
3. Recovery is further complicated because **different impairments recover at different speeds**, demanding a staged approach to intervention and therapy input.
4. Rehabilitation goals are also shaped by the **personal needs of the patient, the environment they will return to, and the support available after discharge** — requiring coordination that ad hoc care could not deliver.

Development and the evidence

5. Over the last two decades of the twentieth century, numerous randomised controlled trials suggested that organised care benefited stroke patients, but **most individual studies were too small to demonstrate a robust statistical benefit**.
6. The **Stroke Unit Trialists' Collaboration (SUTC)** was therefore set up to pool data from these and other ongoing studies in Australia, North America and Europe — a landmark exercise in evidence synthesis.
7. **The pooled meta-analysis of 29 trials and 6,536 patients** showed, at one year, odds reductions with organised care of **0.86 for mortality (95% CI 0.71–0.94)**, **0.78 for death or dependence (0.68–0.89)**, and **0.80 for death or institutionalisation (0.71–0.90)**, **independent of age and gender**.
8. **Crucially, and in contrast with thrombolysis, these benefits are seen in all stroke patients regardless of stroke aetiology or the delay between onset and intervention** — which is why stroke unit care, not thrombolysis, is the intervention that benefits the largest number of patients.
9. This efficacy translated into real-world effectiveness, confirmed in longitudinal studies and repeated national stroke audits.

Which model of organisation works

10. "Organised stroke care" means different things to different people — definitions range from **a team of specialists who consult wherever the patient is**, to **a designated geographical location within the hospital** for patients needing medical, rehabilitation and skilled professional care.
11. Langhorne's analysis showed a definite benefit with **comprehensive stroke units, rehabilitation stroke units and mixed rehabilitation units** (odds ratios **0.85–0.89** in favour of organised care).
12. There was a **possible benefit with acute semi-intensive units (OR 0.88; 95% CI 0.76–1.01)**, which just failed to reach statistical significance.
13. **Mobile stroke teams were associated with no benefit (OR 0.98; 95% CI 0.95–1.05)** — the key message that a **geographically defined unit, not a peripatetic team, is what works**.
14. There are no trials of acute intensive care for stroke. A review of the data suggests that **emphasis on acute intensive care alone is not adequate to change outcomes, and that continuity of care is needed to realise the full potential of stroke unit care**.

Issues that the clinics addressed

15. **Access to hyperacute treatment: hyperacute stroke units (HASUs)** were created to deliver urgent access to brain imaging and stroke expertise, with a target **door-to-needle time under 30 minutes** and safe post-thrombolysis monitoring.
16. **Secondary prevention after TIA:** patients with suspected TIA should receive **aspirin 300 mg immediately and be assessed within 24 hours** by a specialist in a neurovascular clinic or acute stroke unit. **Daily rapid-access TIA clinics are therefore an essential component of a comprehensive stroke service.**
17. **Prevention and early treatment of complications:** dysphagia and aspiration, malnutrition, dehydration, fever and infection, venous thromboembolism, pressure sores, incontinence, shoulder pain, depression and post-stroke fatigue.
18. **The intensity and quality of rehabilitation:** audit showed that even in stroke units patients spend **more than 50% of their time in bed, 28% sitting out of bed, only 13% in therapeutic activities, and are alone 60% of the therapeutic day**.
19. The **CERISE study** confirmed that motor recovery varied significantly across Europe and was determined **not by the therapy resources allocated but by the amount of treatment actually delivered**. In the UK, the greatest resources (70 hours per week) produced the least therapy input per patient (1 hour per day), while structured, strictly timed therapy in Germany produced the best outcomes in Europe.
20. **Consensus position:** well-organised, well-planned rehabilitation guided by defined goals, based on adequate assessment, negotiated with patients and carers, and delivered on a specialist unit, **reduces disability and long-term institutionalisation** — although there is little evidence favouring any specific treatment technique, so a pragmatic, individualised functional approach is recommended.

Latest Updates (2023 – Present)

- **Centralisation is the modern successor to the stroke unit.** Analysis of the reorganisation of acute stroke services in English metropolitan areas showed sustained reductions in mortality and length of stay when services were concentrated into fewer HASUs (*BMJ* 2019, and subsequent sustainability analyses).
- The **2026 AHA/ASA acute stroke guideline** extends organised care to prehospital systems, formalises transfer protocols for thrombectomy, and for the first time includes recommendations for interventional treatment in **paediatric** stroke.
- **Mobile stroke units** (CT-equipped ambulances) — conceptually the opposite of the failed "mobile stroke team" — have shown reduced time to thrombolysis and improved functional outcomes in the BEST-MSU and B_PROUD studies, though cost-effectiveness in most systems is unproven.
- **Telestroke** networks now extend thrombolysis decision-making to hospitals without on-site stroke expertise, and are the single most scalable solution for large countries with uneven specialist distribution.
- **Indian context:** the **Indian Stroke Association** and the **National Programme for Prevention and Control of Non-Communicable Diseases** have promoted a tiered model — Level 1 (comprehensive, with thrombectomy), Level 2 (thrombolysis-capable), Level 3 (stroke-ready with telemedicine support). Studies from Ludhiana, Trivandrum and Hyderabad have shown that **low-cost stroke units without intensive care facilities still reduce mortality and dependency**, which is the single most important message for Indian practice.
- **Early supported discharge** teams and structured caregiver training programmes (Kalra et al., *BMJ* 2004) remain the highest-value additions to stroke unit care in resource-limited settings, where family caregivers carry most of the burden.

Current Practice Update

Aspect	Textbook position	Current evidence / practice
Model of organisation	Geographically defined stroke unit within each hospital	Regional centralisation into fewer high-volume HASUs with formal transfer networks
Mobile services	Mobile stroke teams show no benefit	Still true for in-hospital teams, but CT-equipped mobile stroke ambulances speed thrombolysis
Remote expertise	Not addressed	Telestroke is standard for spoke hospitals
TIA clinics	Assessment within 24 hours	Unchanged, with immediate dual antiplatelet therapy in high-risk minor stroke/TIA
Applicability	Developed-country service models	Low-cost stroke units validated in Indian settings; tiered stroke-ready hospital model

15. Restless Legs Syndrome — Risk Factors, Diagnostic Criteria and Management

Fifteen–Twenty High-Yield Points

Definition and epidemiology

1. **Restless legs syndrome (RLS, Willis-Ekbom disease)** is a sensorimotor disorder characterised by **motor restlessness brought on by rest**, usually or only present in the evening or early night, with an urge to move typically accompanied by an unpleasant sensation in the legs.
2. **Periodic limb movements (PLMs)** are discrete, repetitive, stereotyped movements — rhythmic extension of the big toe and ankle dorsiflexion, sometimes with knee and hip flexion — each lasting **0.5–5 seconds and recurring every 20–40 seconds**. They are present in **90% of RLS patients** but can occur independently.
3. Prevalence varies geographically: **10–20% in northern Europe, 3–10% in southern Europe, 0.7–12.1% in Asia, and under 1% in Africa**. In India, prevalence is at the lower end of the Asian range but rises sharply in dialysis populations.
4. RLS is **more common in women**, but the prevalence in nulliparous women equals that in men — the difference is driven by pregnancy, during which prevalence rises to about **21%**.
5. RLS commonly causes **insomnia and excessive daytime sleepiness**, and in older people this translates into falls, cognitive complaints and depression.

Risk factors

6. **Genetic:** about **60% of patients have a positive family history**; at least **eight major susceptibility loci** are known, mostly autosomal dominant.
7. **The three most commonly associated conditions are end-stage renal disease, pregnancy and iron deficiency anaemia** (present in 25–35% of affected patients).
8. Other associations: stroke and migraine, mood disorders, cardiovascular disease, thyroid disorders and diabetes. **Prevalence increases significantly when three or more comorbidities coexist.**
9. **Drug-induced/aggravated RLS:** antidepressants, antihistamines and dopamine antagonists (including antipsychotics and metoclopramide); also withdrawal from opioids.

Pathophysiology

10. **Brain iron deficiency** is central: patients have **lower CSF ferritin, higher transferrin, and reduced iron concentration in the basal ganglia and thalamus**, even when peripheral iron stores appear adequate.
11. **Dopamine dysfunction:** there is a **presynaptic hyperdopaminergic state with circadian fluctuation and downregulation of postsynaptic dopamine receptors**. Dopamine production falls towards nightfall, producing a functional hypodopaminergic state — which explains both the circadian pattern and the response to dopaminergic drugs.
12. Brain iron deficiency and dopaminergic dysfunction interact by **altering the glutamate and adenosine systems**. **Endogenous opioid deficiency** in the central sensory system also contributes, causing abnormal processing of noxious stimuli and the characteristic urge to move.

Diagnostic criteria (IRLSSG 2014)

13. RLS is a **clinical diagnosis** requiring **all five** criteria: (i) **an urge to move the legs**, usually accompanied or caused by uncomfortable, unpleasant sensations; (ii) the urge **begins or worsens during rest or inactivity**; (iii) the urge is **relieved by movement**, such as walking or stretching; (iv) the urge is **worse in the evening or at night**; (v) **other conditions that mimic RLS have been excluded**.
14. **Supportive features:** positive family history, positive response to dopaminergic therapy, and the presence of periodic limb movements during sleep or wakefulness.
15. **Mimics to exclude:** akathisia, positional discomfort, arthralgia, myalgia, peripheral venous insufficiency, painful peripheral neuropathy, nocturnal leg cramps and lumbar radiculopathy.
16. **Investigations: iron studies must be obtained in every patient** in whom RLS is suspected or diagnosed; renal function tests if uraemia is a concern. **Polysomnography is not necessary** but can objectify severity. A high PLMS rate on polysomnography is **not sufficiently specific**, as it occurs in many conditions and increases with age. Severity is quantified with the **International RLS Severity Scale (IRLS)**.

Management

17. **First step: review the medication list** and stop or reduce any drug that causes or exacerbates RLS. Non-pharmacological measures — sleep hygiene, hot and cold baths, lower limb massage, moderate exercise, avoidance of caffeine and alcohol — should be optimised before drugs.
18. **Iron replacement:** if deficient, give oral iron with vitamin C, or intravenous iron with a carbohydrate bond, especially **ferric carboxymaltose**. **High-molecular-weight iron dextran must not be used** because of severe anaphylactic reactions. Ferritin **below 20 ng/mL** warrants oral supplementation; between **20 and 50 ng/mL** the decision is individualised; some evidence supports raising the threshold to **70 ng/mL in elderly patients with chronic renal failure or pro-inflammatory conditions**. **If iron studies are strictly normal, iron therapy is not effective.**
19. **Pharmacological options:** dopamine agonists and levodopa are approved and widely used; **only ropinirole has been shown to improve sleep efficacy on polysomnography**. **α2δ ligands (gabapentin, pregabalin) can be used as first-line agents.** Second-line agents for severe refractory disease are opioids (oxycodone-naloxone, methadone), limited by side effects. Treatment starts as monotherapy at the lowest dose and is titrated slowly.

20. **Augmentation** is the feared complication of dopaminergic therapy — **iatrogenic worsening with symptoms emerging progressively earlier in the day**. An estimated **76% of patients on dopaminergic drugs need a dose increase or develop augmentation**. To minimise risk: use long-acting agonists at the lowest effective dose, start only when quality of life is clearly affected, and **never use carbidopa/levodopa as the initial agent because of its short half-life**. Cross-titration to a different class is needed if augmentation develops. Consequently, **α2δ ligands are increasingly favoured as initial therapy.**

Latest Updates (2023 – Present)

- **The AASM Clinical Practice Guideline (Winkelman et al., *Journal of Clinical Sleep Medicine* 2025;21:137–152) is a complete reversal of the 2012 practice parameter and is the single most important update in this topic.**
- **Dopamine agonists are now conditionally recommended AGAINST for standard use** — ropinirole, pramipexole, rotigotine and levodopa. The reason is **augmentation, occurring in 7–10% of patients per year**; over three years this affects 20–30% of patients, and over five years 35–50%. Long-term dopaminergic plasticity worsens the underlying disease even though short-term efficacy is excellent.
- **α2δ ligands are now the strong first-line recommendation: gabapentin enacarbil, gabapentin and pregabalin**. Gabapentin enacarbil is not marketed in India or the UK, so **gabapentin or pregabalin are the practical first-line choices**.
- **Iron therapy has been elevated to a strong recommendation. Intravenous ferric carboxymaltose is strongly recommended when serum ferritin is below 100 µg/L** — far above the usual threshold for "low ferritin" — based on five randomised trials. Target **ferritin above 100 µg/L and transferrin saturation above 20%**. Iron studies must be taken **in the morning after a 24-hour avoidance of iron-containing food and supplements**, and should include ferritin, iron and total iron binding capacity.
- **New conditional recommendations: dipyridamole** (an adenosine reuptake inhibitor, addressing the adenosine hypothesis), **extended-release oxycodone and other mu-opioid agonists**, and **bilateral high-frequency peroneal nerve stimulation** — a non-pharmacological device option that is entirely new.
- Where a patient is already established on a dopamine agonist and doing well, guidance is to **monitor regularly for augmentation and impulse-control disorders** rather than to withdraw reflexively; low-dose opioids are often used to facilitate tapering off dopamine agonists.
- **Indian relevance:** RLS is strongly associated with chronic kidney disease and iron deficiency, both highly prevalent in India. In dialysis patients and in iron-deficient older adults, **correcting iron status is both the cheapest and the most effective intervention**, and is consistent with national anaemia control priorities.

Current Practice Update

Aspect	Textbook position	Current evidence / practice (AASM 2025)
First-line drug	Dopamine agonists approved and widely used; α2δ ligands an alternative	α2δ ligands are first-line; dopamine agonists recommended against for standard use
Levodopa	Used less commonly; never as initial agent	Explicitly recommended against
Iron threshold	Oral iron if ferritin <20 ng/mL; individualise 20–50 ng/mL	IV ferric carboxymaltose strongly recommended if ferritin <100 µg/L; target >100 µg/L and TSAT >20%
Augmentation risk	Recognised; minimised by using long-acting agonists	Quantified at 7–10% per year and considered sufficient reason to abandon the class
Non-drug options	Sleep hygiene, baths, massage	Adds bilateral high-frequency peroneal nerve stimulation and dipyridamole

16. Myasthenia Gravis — Presentation, Laboratory Evaluation and Treatment

Fifteen–Twenty High-Yield Points

Definition and epidemiology

1. **Myasthenia gravis (MG)** is an antibody-mediated disorder of neuromuscular transmission. The commonest target antigen is the **postsynaptic muscle nicotinic acetylcholine receptor (AChR)**; these structures are vulnerable because they lie outside the blood-nerve barrier.
2. Overall incidence is **2–10 per 100,000**. It affects all races and can begin at any age after the first year of life. **Incidence rises with age but MG is underdiagnosed in the elderly.**
3. **Late-onset MG (LOMG)** is increasingly recognised; reports of rising incidence may reflect improved diagnosis rather than a true biological change.

Clinical presentation

4. The hallmark is **fatigable weakness** — weakness that worsens with sustained or repeated activity and improves with rest, with characteristic diurnal variation (worse in the evening).
5. **Ocular presentation** is commonest: **ptosis and diplopia**, often asymmetrical, with **Cogan's lid twitch**. MG can mimic blepharospasm.
6. **Bulbar features:** dysarthria (nasal speech that fatigues), dysphagia, nasal regurgitation, jaw fatigue on chewing, and facial weakness with a "myasthenic snarl".
7. **Generalised disease:** proximal limb weakness, **dropped head syndrome**, and respiratory muscle weakness. Unusual presentations include **distal MG** and the **"myasthenic hand"**.
8. **In older patients, MG may present with recurrent falls** — a presentation of major importance in geriatric practice and easily attributed to other causes.
9. Cardiac involvement and cognitive impairment have both been described in MG, and determinants of cognitive impairment in elderly myasthenic patients have been specifically studied.
10. **MG is a treatable disease** — more than 90% of patients return to normal function — so the diagnosis **must be considered in anyone presenting with ptosis, diplopia, dysphagia, or limb or respiratory muscle weakness**.

Laboratory evaluation

11. **Serology: anti-AChR antibodies** are the primary test and are positive in the majority; **late-onset MG is characteristically anti-AChR seropositive**. **Anti-MuSK** (muscle-specific tyrosine kinase) antibodies define a subgroup with predominant facial and bulbar involvement; anti-LRP4 accounts for a smaller fraction.
12. **Neurophysiology: repetitive nerve stimulation** showing a decremental response, and **single-fibre EMG** demonstrating increased jitter, which is the most sensitive test.
13. **Bedside tests:** the **ice pack test** for ptosis (cooling improves neuromuscular transmission) and the **sleep/rest test** — a safe alternative to the edrophonium (Tensilon) test, which is now rarely used because of cardiac risk in older patients.
14. **CT of the thorax is mandatory** in every new diagnosis to look for **thymoma** or thymic hyperplasia. **Paraneoplastic MG** is recognised, including with carcinoid tumour.
15. Additional baseline work: **thyroid function and screening for associated autoimmune disease**; **forced vital capacity** as the objective measure of respiratory reserve; and review of drugs that unmask or worsen MG (aminoglycosides, fluoroquinolones, macrolides, beta-blockers, magnesium, **penicillamine**).

Treatment

16. **Anticholinesterase inhibitors** remain first-line for all patients, acting by inhibiting acetylcholine breakdown at the NMJ. **Pyridostigmine** is started at **30 mg two to four times daily** and titrated. It acts in **10–30 minutes, peaks at two hours, and lasts about four hours**. Muscarinic side effects (abdominal cramps, diarrhoea, sweating, bronchial and oral secretions) can be treated with propantheline, which may however mask overdose; nicotinic effects are fasciculation and cramps. **Chronic high doses may increase weakness by downregulating AChRs.**
17. **Steroids** are short-term immunosuppressives used while other agents take effect. A **"steroid dip"** — temporary worsening **4–10 days** after starting high-dose steroids — is avoided by starting low and going slow, or by alternate-day treatment. **In elderly patients, steroids and immunosuppressives carry greater risk, notably steroid-induced osteoporosis, so prophylaxis is required.**
18. **Steroid-sparing agents:** azathioprine, mycophenolate mofetil, methotrexate, ciclosporin and tacrolimus. **Rituximab** (B-cell depletion) is particularly effective in **MuSK-associated MG**.
19. **Rapid therapies: plasma exchange (PE)** and **intravenous immunoglobulin (IVIG)**. IVIG is given as **0.4 g/kg/day for five days**, improves most patients within four to five days, and lasts weeks to months. Adverse effects of IVIG (fever, headache, nausea, allergic reaction; anaphylaxis in IgA deficiency; volume overload; renal failure; thrombosis and stroke with high infusion rates) are **less severe than those of PE**, whose complications relate mainly to vascular access, fluid shifts, and citrate-induced hypocalcaemia.
20. **Thymectomy** is the treatment of choice for thymoma (radiotherapy if unfit). In non-thymoma patients it is usually performed in generalised, AChR-positive MG **under the age of 50–60**. The randomised trial of transsternal thymectomy plus alternate-day steroids versus steroids alone showed **improved clinical outcomes over three years**. Benefit may take **6–12 months** to appear; predictors of good response are female sex, limb-predominant weakness and AChR antibody positivity.
21. **Crises: myasthenic crisis** (weakness requiring ventilation) is precipitated by infection, intercurrent illness, surgery or

too-rapid reduction of immunotherapy; **cholinergic crisis** results from excessive anticholinesterase. If the two cannot be distinguished, it is **safe to stop anticholinesterases once the patient is ventilated**. Treat infection aggressively and give IVIG or PE. For anaesthesia, prefer local or spinal; if general anaesthesia is needed, insert a nasotracheal tube, keep it for one to two days, avoid anticholinesterases on the morning of surgery and for 24 hours afterwards, and continue immunosuppressants.

Latest Updates (2023 – Present)

- **The treatment landscape has been transformed by three targeted mechanisms — complement inhibition, FcRn blockade and B-cell depletion.**
- **Complement (C5) inhibitors: eculizumab** (2017, REGAIN), **ravulizumab** (extended 8-weekly dosing) and **zilucoplan** (RAISE, 2023) — a cyclic peptide that is the first **self-administered subcutaneous** option. **Meningococcal vaccination is mandatory before starting any C5 inhibitor.**
- **FcRn antagonists** block neonatal Fc receptor-mediated IgG recycling, lowering pathogenic IgG: **efgartigimod** (IV and subcutaneous Vyvgart Hytrulo), **rozanolixizumab** (Rystiggo, 2023 — approved for both AChR- and MuSK-positive disease), and **nipocalimab (Imaavy)**, approved **30 April 2025** for AChR- or MuSK-positive gMG in patients aged 12 and above, with sustained disease control across the broadest population.
- **Inebilizumab (Uplizna)**, an anti-CD19 B-cell depleting antibody, was approved in **late 2025** for AChR- and MuSK-positive gMG, offering long-term control with only **two maintenance doses per year** after loading.
- **CAR-T cell therapy** targeting BCMA and IL-6 receptor blockade are in early trials, raising the prospect of drug-free remission (*Journal of Neurology* 2025;272:226).
- The **2020 International Consensus Guidance** and updated national guidelines (German 2023) integrate these agents for **refractory or highly active disease**, positioning them after failure of conventional immunosuppression rather than as first-line.
- **Geriatric relevance:** reviews now specifically highlight the growing cohort of **older patients with late-onset MG**. Targeted biologics are attractive in this group because they avoid the osteoporosis, diabetes, infection and cognitive effects of long-term steroids — but cost is prohibitive in most Indian settings, where pyridostigmine, prednisolone, azathioprine, IVIG and plasma exchange remain the working armamentarium.

Current Practice Update

Aspect	Textbook position	Current evidence / practice
Biologic therapy	Rituximab for MuSK MG; eculizumab and belimumab at early evaluation	Five-plus approved targeted agents across three mechanisms; eculizumab, ravulizumab, zilucoplan, efgartigimod, rozanolixizumab, nipocalimab, inebilizumab
Route of administration	Intravenous, hospital-based	Subcutaneous and self-administered options; twice-yearly dosing with inebilizumab
Position in algorithm	Steroids and azathioprine, then IVIG/PE	Same, with targeted agents for refractory or highly active disease and increasing use to spare steroids in the elderly
Thymectomy	Generalised AChR-positive MG under age 50–60	Unchanged; MGTX trial evidence confirmed at long-term follow-up
Late-onset MG	Noted to be underdiagnosed	Recognised as a distinct, expanding phenotype requiring steroid-sparing strategies

17. Motor Neuron Disease

Fifteen–Twenty High-Yield Points

Definition and classification

1. **Motor neuron diseases (MNDs)** are a heterogeneous group characterised by degeneration or dysfunction of the **upper motor neurons (UMNs) of the precentral gyrus** and/or the **lower motor neurons (LMNs) of the ventral horn**.
2. **Amyotrophic lateral sclerosis (ALS)** is the commonest MND in adults. It is best regarded as a **syndrome** with considerable phenotypic heterogeneity — sporadic and genetic, pure motor and with extra-motor signs, and forms affecting only LMN or only UMN.
3. Related entities include **primary lateral sclerosis (PLS)** (pure UMN), **progressive muscular atrophy (PMA)** (pure LMN) and **progressive bulbar palsy**. Although histopathologically similar, they must be separated because **prognosis differs**.

Clinical presentation

4. Motor symptoms are frequently preceded by months of non-specific features: **fatigue, weight loss secondary to hypermetabolism, cramps and diffuse fasciculation** from LMN loss and hyperexcitability.
5. **Limb-onset ALS** (commonest): difficulty with fine hand movements, raising the arms, or walking.
6. **Bulbar-onset ALS** (about one-third): swallowing and phonation disorders with sialorrhoea. Rarely, **trunk-onset** with isolated restrictive respiratory impairment causing exertional dyspnoea and orthopnoea with unexplained weight loss.
7. **LMN signs:** fasciculation, amyotrophy, weakness and cramps. **UMN signs:** pyramidal signs with spastic hypertonia, stiffness, clonus and gait disorder. At bulbar level, **pseudobulbar** features are dysarthria and dysphagia from hypertonia without atrophy, frequently with **pseudobulbar affect** (inappropriate crying and laughing).
8. **Oculomotor nuclei and Onuf's nucleus are spared** until very advanced disease — hence eye movements and sphincter function are preserved, a key diagnostic point.
9. Progression follows a pattern consistent with **cell-to-cell, prion-like spread**, extending rostrocaudally or contralaterally. The insidious, non-specific onset explains a **diagnostic delay frequently exceeding one year**.

Cognitive involvement

10. Extra-motor involvement is frequent: with appropriate routine tests, **cognitive or behavioural changes are detected in about 50% of patients, and 5–15% have frank frontotemporal dementia (FTD)**.
11. The cognitive profile is dominated by **executive dysfunction and reduced verbal fluency**, with impaired social cognition. **Memory and visuoconstructive disorders are uncommon and non-specific.**
12. Neuropsychological impairment matters clinically: it is associated with **lower quality of life, greater caregiver burden, altered capacity to engage in life-prolonging interventions, and reduced survival**. The revised **Strong criteria** classify this on three axes: Axis I motor neuron disease variant, Axis II cognitive and behavioural dysfunction, Axis III additional non-motor manifestations.

Pathophysiology and genetics

13. **TDP-43** is central: except for SOD1- and FUS-linked familial ALS (under 3% of cases), the neuropathological hallmark is **cytoplasmic inclusions of ubiquitinated, hyperphosphorylated TDP-43**. Nuclear depletion causes aberrant RNA metabolism and impaired chromatin remodelling; cytoplasmic accumulation disturbs autophagy, proteasome function and mitochondrial function, and the aggregates show **seeding and prion-like cell-to-cell transmission**.
14. Common mechanisms are therefore: **disrupted RNA metabolism, aberrant protein quality control, mitochondrial dysfunction, oxidative stress and trafficking abnormalities**.
15. **Genetics:** up to **20% of European ALS patients have a family history** of ALS or FTD. About **10% of apparently sporadic cases carry a known familial ALS mutation**, and first-degree relatives of a sporadic patient have roughly an **eight-fold** increased risk.
16. **Four genes account for up to 70% of familial ALS: C9orf72, SOD1, FUS and TARDBP.** A **GGGGCC hexanucleotide repeat expansion in C9orf72** is found in nearly **40% of familial ALS**, about 25% of familial FTD-ALS, and about 5% of sporadic ALS. C9orf72 is pleiotropic, also causing FTD, PLS, PMA, Huntington phenocopies, parkinsonism and corticobasal degeneration.
17. **Environmental risk factors:** exposure to lead and heavy metals (with PON1 polymorphisms as a susceptibility factor), pesticides, and sports-related trauma. Relative risk is **4.07 for professional sport versus 1.13 for non-professional**, **5.98 for sports with repetitive spinal and head trauma**, and **8.52 when both risks combine**.

Diagnosis and treatment

18. In the **absence of positive diagnostic markers**, diagnosis rests on precise clinical and electrophysiological analysis, observation of the course, and exclusion of **ALS mimics** — cervical spondylotic myelopathy, multifocal motor neuropathy with conduction block, inclusion body myositis, myasthenia gravis, Kennedy's disease and thyrotoxic myopathy.
19. **Riluzole**, approved in 1995, was developed to reduce excitotoxicity by inhibiting glutamatergic transmission and probably acts on Na^+ and K^+ currents. Two pivotal studies in over 1,100 patients showed a **median survival gain of about three months**, confirmed by meta-analysis, with the probability of surviving one year rising by **9% (58% versus 49%)**, and small effects on bulbar and limb function. **Despite more than 200 trials, ALS remains incurable.**

20. **Multidisciplinary symptomatic care is what improves survival and quality of life** — non-invasive ventilation, nutritional support with gastrostomy, secretion management, treatment of spasticity and cramps, pseudobulbar affect, communication aids and mobility support. ALS is also the model disease for **discussing advance directives and palliative end-of-life care early**, because cognitive capacity is preserved in most patients throughout.

Latest Updates (2023 – Present)

- **Tofersen (Qalsody)** — an **intrathecal antisense oligonucleotide targeting SOD1 mRNA** — received FDA accelerated approval in **April 2023**, the first therapy addressing a genetic cause of ALS. SOD1-ALS accounts for about 2% of cases. In **VALOR** it missed its primary endpoint at 28 weeks, but produced a **greater than 50% fall in CSF and plasma neurofilament light**, and open-label extension data showed better outcomes with earlier initiation. It has since been approved in the EU and, in **July 2025**, by the UK MHRA.
- **Neurofilament light chain is now an established pharmacodynamic biomarker** in ALS — it tracks progression, predicts survival, and was the surrogate endpoint that supported tofersen's accelerated approval. This is the clearest example in neurology of a fluid biomarker changing a regulatory decision.
- **The ATLAS trial** is testing tofersen in **presymptomatic** SOD1 carriers with rapidly progressive mutations, asking whether treatment can delay symptom onset — the first true prevention trial in MND.
- **Relyvrio/Albrioza (sodium phenylbutyrate/taurursodiol, AMX0035)** was **withdrawn from the US and Canadian markets in 2024** after the Phase 3 **PHOENIX** trial failed to confirm the Phase 2 CENTAUR result — an important cautionary example of accelerated approval without confirmatory evidence.
- **Reldesemtiv** (fast skeletal muscle troponin activator) was negative in **COURAGE-ALS** (*JAMA Neurology* 2025;82:477–485).
- **Edaravone** (intravenous and oral suspension) remains available in several countries for patients meeting narrow eligibility criteria, with modest slowing of functional decline.
- Emerging strategies include further antisense oligonucleotides (targeting C9orf72 and FUS), **Exportin-1 inhibition** to modulate SOD1 nuclear export, and gene and stem cell therapies.
- **Indian relevance:** riluzole is available and affordable; edaravone and tofersen are not routinely accessible. The realistic priority is **early multidisciplinary care, non-invasive ventilation and gastrostomy**, with advance care planning under the framework established by the Supreme Court's *Common Cause* judgments on advance medical directives.

Current Practice Update

Aspect	Textbook position	Current evidence / practice
Disease-modifying drugs	Riluzole only; ALS incurable despite 200+ trials	Riluzole plus edaravone; tofersen for SOD1-ALS — the first gene-targeted therapy
Biomarkers	"No positive diagnostic markers"	Neurofilament light is a validated prognostic and pharmacodynamic biomarker
Genetic testing	For counselling in familial cases	Mandatory before therapy — SOD1 status determines tofersen eligibility
AMX0035	Not discussed	Approved 2022, withdrawn 2024 after negative PHOENIX trial
Presymptomatic treatment	Not conceived	ATLAS trial in asymptomatic SOD1 carriers

18. Dizziness in the Elderly

> **Coverage note:** the uploaded textbooks address dizziness within the chapters on falls, syncope and comprehensive geriatric assessment rather than as a dedicated chapter. Points 1–14 are textbook-derived; points 15–20 and the manoeuvre details are drawn from current peer-reviewed guidance, which is stated explicitly.

Fifteen–Twenty High-Yield Points

Concept and classification

1. **Dizziness in the elderly is a geriatric syndrome, not a single diagnosis** — it usually results from the accumulation of deficits in the visual, vestibular, proprioceptive, cardiovascular and cognitive systems, aggravated by drugs.
2. The classical classification distinguishes four types: **vertigo** (a sensation of rotational movement), **presyncope/light-headedness**, **disequilibrium** (unsteadiness on standing or walking), and **non-specific dizziness** (often psychogenic).
3. **Vertigo is a more specific symptom** and is most commonly associated with inner ear disorders. **Light-headedness is non-specific and should be interpreted with caution.**
4. Screening question in comprehensive geriatric assessment: **"Do you get dizzy, or does your head spin around?"** The recommended assessments are **checking for postural hypotension, the Hallpike manoeuvre for BPPV, haemoglobin, and the Geriatric Depression Scale**.

Peripheral vestibular causes

5. **Benign paroxysmal positional vertigo (BPPV)** is the commonest cause in older people — brief episodes triggered by head position change, from otoconial debris in the posterior semicircular canal. Diagnosed by the **Dix-Hallpike manoeuvre**, which reproduces vertigo with characteristic upbeating-torsional nystagmus.
6. Other inner ear causes: **acute labyrinthitis and vestibular neuritis** (single prolonged episode with nausea and vomiting) and **Ménière's disease** (episodic vertigo with fluctuating hearing loss and tinnitus).
7. Nystagmus can be demonstrated at the bedside by **having the patient lie down quickly with the head turned to one side in a single motion**; patients with vertigo from organic disorders often have nystagmus.
8. Patients with symptoms suggestive of vertigo benefit from a **thorough otological examination including auditory testing**, which helps differentiate inner ear from central nervous system involvement.

Cardiovascular causes

9. **Orthostatic hypotension** is best detected by measuring blood pressure and pulse **supine, after one minute sitting, and after one and three minutes standing**. A fall of **more than 20 mmHg systolic** is significant; many cases are asymptomatic.
10. Causes of orthostatic hypotension: **low cardiac output from heart failure or hypovolaemia, overtreatment with cardiovascular drugs, autonomic dysfunction (diabetes, Parkinson's disease), impaired venous return, and prolonged bed rest with deconditioning**. **Postprandial hypotension** is a distinctly geriatric cause — simply eating a full meal can lower blood pressure enough to precipitate a fall on standing.
11. **Carotid sinus hypersensitivity** and **reflex (vasovagal) syncope** become increasingly common with age; carotid sinus syndrome is a modifiable risk factor for non-accidental falls, and carotid sinus massage should be considered in unexplained or recurrent falls.
12. Cardiac causes include **bradyarrhythmias, tachyarrhythmias and aortic stenosis**. In general, **cardiac monitoring should document a temporal association between a specific arrhythmia and the symptom** before it is accepted as causal. **A precise cause for syncope remains unidentified in 40–60% of older patients.**

Central, drug-related and psychological causes

13. **Central causes:** vertebrobasilar ischaemia and infarction, **cerebellar infarction**, cerebellar degeneration, posterior fossa tumour, and multiple sclerosis. Cerebellar stroke is the diagnosis that must not be missed in an older patient with sudden isolated vertigo.
14. **Drugs are the most important reversible cause.** Culprits are diuretics (hypovolaemia), antihypertensives, antidepressants (postural hypotension), sedatives, antipsychotics (sedation, rigidity, hypotension), hypoglycaemics, anticonvulsants, aminoglycosides (ototoxicity) and alcohol. **Combinations greatly increase the risk.**
15. **Psychological factors:** many older patients with dizziness and unsteadiness are **anxious, depressed and chronically afraid of falling**, which makes evaluation difficult and means that mood must always be assessed. **Drop attacks** — sudden leg weakness causing a fall without loss of consciousness — are often attributed to vertebrobasilar insufficiency but are poorly understood and over-diagnosed.

Evaluation and management

16. **The single most useful clinical discipline is to distinguish falls from syncope from seizure, and "dizziness" from true vertigo.** Take a detailed history of timing, triggers, duration and associated symptoms, and always seek a witness account.
17. **Core examination:** postural blood pressure, cardiovascular examination including murmurs, cranial nerve and cerebellar examination, gait and balance assessment (**Timed Up and Go**, gait speed over 10 metres), visual acuity, otoscopy with removal of **cerumen**, and the Dix-Hallpike manoeuvre.
18. **Targeted investigation only:** haemoglobin, glucose, electrolytes, renal and thyroid function, vitamin B12, ECG; ambulatory monitoring or an implantable loop recorder for suspected arrhythmia; tilt-table testing and carotid sinus massage for unexplained syncope; MRI where central pathology is suspected. **Do not send all patients for extensive**

diagnostic studies.

19. **Management principles:** treat the specific cause; **rationalise medications**; correct visual and hearing impairment; ensure adequate hydration and salt intake; use compression stockings and counter-pressure manoeuvres for orthostatic hypotension; refer for rehabilitation with strengthening, balance and gait training; and address environmental hazards.
20. **Specific therapies** (current literature): **the Epley canalith repositioning manoeuvre** is the treatment of choice for posterior canal BPPV; **vestibular rehabilitation exercises** for unilateral vestibular hypofunction; a short course of vestibular sedatives only for acute vestibular neuritis, since **drugs such as meclizine and benzodiazepines compromise alertness and cognition** and delay central compensation; midodrine or fludrocortisone for refractory orthostatic hypotension; and pacing for documented carotid sinus syndrome with cardioinhibition.

Latest Updates (2023 – Present)

- **HINTS examination** (Head Impulse, Nystagmus, Test of Skew) is now the standard bedside tool in **acute vestibular syndrome**. Performed by a trained clinician, it is **more sensitive than early MRI-DWI** for posterior circulation stroke. A **normal head impulse test, direction-changing nystagmus, or skew deviation** indicates a central cause requiring urgent imaging.
- The Bárány Society's international classification of vestibular disorders has established diagnostic criteria for **persistent postural-perceptual dizziness (PPPD)** — now recognised as a leading cause of chronic dizziness, treated with vestibular rehabilitation, SSRIs and cognitive behavioural therapy rather than vestibular sedatives.
- **Presbyvestibulopathy** has been defined as a distinct Bárány Society entity — age-related bilateral vestibular hypofunction presenting with chronic unsteadiness and oscillopsia — validating "dizziness of ageing" as a diagnosable and rehabilitable condition.
- The **AAO-HNS BPPV clinical practice guideline update** reaffirms that **canalith repositioning is first-line and that vestibular suppressants should not be used for BPPV**; imaging and vestibular testing are not indicated in uncomplicated BPPV.
- The **"TiTrATE" framework** (Timing, Triggers, And Targeted Examination) has largely replaced the older "what do you mean by dizzy?" approach, because symptom-quality descriptions in older people are unreliable and inconsistent. **Timing and triggers, not the quality of the sensation, drive the differential.**
- Evidence continues to support **vestibular rehabilitation** as effective for chronic dizziness in older adults, with benefits for balance confidence and fall reduction, and it is increasingly delivered by telerehabilitation.
- **Deprescribing** is now framed as a therapeutic intervention in its own right: STOPP/START and Beers criteria specifically flag drugs causing orthostatic hypotension and sedation in older patients presenting with dizziness or falls.

Current Practice Update

Aspect	Textbook position	Current evidence / practice
Diagnostic approach	Classify by symptom quality (vertigo, light-headedness, unsteadiness)	TiTrATE — classify by timing and triggers; symptom quality is unreliable
Acute vertigo	Nystagmus assessment and otological examination	HINTS examination, more sensitive than early MRI for posterior circulation stroke
BPPV treatment	Diagnosed by Hallpike manoeuvre	Treated definitively with the Epley repositioning manoeuvre; vestibular suppressants contraindicated
Chronic dizziness	Attributed to anxiety, depression, fear of falling	Defined entities: PPPD and presbyvestibulopathy, both with specific treatment
Drug therapy	Vestibular sedatives used symptomatically	Minimised — they impair compensation, alertness and cognition

19. Ring-Enhancing Shadow on CT Brain

> **Coverage note:** the uploaded geriatric textbooks do not cover ring-enhancing intracranial lesions as a discrete topic. This answer is therefore constructed from current neuroradiology and infectious disease literature, with emphasis on the Indian epidemiological context, as instructed.

Fifteen–Twenty High-Yield Points

Definition and mechanism

1. A **ring-enhancing lesion** is a rounded intracranial lesion with a peripheral rim of contrast enhancement and a non-enhancing centre, usually with surrounding vasogenic oedema and mass effect.
2. The mechanism is **breakdown of the blood-brain barrier** in a peripheral zone of vascularised, inflamed or proliferating tissue, surrounding a central core of necrosis, pus or cyst fluid that has no blood supply and therefore does not enhance.

Differential diagnosis — the classical mnemonic

3. **MAGIC DR** is the standard aide-mémoire: **M**etastasis, **A**bscess, **G**lioblastoma, **I**nfarct (subacute), **C**ontusion, **D**emyelination, **R**esolving haematoma. Radiation necrosis and tuberculoma are the two important additions.
4. **In India, neurocysticercosis and tuberculoma together account for the majority of ring-enhancing lesions**, whereas in Western series metastasis and glioblastoma predominate. This epidemiological difference is the single most examinable point.
5. **In an elderly Indian patient specifically**, the probability shifts towards **metastasis, glioblastoma, pyogenic abscess and tuberculoma**, with neurocysticercosis less likely than in a young adult presenting with a first seizure.

Distinguishing features — infective causes

6. **Neurocysticercosis (NCC):** typically a **small (<20 mm) lesion at the grey-white junction**, often solitary in the Indian "solitary cysticercus granuloma" form, with **disproportionately little oedema for its size** and an **eccentric hyperdense scolex** ("hole-with-dot" sign) that is pathognomonic. Presents with seizures; resolves or calcifies over months.
7. **Tuberculoma:** usually **larger, with thick irregular walls**, marked perilesional oedema, and characteristically **T2 hypointense central caseation** on MRI. Often multiple, may be basal, and is frequently accompanied by **basal exudates and hydrocephalus** if there is coexistent tuberculous meningitis. MR spectroscopy shows a **lipid peak with a low choline-to-creatine ratio**.
8. **Pyogenic brain abscess:** a **smooth, thin, uniform ring that is thinner on the ventricular side** (because of poorer vascularity of white matter), with marked oedema. The distinguishing feature is **restricted diffusion on DWI with a low ADC value in the centre** (pus). MR spectroscopy shows amino acid, acetate, succinate and lactate peaks.
9. **Fungal abscess** (aspergillosis, mucormycosis) should be considered in diabetics, transplant recipients and the immunosuppressed; look for sinus and orbital involvement and vascular invasion.
10. **Toxoplasmosis** causes multiple lesions in the basal ganglia and at the corticomedullary junction, with an **"eccentric target sign"**, in HIV-infected patients with CD4 counts below 100/µL.

Distinguishing features — neoplastic causes

11. **Metastasis:** characteristically **multiple, at the grey-white junction**, with **oedema markedly out of proportion to lesion size** and an irregular, often nodular ring. In an elderly patient, the primary is usually lung, breast, kidney, colon or melanoma; a contrast CT chest, abdomen and pelvis is the first staging step.
12. **Glioblastoma:** a **thick, irregular, shaggy ring** with central necrosis, marked oedema, and characteristic **spread across the corpus callosum ("butterfly glioma")**. Perfusion imaging shows increased relative cerebral blood volume in the enhancing rim, and spectroscopy shows a **raised choline-to-creatine ratio with reduced NAA**.
13. **Primary CNS lymphoma** is usually homogeneously enhancing but may show a ring pattern in immunosuppressed patients; it is characteristically periventricular and steroid-responsive, so **steroids should be withheld until biopsy** where lymphoma is suspected.

Distinguishing features — vascular and other causes

14. **Subacute infarct:** ring or gyriform enhancement appearing at **1–3 weeks** ("luxury perfusion"), confined to a vascular territory, with a wedge shape involving cortex and no mass effect out of proportion to the infarct.
15. **Resolving haematoma** and **contusion:** a peripheral enhancing rim around an evolving clot; the history of trauma or hypertension and the changing density on serial imaging give the diagnosis.
16. **Demyelination (tumefactive multiple sclerosis, ADEM):** an **incomplete or open ring** that is open towards the cortex, with **little mass effect relative to lesion size** — the "open ring sign" is highly specific for demyelination.
17. **Radiation necrosis:** occurs months to years after cranial radiotherapy in the previously irradiated field; distinguished from tumour recurrence by low perfusion and low choline on advanced imaging.

Approach and management

18. **MRI with contrast, diffusion-weighted imaging and MR spectroscopy is far superior to CT** and should be requested in every case where the diagnosis is not immediately obvious. **DWI is the single most useful sequence**, since restricted diffusion strongly favours abscess over necrotic tumour.
19. **Work-up:** full history including fever, weight loss, immunosuppression, cancer and travel; examination for a primary tumour, source of sepsis and pulmonary tuberculosis; chest radiograph or CT; HIV testing, blood cultures, ESR, CRP;

and echocardiography if endocarditis is suspected. **Lumbar puncture is contraindicated in the presence of significant mass effect.**

20. **Management** depends on the diagnosis: neurosurgical aspiration plus prolonged antibiotics for abscess; antitubercular therapy with corticosteroids for tuberculoma; albendazole with corticosteroids and antiepileptics for neurocysticercosis; surgery, radiotherapy and oncological referral for tumours. **Where the diagnosis is uncertain and the lesion is accessible, stereotactic biopsy is definitive** — but in India a trial of antitubercular therapy with close radiological follow-up at 8–12 weeks remains an accepted strategy for a probable tuberculoma.

Latest Updates (2023 – Present)

- **Advanced MRI is now standard for lesion characterisation:** DWI/ADC, dynamic susceptibility contrast perfusion, and MR spectroscopy together achieve over 90% accuracy in distinguishing abscess from necrotic tumour, and tumour recurrence from radiation necrosis (multiple systematic reviews, 2023–2025).
- **Amide proton transfer (APT) imaging** and **radiomics/machine-learning classifiers** applied to routine MRI are emerging as non-invasive methods to differentiate ring-enhancing lesions without biopsy.
- **Indian consensus on neurocysticercosis** continues to support albendazole (with or without praziquantel for multiple lesions) plus corticosteroids and antiepileptic drugs for solitary cysticercus granuloma, with antiepileptics usually withdrawn after radiological resolution.
- **WHO and NTEP (National Tuberculosis Elimination Programme) guidance** recommends **12 months of antitubercular therapy for CNS tuberculosis**, with adjunctive corticosteroids; **paradoxical enlargement of tuberculomas on treatment** is well recognised and does not by itself indicate treatment failure — a frequently examined point.
- **Cell-free DNA and next-generation sequencing of CSF** are being introduced for pathogen identification in brain abscess where cultures are negative, and for detection of tumour-derived mutations.
- In the elderly specifically, the **threshold for suspecting metastasis is lower**, and current practice favours early whole-body imaging rather than empirical antimicrobial or antitubercular trials.

Current Practice Update

Aspect	Textbook position	Current evidence / practice
Coverage	Not addressed in the uploaded geriatric textbooks	Standard neuroradiology topic; MAGIC DR plus tuberculoma and neurocysticercosis
Imaging modality	—	Contrast MRI with DWI, perfusion and spectroscopy replaces CT for characterisation
Diagnostic confirmation	—	Stereotactic biopsy where uncertain; CSF next-generation sequencing for culture-negative infection
Indian practice	—	Empirical ATT with 8–12 week radiological review accepted for probable tuberculoma; lower threshold for biopsy in the elderly

20. Causes of Coma in the Elderly

Fifteen–Twenty High-Yield Points

Definition and anatomy

1. **Coma is a state of unarousable unresponsiveness** — the deepest grade on the scale of consciousness used in delirium assessment: alert, vigilant, lethargic, **stupor (difficult to arouse)**, and **coma (unarousable)**.
2. Consciousness requires two components: **arousal**, dependent on the ascending reticular activating system in the brainstem and its thalamic projections, and **awareness**, dependent on the cerebral cortex. **Coma therefore requires either a brainstem lesion, bilateral hemispheric dysfunction, or a diffuse metabolic insult.** A single unilateral hemispheric lesion causes coma only if it produces mass effect and herniation.
3. In the elderly, **the threshold for coma is lower** because of reduced neuronal reserve, pre-existing cognitive impairment, polypharmacy and multimorbidity — the same insult that causes mild confusion in a young adult may cause coma in an older person.

Metabolic and toxic causes

4. **Hypoglycaemia** is the single most important immediately reversible cause and must be excluded at the bedside in every patient — particularly in older people on sulphonylureas or insulin with declining renal function or poor intake.
5. Other metabolic causes listed among the causes of delirium and confusion, and which at their extreme produce coma: **hypoxia, hypercarbia, hyperglycaemia, hyponatraemia and azotaemia**.
6. **Hyperglycaemic emergencies: hyperosmolar hyperglycaemic state** is the characteristic presentation in the elderly and carries high mortality; **diabetic ketoacidotic coma** is less common in this age group.
7. **Endocrine: myxoedema coma** (hypothermia, bradycardia, hyponatraemia, precipitated by infection or cold) and **adrenal (Addisonian) crisis**; both are classic geriatric causes of unexplained coma with hypothermia.
8. **Organ failure:** hepatic encephalopathy, uraemic encephalopathy, carbon dioxide narcosis in type 2 respiratory failure, and severe hypothyroidism.
9. **Electrolyte and mineral disturbance:** severe hyponatraemia (and over-rapid correction causing osmotic demyelination), hypercalcaemia (often malignancy-related), and severe hypo- or hypermagnesaemia.
10. **Nutritional: Wernicke's encephalopathy** from thiamine deficiency — frequently missed in older patients with poor intake, malignancy or after refeeding, and not confined to alcohol misuse.
11. **Drugs and toxins** are among the commonest causes: opioids, benzodiazepines, tricyclic antidepressants, anticholinergics, lithium, anticonvulsants, alcohol intoxication, and **carbon monoxide poisoning**. Polypharmacy and altered pharmacokinetics make the elderly especially vulnerable, and drug intoxication is explicitly listed among the causes of acute confusional states.
12. **Thermal: hypothermia and hyperthermia** are listed causes; both are more frequent in older people because of impaired thermoregulation, and heat stroke is an increasing problem in Indian summers.

Structural intracranial causes

13. **Stroke:** massive hemispheric infarction with cerebral oedema (**malignant MCA infarction**) causing midline shift, raised intracranial pressure and **transtentorial herniation with brainstem compression**; and brainstem or bilateral thalamic infarction, where coma occurs directly.
14. **Intracerebral haemorrhage**, especially with intraventricular extension or cerebellar haemorrhage — where **surgical evacuation of a cerebellar haematoma is a life-saving measure**.
15. **Subarachnoid haemorrhage:** sudden severe headache with vomiting, neck stiffness, **altered conscious level and collapse**. Mortality is **35–65%**, and outcome is predicted by **Glasgow Coma Scale score, age and the volume of intracranial blood**.
16. **Subdural haematoma** is a particularly geriatric diagnosis. It **occurs with trivial trauma and sometimes without any trauma**, especially in those taking anticoagulants, and may present as fluctuating consciousness or confusion. Poor outcome is predicted by **advanced age, low GCS at presentation, comorbid injuries and anticoagulant use**.
17. **Intracranial infection:** bacterial meningitis, encephalitis (herpes simplex), cerebral abscess and, in India, tuberculous meningitis with hydrocephalus — where the classical triad may be absent and confusion or coma may be the only feature.
18. **Space-occupying lesions:** primary and metastatic brain tumours; brain tumours are responsible for 10–30% of new seizures in the elderly and may present with progressive obtundation from oedema.

Other causes and approach

19. **Seizure-related:** post-ictal state, and importantly **non-convulsive status epilepticus**, which in older patients may present as unexplained prolonged coma without motor manifestations and requires **EEG** for diagnosis. Cerebrovascular disease is the leading cause of seizures and status epilepticus in later life.
20. **Approach:** stabilise airway, breathing and circulation; **check capillary glucose immediately**; give thiamine before glucose where deficiency is possible; consider naloxone; measure temperature; assess **GCS, pupils, brainstem reflexes and any focal or lateralising signs**; take a full drug and collateral history; obtain urgent **non-contrast CT brain**; and perform lumbar puncture once mass effect is excluded. Remember that **coma in the elderly is frequently multifactorial**, and treatable contributors such as sepsis, hypoxia, hypoglycaemia and drug toxicity should all be corrected simultaneously.

Latest Updates (2023 – Present)

- **Terminology has been standardised** by the European Academy of Neurology and the American Academy of Neurology: "vegetative state" is replaced by **unresponsive wakefulness syndrome**, and **minimally conscious state** and **cognitive motor dissociation** are formally defined. The **Coma Recovery Scale-Revised (CRS-R)** is the recommended assessment instrument for prolonged disorders of consciousness.
- **Cognitive motor dissociation** — covert awareness detectable on task-based fMRI or EEG in patients who appear unresponsive — is now recognised in roughly a quarter of behaviourally unresponsive patients (*NEJM* 2024), with major implications for prognostication and for decisions to withdraw treatment.
- **Full Outline of UnResponsiveness (FOUR) score** is increasingly preferred to the GCS in intubated patients, as it assesses brainstem reflexes and respiratory pattern and can be scored when the verbal component of the GCS is untestable.
- **Point-of-care ultrasound of the optic nerve sheath diameter** and automated pupillometry (neurological pupil index) are being adopted as non-invasive bedside measures of raised intracranial pressure.
- **Middle meningeal artery embolisation** has emerged as an important addition to burr-hole drainage for chronic subdural haematoma, addressing the underlying membrane vascularity and reducing the recurrence rate, which is otherwise as high as **33%** after standard surgery — a significant advance for frail older patients.
- **Serum neurofilament light and neuron-specific enolase**, combined with continuous EEG and MRI, form the current multimodal approach to prognostication after hypoxic-ischaemic brain injury, replacing reliance on single clinical signs.
- **Non-convulsive status epilepticus** is increasingly recognised in older patients with unexplained coma; the **Salzburg criteria** standardise EEG diagnosis, and continuous EEG monitoring is recommended for any unexplained persistent alteration of consciousness.

Current Practice Update

Aspect	Textbook position	Current evidence / practice
Terminology	Coma described as a level of consciousness within delirium assessment	Formal taxonomy: coma, unresponsive wakefulness syndrome, minimally conscious state, cognitive motor dissociation
Assessment scale	Glasgow Coma Scale	GCS plus FOUR score in intubated patients; CRS-R for prolonged disorders of consciousness
Covert awareness	Not recognised	Detected by task-based fMRI/EEG in about 25% of unresponsive patients
Chronic subdural haematoma	Burr-hole or twist-drill craniostomy with subdural drain	Middle meningeal artery embolisation as adjunct or alternative, reducing recurrence
Unexplained coma	Structural and metabolic causes sought	Continuous EEG mandated to exclude non-convulsive status epilepticus (Salzburg criteria)

21. Normal Pressure Hydrocephalus

Fifteen–Twenty High-Yield Points

Definition and historical background

1. **NPH is an adult-onset chronic condition** with **enlarged cerebral ventricles on imaging** plus the clinical **Hakim and Adams triad — gait disturbance, cognitive decline and urinary incontinence** — occurring **without high CSF pressure or obstruction** to flow through the ventricular system and subarachnoid space.
2. **It is a potentially reversible neurological disease**, but the mechanisms are unclear and awareness is poor, leading to **under-recognition and undertreatment**. In older people the triad overlaps other age-related diseases and may be wrongly attributed to ageing itself as a result of ageism.
3. **Hakim and Adams** recognised the syndrome in 1957 and published it in 1965, linking the triad definitively to CSF accumulation in the ventricles and showing improvement after lumbar puncture and shunt diversion. **Evans' ratio** was described in 1942 following the introduction of air ventriculography in 1918.

Epidemiology

4. Crude prevalence is **10–22/100,000 for probable NPH and 29/100,000 for possible NPH**. Age-specific rates rise from **3.3/100,000 at age 50–59 to 5.9% in those aged 80 and above**. Incidence rises from 0.07/100,000/year under 60 to **1.2/1,000/year over 70**. In **nursing homes, incidence rates are 9–14%** depending on the criteria used.

Pathophysiology

5. Symptoms **do not result simply from impaired CSF dynamics**. The interacting mechanisms are **altered CSF dynamics and impaired resorption, reduced intracranial compliance, abnormal cerebral perfusion with periventricular ischaemia, altered metabolism, oedema and demyelination, and accumulation of neurotoxic substances** — together producing "cerebral dyshomeostasis".
6. Recognised associations include **cardiac systolic dysfunction** and **obstructive sleep apnoea**, which is frequently co-existent and should be actively sought.

Clinical presentation

7. **Gait disturbance is the earliest and most prominent symptom — wide-based, slow, unsteady and magnetic**, with disturbed tandem walking and turning, small steps, **reduced floor foot clearance**, start hesitation, tendency to fall and disturbed trunk balance. The Dutch NPH study quantified this as **25 steps and 16 seconds for a 10-metre walk versus 15 steps and 8 seconds in controls**.
8. **Cognition** shows an atypical profile combining **frontal-executive, subcortical and posterior cortical impairment**. **Memory and orientation are relatively preserved**; frontal impairment affects **recall disproportionately to recognition**, with attention deficit and reduced psychomotor speed. **In Alzheimer's disease the temporal-lobe profile impairs both recall and recognition** — the key clinical contrast.
9. **Urinary symptoms** begin as frequency and urgency and progress to urge and then complete incontinence as frontal deterioration prevents recognition of the urge. Mechanisms are **reduced bladder capacity (mean 200 mL), detrusor overactivity and enhanced parasympathetic activity**, reversible after LP and shunting. **Faecal incontinence occurs only in advanced stages.**
10. Severity is graded using the **iNPH Grading Scale (iNPHGS)**, which scores gait, cognition and urinary function from 0 to 4 each.

Differential diagnosis

11. Must be distinguished from **Alzheimer's disease, frontotemporal degeneration and vascular cognitive impairment; Parkinson's disease, vascular parkinsonism and Parkinson-plus syndromes; primary progressive freezing gait; and lumbar canal stenosis**. **NPH gait does not respond to external cues or to levodopa**, unlike Parkinson's disease, and there is **no tremor or rigidity**.

Imaging and diagnostic criteria

12. **Imaging features:** ventricular enlargement not entirely attributable to atrophy (**Evans' index >0.3**), no macroscopic obstruction to CSF flow, an **aqueductal or fourth ventricular flow void** on MRI, plus at least one of — enlarged temporal horns, **callosal angle of 90 degrees or less**, or altered brain water content not attributable to microvascular ischaemia or demyelination.
13. **DESH — disproportionately enlarged subarachnoid space hydrocephalus**, i.e. narrowed sulci and subarachnoid spaces over the high convexity and midline with dilated Sylvian fissures — is stressed by the **revised 2012 Japanese criteria** as a very important diagnostic criterion, which can only be overcome by evidence of improvement after a CSF tap or drainage test.
14. **American guidelines (Relkin, Marmarou et al.)** classify NPH as **probable, possible or unlikely**. Supporting history: insidious onset after age 40, duration of at least 3–6 months, progression over time, no antecedent cause of secondary hydrocephalus, and no alternative explanation. **Gait/balance disturbance must be present plus impairment of cognition, urinary function or both.** Probable NPH requires an **opening pressure of 5–18 mmHg (70–245 mm H_2O)**; higher or lower pressures are inconsistent with the diagnosis.
15. **Japanese criteria:** *possible* iNPH — onset in the 60s or later, more than one triad element, Evans' index >0.3, no other explanation, and no obvious antecedent cause; *probable* — adds CSF pressure of 200 mm H_2O or less with normal content, plus either DESH, or improvement after the CSF tap test, or improvement after the drainage test; **definite iNPH — improvement of symptoms after the shunt procedure.**

Supplementary predictive tests

16. **CSF tap test (high-volume LP):** walking speed is assessed in single-task and dual-task conditions, with **maximal improvement 24–48 hours after LP for single-task tests and 48–72 hours for dual-task tests**, returning to the pre-LP state after a few weeks, which further supports the diagnosis. **Sensitivity is low (26–61%), so a negative tap test cannot exclude NPH**; a positive test carries greater certainty of shunt response than clinical examination alone.
17. **Extended lumbar drainage (ELD):** a lumbar catheter draining **100–150 mL of CSF per day for about four days**, with daily assessment of gait and cognition. **Sensitivity 50–100% and positive predictive value 80–100%**, better than LP alone, but a **negative result has deceptively low value** because of frequent false negatives. Discontinue for low-pressure headache, nerve root irritation or signs of meningitis. **Prior stroke predicts a worse outcome; DESH predicts a better one.**
18. **CSF outflow resistance (Ro) by infusion test** has sensitivity **57–100%** and PPV **75–92%**. **Static ICP measurement does not predict surgical outcome** — it is **ICP pulsatility and B-waves** (slow rhythmic oscillations at 0.33–3 cycles/minute) that carry clinical significance, indicating reduced intracranial compliance.
19. **CSF biomarkers: a decrease in A-beta-42 is seen in both NPH and Alzheimer's disease, but a rise in phospho-tau is seen only in Alzheimer's** — useful for differential diagnosis, although **no CSF test has prognostic value before shunting**.

Treatment, outcome and complications

20. **CSF diversion is the only evidence-based curative treatment**; repeated high-volume lumbar punctures have no long-term efficacy in non-operated patients. **Ventriculoperitoneal shunting is standard care and succeeds in about 80% of cases.** Improvement is greatest for **gait (81–86%) and bladder control (70%) and least consistent for cognition (40–44%)**; there is a **96% chance of subjective improvement and 83% chance of improvement on timed walk at six months**, against a **serious adverse event risk of about 11%**. **Age is not a prognostic factor.** Complications include **shunt infection (a neurosurgical emergency), subdural effusion (managed by raising the setting of an adjustable valve), obstruction, catheter fracture and transient hearing loss**; **ventricles do not necessarily shrink after successful shunting**. **Endoscopic third ventriculostomy** avoids shunt hardware but may fail if the stoma closes.

Latest Updates (2023 – Present)

- **The PENS trial is the single most important development and is now the definitive evidence base.** *Placebo-Controlled Effectiveness in iNPH Shunting* (Luciano, Williams and Hamilton et al., ***NEJM*** **2025;393:2198–2209**) randomised **99 patients across 17 centres in the US, Canada and Sweden** — all of whom had responded to temporary CSF drainage — to an **open versus placebo setting on a non-invasively adjustable valve**, keeping patients and assessors blinded.
- **Result:** gait velocity improved by **0.23 m/s with an open shunt versus 0.03 m/s with placebo — treatment difference 0.21 m/s (p<0.001)**. Lateral ventricular volume fell by 27.4 mL versus 4.4 mL. **Falls were significantly fewer with an open shunt (24.5% versus 46%, p=0.03).** The DSMB **stopped enrolment early at the second interim analysis (January 2025)** for efficacy.
- Crucially, PENS confirmed benefit for **gait and balance but NOT for cognition or incontinence at three months** — validating the textbook observation that gait responds best and providing a realistic basis for counselling patients and families.
- PENS answers long-standing scepticism about shunting: there had been **calls for a moratorium** on grounds of variable results, questionable durability of benefit, surgical risk and a potential strong placebo effect. The **2024 Cochrane review (CD014923)** had concluded that further studies were needed to increase certainty; PENS supplies that certainty.
- **Prevalence data have been refined:** a Swedish population-based MRI study of **791 seventy-year-olds (*Neurology* 2024;102:e208037)** confirms possible iNPH is far commoner in the community than clinic-based figures suggest, reinforcing that this is an under-diagnosed treatable cause of disability in old age.
- **Glymphatic dysfunction** is now central to the accepted pathophysiology — impaired perivascular clearance with accumulation of neurotoxic metabolites — linking iNPH mechanistically to Alzheimer's co-pathology and explaining why cognition responds less reliably than gait.
- Contemporary reviews (*Neurosurgery Clinics of North America* 2024) reaffirm that **DESH and reduced callosal angle support the diagnosis but have limited predictive value for treatment response** — the provocative CSF removal tests (tap test, infusion test, extended lumbar drainage) remain what actually stratifies who will respond, and adding them progressively increases predictive power.
- **Programmable, non-invasively adjustable valves with anti-siphon devices are now standard**, allowing pressure adjustment for over-drainage and subdural collections without reoperation — and it is precisely this technology that made the blinded placebo-controlled PENS design possible. **VP versus lumboperitoneal shunting** is being compared head-to-head (COVLINPH-1, primary outcome shunt failure at five years).
- **Indian context:** iNPH is markedly under-recognised, and the practical barriers are access to neurosurgical services and to programmable valves. A **tap test with objective 10-metre gait speed measured before and 24–72 hours after LP** is a cheap, reproducible screening strategy any geriatric unit can implement, and should be considered in every older patient with unexplained gait apraxia plus cognitive or urinary symptoms.

Current Practice Update

Aspect	Textbook position	

		Current evidence / practice
Evidence for shunting	About 80% success from observational series; effectiveness questioned, with calls for a moratorium	PENS trial (*NEJM* 2025) — first adequately powered blinded RCT; confirms benefit and was stopped early for efficacy
Which symptom improves	Gait and bladder best, cognition least consistent	Confirmed and sharpened: significant gain in gait velocity and balance, **no** significant gain in cognition or incontinence at 3 months
Falls	Not a formal endpoint	Shunting roughly halves falls (24.5% versus 46%) — a directly relevant geriatric outcome
Pathophysiology	Altered CSF dynamics, perfusion, ischaemia, neurotoxic accumulation	Adds **glymphatic system dysfunction** as a unifying mechanism
Imaging markers	DESH stressed as a key diagnostic criterion (Japanese 2012)	DESH and callosal angle support diagnosis but **poorly predict shunt response**; CSF removal tests remain the selection tool
Valve technology	Adjustable valves available	Non-invasively adjustable valves with anti-siphon devices are standard, and enabled the placebo-controlled trial design

22. Stroke Rehabilitation

Fifteen–Twenty High-Yield Points

Definition and conceptual framework

1. **Stroke is the leading cause of severe physical disability in adults.** Rehabilitation is defined as a **multidisciplinary problem-solving educational process focusing on disability and intended to reduce handicap** — it is not simply treatment by a therapist or group of therapists.
2. The **WHO/ICF framework** underpins assessment: **impairment** (loss of structure or function), **disability** (inability to perform tasks — the ability to undertake basic self-care is fundamental to any physical rehabilitation programme), and **handicap** (the social consequence, i.e. limitation in fulfilling a normal role in society). Disability and handicap are often combined pragmatically as **social disability**.
3. **Four basic principles:** document and measure impairments, disabilities and handicaps using simple valid scales; **maximise independence and minimise learned dependency**; adopt a **holistic approach** covering physical and psychosocial background, support mechanisms and environment; and **support caregivers**, helping them develop the physical and psychological skills for long-term sustainable care.

Basis of recovery

4. **The brain is capable of significant recovery after stroke, provided appropriate treatments are applied in adequate amounts and at the right time.** Recovery depends on resolution of oedema and penumbral salvage early, then on **neuroplasticity** — cortical reorganisation, unmasking of latent pathways, synaptogenesis and stroke-induced neurogenesis.
5. **The more severe the initial deficit, the less likely complete recovery.** The pattern is not uniform, varying between individuals and between different deficits in the same individual, and **there is no validated method for predicting the precise mode or degree of recovery for a given individual**.
6. **Rates of recovery differ by deficit:** homonymous hemianopia, dysphagia and sitting balance resolve quickly; **arm paralysis and language impairment recover more slowly and less completely**; perceptual problems may persist or take very long. Overall, **62% are independent in self-care at three months and 66% at one year**, despite persisting neurological deficit in some.
7. Day-to-day variation may mask recovery or give false optimism, so **monitor trends over time rather than relying on one-off assessments**. **Comorbidity and stroke-related complications adversely affect recovery. Spontaneous recovery may not plateau until six months**, which is the most appropriate time point for measuring neurological and functional outcome; participation and life satisfaction are best measured once the social situation has stabilised.

The four components of the rehabilitation process

8. **Assessment** defines the nature and severity of deficits and treatment goals before starting. Its three purposes are **prognostication** (type of patient, extent of disability, potential for recovery), identifying the main problems and the expectations of patient and family, and **monitoring and outcome evaluation**. Measures must be **valid, reliable, sensitive, simple and communicable**, and existing measures should be used wherever possible.
9. **Composite global severity scores are unreliable** because speech and language function dominates the index, and because combining very different disabilities into one score loses specific information and ignores their interactions.
10. Predicting when a patient has reached full recovery potential is an imprecise science, and matters most for **chronic stroke patients beyond one year**, who are most likely to be denied further therapy. **Combined TMS and MRI** — motor-evoked potentials in the affected limb with minimal fractional anisotropy asymmetry in the internal capsule — predicted potential for improvement up to **three years** after stroke.
11. **Planning is goal setting.** Immediate goals concern basic ADLs (sitting balance, transfers, toileting); later goals concern household, social, leisure and occupational activity. Three practical domains must be addressed: **accommodation** (where will the patient live and what adaptations are needed), **personal support** (what support exists and what extra help is essential), and **life satisfaction** (what roles will be fulfilled and how will time be occupied).
12. **The two major problems in goal setting are failure to use a common language between professionals and with patients, and failure to agree a time frame.** The goals of the hospital (early discharge), the patient (return to premorbid status) and the carer (minimise input, even at the cost of institutionalisation) frequently conflict; an essential team function is to **identify and modify unrealistically high — and sometimes unjustifiably low — expectations**.
13. **Intervention:** the minimum requirement is to maintain the status quo and prevent deterioration from poorly managed complications; beyond that, to facilitate recovery, minimise disability and prevent handicap. **No rehabilitation technique has been shown to be superior to any other** in physical therapy or in speech and language function, so a **pragmatic functional approach individualised to each patient** is recommended and rigid adherence to unproven theories should be discouraged.
14. **Evaluation** is confounded by spontaneous recovery, differing perceptions of a good outcome, and the profusion of instruments. **ADL scales predominate** because independence in ADLs determines care needs and resource use. Simple assessments used frequently are more useful than complex ones used rarely.

Specific rehabilitation techniques

15. **Early mobilisation** is associated with good functional outcomes in observational and controlled studies, but review of pooled data found **no unequivocal independent benefit**; it is, however, **not harmful to most stroke patients** and contributes as part of routine stroke unit care to good long-term outcomes.

16. **Motor recovery:** balance and motor therapy with or without sensory feedback improves function and mobility; **speed-dependent treadmill training** benefits ambulant hemiparetic patients; robotic devices allow repetitive practice. **Constraint-induced movement therapy (CIMT)** prevents learned non-use; the **EXCITE trial** showed statistically significant and clinically relevant improvement in arm motor function persisting at least one year, but doubts remain about practicality and cost-effectiveness, and meta-analysis suggests benefit is **proportional to the amount of exercise given to the affected limb**, potentially achievable by conventional therapy.
17. **Neuromuscular stimulation:** peripheral electrostimulation improves functional motor ability, motor impairment and normality of movement. **rTMS** aids recovery of motor deficit, visuospatial neglect and aphasia — **low-frequency stimulation over the unaffected hemisphere to restore inhibition, or high-frequency stimulation over the affected hemisphere to reactivate hypoactive regions**.
18. **Motor imagery / mental practice** positively affects arm recovery and shows promise for the leg, with **greater effect when combined with conventional rehabilitation** in subacute patients; **mirror therapy** enhances lower-limb motor recovery.
19. **Spatial neglect:** the conceptual shift is from a spatial lack of awareness to a **generalised reduction in attentional ability**. Spatial techniques such as prism adaptation have limited success, whereas **non-spatial attention training** improves neglect with corresponding change in cortical activation. **There is no effective treatment for apraxia** — management focuses on early recognition, increasing awareness, and teaching adaptive skills and coping strategies.

Complications, organisation and psychosocial care

20. **Complications occur in about 60% of patients undergoing rehabilitation** and are commoner with severe disability. Key problems are **dysphagia** (21–55% clinically, 44–78% on videofluoroscopy, with frequent **silent aspiration**; associated with higher mortality, longer stay and a **three- to fivefold increase in institutionalisation at five years**; videofluoroscopy is the gold standard and the water swallow test the practical bedside screen); **dysphasia** (comprehension is routinely overestimated); **spasticity** (**botulinum toxin has replaced baclofen and dantrolene** for focal spasticity — it reduces upper-limb tone but functional improvement is not established); the **hemiplegic shoulder** (subluxation in about one in five; pain from spasticity, subluxation, shoulder–hand syndrome or rotator cuff pathology, aggravated by careless handling; managed by positioning, avoiding traction on the arm and early passive movement); **post-stroke depression** (**30–60%, pooled prevalence about 33%**, higher in women, younger patients and those with greater disability, lasting seven to eight months untreated and correlating with failure to resume activities); plus central post-stroke pain, **fatigue** (no specific treatment shown to benefit), seizures, VTE, incontinence, pressure sores and **falls with fractures on the hemiplegic side**.
21. **Organised (stroke unit) care both facilitates neurological recovery and expedites discharge.** Audit shows patients spend **over 50% of time in bed, 28% sitting out, only 13% in therapeutic activity, and are alone 60% of the therapeutic day**; the **CERISE study** showed outcomes depend on **therapy actually delivered rather than resources allocated**. **Caregiver training is a proven intervention.** The reaction to stroke is **akin to bereavement**, and staff attitude matters — esteem plummets when patients are depersonalised as "CVAs" or "hemis". **Organised, coordinated rehabilitation in partnership with patients and caregivers offers the only realistic hope of reducing disability and handicap after stroke.**

Latest Updates (2023 – Present)

- **The 2024 VA/DoD Clinical Practice Guideline for the Management of Stroke Rehabilitation** (released July 2024; synopsis in *Annals of Internal Medicine* 2025) is the most current comprehensive guideline, updating the 2019 version using GRADE methodology.
- **Change of direction on antidepressants:** the guideline now **suggests AGAINST antidepressants for the prevention of post-stroke depression** (moved from "neither for nor against" in 2019 to "weak against" in 2024). This follows **FOCUS (n=3,127), AFFINITY and EFFECTS**, which showed **fluoxetine does not improve functional outcome and doubles the risk of bone fracture** — critical in an osteoporotic elderly population. Antidepressants remain indicated to **treat** established depression.
- **Very early mobilisation has been redefined by AVERT phase 3:** very early, high-dose, out-of-bed activity within 24 hours produced **worse** odds of a favourable outcome at three months than usual care. Current practice is **early but not aggressive** mobilisation — short, frequent, lower-dose sessions, avoiding prolonged upright activity in the first 24 hours. This resolves the textbook's uncertainty in a specific direction.
- **Vagus nerve stimulation paired with rehabilitation** is the major new modality. **VNS-REHAB (*Lancet* 2021)** led to approval of the **Vivistim Paired VNS System** for chronic ischaemic stroke with moderate-to-severe upper limb impairment; long-term outcomes (*Stroke* 2025) and a 2025 systematic review and meta-analysis (*Neurosurgery* 2025) support durable gains. The **2024 VA/DoD guideline** gives the clearest stance, noting that pairing VNS with task-specific rehabilitation may enhance motor recovery through neuroplasticity, while cautioning that the evidence is limited in quality and scope. **Transcutaneous auricular VNS** is under trial as a non-implanted alternative.
- **Robotics have been tempered by trial evidence:** the **RATULS trial** showed robot-assisted training did **not** improve upper limb function beyond enhanced conventional therapy, reinforcing that **dose and task specificity — not the device — drive recovery**.
- **Telerehabilitation is now established as non-inferior** to in-clinic therapy for motor recovery, and is the most scalable delivery model where therapists are unevenly distributed. In India, the **ATTEND trial (*Lancet* 2017)** found structured family-led home rehabilitation was **not superior to usual care** — an important negative result cautioning against substituting trained therapy with caregiver delivery alone.
- **Fitness training after stroke** (Cochrane, updated) consistently improves walking speed, endurance and balance and reduces disability — cardiorespiratory and mixed training now carry stronger recommendations than most specific

neurofacilitation techniques.

- Apart from the negative fluoxetine data, **no pharmacological adjunct has been established**; amphetamines, levodopa and citicoline have all failed to show consistent benefit.
- **Aphasia:** high-intensity, high-dose speech and language therapy is supported by Cochrane evidence, with **intensity and total dose** the critical variables; computer- and app-delivered therapy extends dose economically.
- **Indian context:** the Indian Stroke Association and NP-NCD promote a tiered rehabilitation model; the practical constraints are shortage of therapists, out-of-pocket cost and early discharge. **Early supported discharge with structured caregiver training** remains the highest-value model, and low-cost stroke units have been shown in Indian series to reduce dependency without intensive care facilities.

Current Practice Update

Aspect	Textbook position	Current evidence / practice
Early mobilisation	Benefit unproven but "not harmful to most patients"	AVERT 3: very early **high-dose** mobilisation worsens outcome — mobilise early but in short, frequent, low-dose sessions
Antidepressants	Post-stroke depression treated; prophylaxis not addressed	VA/DoD 2024 **suggests against** antidepressants for prevention; FOCUS/AFFINITY/EFFECTS negative, fluoxetine doubles fracture risk
Neuromodulation	rTMS and peripheral electrostimulation described	Adds approved **paired vagus nerve stimulation** for chronic upper limb impairment
Robotic therapy	Listed as an emerging technique	RATULS negative — dose and task specificity matter more than the device
Delivery of therapy	Inpatient, therapist-delivered, unit-based	Telerehabilitation non-inferior; early supported discharge and home-based programmes standard
Exercise and fitness	Mentioned within physical therapy	Cardiorespiratory fitness training has among the strongest evidence for walking and disability outcomes
Guideline framework	Consensus and pooled trial data	Formal GRADE-based VA/DoD 2024 guideline with explicit strength of recommendation

MD GERIATRIC MEDICINE — EXAMINATION REVISION

Nutrition in the Elderly

A Compiled Set of High-Yield Examination Answers

Five Questions · 15-Mark Format

Textbook Content • Latest Updates (2023–Present)

Current Practice Update • Examiner's Tips

Rapid Revision Appendix

Primary sources: Pathy's Principles and Practice of Geriatric Medicine, 6th edition

Essentials of Clinical Geriatrics, 7th edition

Updates current to August 2026 · Indian clinical context integrated throughout

Contents

Five questions on nutritional medicine in old age, in the order discussed.

#	Question	Core Focus
1	**Various nutritional disorders in the elderly and their management**	Whole spectrum — under- and over-nutrition
2	**Malnutrition in older adults — causes, approach to diagnosis, assessment and management**	Causes → GLIM → CGA → treatment ladder
3	**Nutritional assessment in older persons**	Screen → Diagnose → Grade
4	**Anorexia of ageing**	Physiology, hormones, management
5	**Obesity in the elderly and its management**	Obesity paradox, sarcopenic obesity, therapy
A	**Appendix — Rapid Revision Grid**	Numbers, criteria, trials, mnemonics

How to Use This Document

- Each answer follows the same architecture: **numbered high-yield points from the textbook** → **Latest Updates (2023–present)** with inline citations → **Current Practice Update** table contrasting textbook with current evidence → **Examiner's Tip** panel of verbatim mark-earning lines.
- Textbook content and recent evidence are kept strictly separate. Where they conflict, the conflict is shown explicitly in the comparison table.
- The five answers overlap deliberately — GLIM criteria, weight-loss thresholds and the ONS dose-duration rule recur, because examiners test them from several angles.
- The **Rapid Revision Appendix** at the end compiles every number, cut-off, criterion and named trial for night-before revision.

Question 1. Various Nutritional Disorders in the Elderly and Their Management

15 marks · Source: Pathy's 6th ed. (Ch. 12, 13, 14, 15, 16, 22); Essentials of Clinical Geriatrics 7th ed. (Ch. 3, 12)

20 High-Yield Points

DEFINITION AND SPECTRUM

1. **Definition.** As per **WHO**, malnutrition means **deficiencies, excesses or imbalances** in a person's energy and nutrient intake. It therefore includes **both undernutrition and obesity**.
2. **Spectrum of nutritional disorders in old age.** (a) **Protein-energy undernutrition**, (b) **Anorexia of ageing**, (c) **Cachexia**, (d) **Sarcopenia and sarcopenic obesity**, (e) **Micronutrient deficiencies**, (f) **Dehydration and electrolyte disorders**, (g) **Obesity**.
3. **Epidemiology.** Using MNA, overall prevalence is **22.8%** — highest in **rehabilitation units (50.5%)**, then **hospital (38.7%)**, lowest in **community (5.8%)**. European meta-analysis: community 6%, hospital 22%, nursing home 17.5%, **long-term care 28.7%**. Involuntary weight loss occurs in **5–15% of community elders** and **27% of frail elders**.

ANOREXIA OF AGEING

4. **Anorexia of ageing.** A **physiological**, age-related fall in appetite and energy intake that exceeds the fall in energy expenditure, causing weight loss. Prevalence rises from **3.3% in the community to 33% in nursing homes**. **Loss of appetite is the key predictor of malnutrition.**
5. **Pathophysiology.** Four mechanisms — **loss of homeostatic regulation** (elders do not compensate after underfeeding), the **ageing gut** (myenteric plexus cell loss, early satiation, reduced sensitivity to distension), **declining taste and smell**, and **hormonal changes** — chiefly **raised CCK levels with increased sensitivity to CCK**; leptin, NPY and CART changes are also implicated.
6. **Pathological causes (all at least partly treatable). Depression** (2–10% community, higher in institutions), **dementia** (~2-fold risk of anorexia; up to **50% of institutionalised dementia patients have PEM**), **COPD, CHF** (anorexia in >40% of end-stage CHF), **CKD, cancer**, **drugs and polypharmacy**, **poor dentition and dysphagia**, immobility, tremor, poor vision, poverty and isolation. Use the **"MEALS ON WHEELS" mnemonic** to search for treatable causes.

CLASSIFICATION AND DIAGNOSIS OF UNDERNUTRITION

7. **Classification by inflammatory grade. Starvation** (pure protein-energy deficit, **CRP <3 mg/L**) → **inflammageing** (low–medium) → **cachexia** (medium-grade inflammation, disease-related) → **hypercatabolic malnutrition** (sepsis, trauma; high-grade inflammation).
8. **GLIM diagnostic criteria** — need **one phenotypic + one etiologic** criterion.
   - **Phenotypic:** non-volitional **weight loss >5% in 6 months or >10% beyond 6 months**, **low BMI**, **reduced muscle mass**.
   - **Etiologic: reduced food intake or assimilation**, **inflammation / disease burden**.
9. **Clinically significant weight loss. 5% in 1 month, 7.5% in 3 months, or 10% in 6 months**. **BMI <22 kg/m²** predicts higher one-year mortality and poorer function; **ideal BMI ranges are higher in older than younger adults**. Weight change is preferred to BMI because kyphosis makes height unreliable.
10. **Screening tools. MNA and MNA-SF** are standard: **12–14 normal, 8–11 at risk, 0–7 malnourished**; uses **BMI or calf circumference (<31 cm)**. MNA is **96% sensitive, 60% specific** for weight loss and is a **powerful prognostic factor**, especially in cancer. Alternatives: **SGA, SNAQ**.
11. **Laboratory assessment. Serum albumin** is practical but confounded by liver disease, proteinuria and protein-losing enteropathy; **prealbumin and retinol-binding protein are more sensitive**. **Total lymphocyte count** is a useful marker. **Anaemia is one of the earliest manifestations of protein-calorie malnutrition.**

MICRONUTRIENT DISORDERS

12. **Micronutrients of public health concern in elders. Vitamin D, calcium, B12, folate, B2, B6, vitamin C, vitamin E, vitamin A, choline, zinc, magnesium, potassium, selenium**. An energy intake **<1500 kcal/day generally guarantees micronutrient inadequacy** — hence the emphasis on **nutrient-dense foods**.
13. **Vitamin B12 deficiency. 10–30% of elders malabsorb food-bound B12** (atrophic gastritis); **pernicious anaemia in 1–2%**. Features: **macrocytic anaemia, fatigue, anorexia, weight loss, paraesthesiae, ataxia, depression, dementia, glossitis**. Diagnosis: **low B12 with raised MMA and homocysteine**; **oral high-dose is as effective as intramuscular**.
14. **Folate deficiency.** Stores are small, so deficiency develops in **weeks to months**. **Homocysteine raised, MMA**

normal (distinguishes it from B12 deficiency). Causes: alcohol, malabsorption, **methotrexate, sulfasalazine, trimethoprim, phenytoin**. Treat with **folic acid 1 mg/day**.

15. **Zinc and iron. Zinc deficiency** causes **anorexia through impaired taste and smell**, impaired immunity, poor collagen synthesis and wound healing, diarrhoea and confusion; common in diabetics. **Iron deficiency is less common than in the young** — always exclude **chronic GI blood loss** and distinguish from anaemia of chronic disease (ferritin, CRP).
16. **Calcium and vitamin D.** Recommended intake after age 50: **calcium 1000–1200 mg/day, vitamin D 800 IU/day**; **upper limits 2000 mg and 4000 IU**. Treat **25(OH)D <10–12 ng/mL**; adequacy usually taken as **≥20 ng/mL**. Supplementation reduces **bone loss, fractures and falls** and improves **sarcopenia**.

DEHYDRATION, OBESITY AND MANAGEMENT

17. **Dehydration.** Very common and potentially lethal — **hospital mortality up to 50%**. Due to **reduced thirst**, a **fall in total body water from 60% to 50% after age 75**, and **reduced aquaporin-2 responsiveness to ADH**. Commonest precipitants: **infection, altered consciousness, cognitive impairment, diuretics**. **Acute loss ≥3% body mass or >1 kg/day** suggests dehydration. Advise **~1.5 L fluid daily**. Correct **hypernatraemia by <10 mEq/L/24 h** and **hyponatraemia by 6–8 mEq/L/24 h** to avoid cerebral oedema and osmotic demyelination.
18. **Obesity and sarcopenic obesity.** In those ≥65, **overweight (BMI 25–29) carries no excess mortality** and obesity only a modest increase. **Sarcopenic obesity = low lean mass with increased fat mass.** Weight loss is indicated **only when there is functional impairment or metabolic complication**, using a **modest deficit of 500–700 kcal/day plus resistance exercise** to protect muscle and bone. **Bariatric surgery only for highly selected older patients.**
19. **Management of undernutrition — five steps.**
    - **Step 1 — treat reversible causes** through **comprehensive geriatric assessment** (cognition, depression, drugs, dentition, swallowing, constipation, social and financial support).
    - **Step 2 — food-first measures:** oral and dental care, **texture modification**, **flavour enhancement**, feeding assistance, food fortification, meals-on-wheels.
    - **Step 3 — oral nutritional supplements (ONS):** meta-analysis of 42 trials showed **2.3% weight gain** and **reduced mortality (RR 0.74)** — benefit was significant when **≥400 kcal/day was given for ≥35 days** to **unwell, malnourished patients aged ≥75** in hospital or nursing homes. **No proven improvement in functional status or length of stay.**
    - **Step 4 — exercise: progressive resistance training 2–3 times/week** is the **primary intervention for sarcopenia**, improving strength, gait speed and chair rise even in the very old.
    - **Step 5 — enteral nutrition** if oral intake is inadequate; **parenteral** only if enteral is impossible. **PEG feeding in nursing-home residents with severe cognitive impairment did not improve 24-month survival, and aspiration pneumonia occurred at the same rate with or without tube feeding.**
20. **Palliative/orexigenic drugs and prognosis. Megestrol acetate** is the best studied (increases appetite, weight and quality of life; **oedema** is the significant adverse effect). **Corticosteroids** improve appetite without weight gain; **cyproheptadine** likewise; **cannabinoids (dronabinol, nabilone)** and **thalidomide (anti-TNF)** have limited evidence. **Unintentional weight loss is an independent predictor of mortality, functional decline, disability and institutionalisation** — it is a marker of poor prognosis, not merely a nutritional problem.

Latest Updates (2023–Present)

1. **GLIM criteria — 5-year update.** A 43-member working group reviewed >400 publications (2019–2024) and confirmed strong construct and predictive validity. No revisions were made to the weight loss, low BMI or reduced food intake/assimilation criteria; assessment of low muscle mass should be guided by experience and available technological resources, and clinical judgement may suffice for the inflammation/disease burden criterion. *(Jensen GL, et al. Clin Nutr / JPEN. 2025.)*
2. **Practical bedside GLIM muscle-mass surrogates validated.** Using calf circumference <31 cm in both sexes, or MUAC <23 cm in men and <22 cm in women, gave 84.6% and 83.4% accuracy against SGA, with sensitivity ~89% and ~84%. Directly relevant to Indian practice where DXA/BIA is unavailable. *(Shahraki Jazinaki M, et al. Front Nutr. 2024.)*
3. **GLIS — first global conceptual definition of sarcopenia.** Muscle mass (89.4%), muscle strength (93.1%) and muscle-specific strength (80.8%) were all accepted as components of sarcopenia, whereas impaired physical performance was accepted as an *outcome* rather than a component. This supersedes the textbook's EWGSOP-based framing. *(Kirk B, et al. Age Ageing. 2024.)*
4. **ESPEN practical guideline, geriatrics.** Eighty-two evidence-based recommendations: all older persons shall be routinely screened for malnutrition; oral intake supported by nursing interventions, counselling, food modification and ONS; enteral nutrition if oral is insufficient and parenteral if enteral is insufficient or impossible, when prognosis is favourable; dietary restrictions should generally be avoided. Low-intake dehydration should be diagnosed on serum

osmolality and corrected with hypotonic fluids given orally, subcutaneously or intravenously depending on severity; weight loss should be considered only in obesity with associated health problems, using moderate energy reduction with exercise. *(Volkert D, et al. Clin Nutr. 2022;41:958–989.)*

5. **ESPEN guideline on nutrition and hydration in dementia — Update 2024.** A full guideline update giving evidence-based recommendations for nutritional care in persons with dementia, to prevent and treat malnutrition and low-intake dehydration. *(Volkert D, et al. Clin Nutr. 2024.)*
6. **ESPEN practical guideline: nutritional support for polymorbid medical inpatients (2024).** Reaffirms screening-then-treat. The EFFORT trial showed that reaching ≥75% of estimated nutrition goals, versus lower achievement, significantly lowered the risk of adverse events and mortality. However, the 6-month secondary analysis found that while individualised nutritional support during hospital stay significantly reduced short-term mortality, there was no legacy effect on longer-term outcomes. *(ESPEN. Clin Nutr. 2024; Schuetz P, et al. Lancet. 2019; Clin Nutr. 2020.)*
7. **Vitamin D — Endocrine Society guideline 2024.** Empiric vitamin D supplementation is suggested in the general population aged 75 years and older because of the potential to lower mortality risk, with routine 25(OH)D testing suggested against in this group. For non-pregnant people over 50 in whom vitamin D is indicated, daily administration is suggested rather than intermittent high doses. *(Demay MB, et al. J Clin Endocrinol Metab. 2024.)*
8. **Vitamin D — USPSTF draft recommendation (Dec 2024).** Moderate certainty that vitamin D with or without calcium has no net benefit for primary prevention of fractures, and vitamin D has no net benefit for prevention of falls, in community-dwelling postmenopausal women and men aged 60 and older — a **D grade**. It does not apply to people taking these for other medical reasons, to those in assisted living or nursing homes, or to those diagnosed with osteoporosis or vitamin D deficiency. *(USPSTF Draft Recommendation Statement. 2024.)*
9. **Megestrol now discouraged.** The 2023 AGS Beers Criteria lists megestrol as **avoid** (strong recommendation, moderate quality) — minimal effect on weight, with increased risk of thrombotic events and possibly death in older adults. *(AGS Beers Criteria Update Expert Panel. J Am Geriatr Soc. 2023;71.)*
10. **Indian data — LASI Wave 1.** Among those aged 60 and above, 28% of men and 25% of women are underweight, while 25% of men and 37% of women aged 45–59 are overweight or obese; the urban population is less likely to be underweight and more than twice as likely to be overweight, and older adults from low economic and social strata are more likely to be underweight. Using a modified MUST, about 26% were classified as undernourished high-risk, characterised by low BMI, recent hospital admission and food-insecure households. *(Khan J, et al. Sci Rep. 2023;13; LASI analysis. BMC Public Health. 2023.)*
11. **WHO BMI cut-offs may misclassify Indian elders.** CART analysis of LASI data derived age-appropriate cut-offs — the underweight threshold fell from <18.5 to <17.4 for those aged 60–74 and <13.3 for those 75 and above. *(Singh A, Chattopadhyay A. Sci Rep. 2024;14.)*
12. **ICMR-NIN Dietary Guidelines for Indians 2024.** Seventeen guidelines addressing India's double burden of malnutrition, introducing the **"My Plate for the Day"** model and integrating RDA 2020 (safe protein requirement **0.83 g/kg/day**, EAR 0.66 g/kg/day, with **DIAAS** replacing PDCAAS for protein quality). The guidelines discourage the use of protein supplements and high-protein concentrates for muscle building, citing bone mineral loss and kidney damage with prolonged intake. *(ICMR-NIN. Dietary Guidelines for Indians. 2024.)*
13. **GLP-1 receptor agonists and the older adult — a new nutritional concern.** GLP-1 RAs effectively induce weight loss, but concomitant muscle mass loss is common, raising concerns about sarcopenia and sarcopenic obesity in older adults. A meta-analysis of eight RCTs found participants regained 9.69 kg over 48–52 weeks after stopping semaglutide or tirzepatide. Short-to-mid-term trials show handgrip strength is statistically preserved despite reductions in lean soft tissue, but longitudinal and retrospective studies in older adults with type 2 diabetes report reduced handgrip strength and accelerated sarcopenia with prolonged semaglutide use. **Practical implication: combine with adequate protein and resistance training in elders.** *(Curr Nutr Rep. 2026; Maturitas. 2025.)*

Current Practice Update

Textbook Position (Pathy's 2022 / Essentials 7e)	Current Evidence-Based Practice (2023–present)
GLIM criteria described as newly established (2019)	**GLIM 5-year update 2025** confirms validity; criteria unchanged; muscle mass measured by whatever tool is available, **calf circumference/MUAC acceptable**
Sarcopenia framed by EWGSOP; screening based on muscle performance	**GLIS 2024** — muscle mass + strength + **muscle-specific strength** are components; **physical performance is an outcome, not a component**
Vitamin D supplementation reduces falls and fractures	**USPSTF 2024 (draft, D grade)**: no net benefit for falls/fractures in healthy community-dwelling elders; **Endocrine Society 2024** still supports **empiric vitamin D ≥75 years** for mortality and **against routine 25(OH)D testing**
Megestrol acetate is "the most widely studied" orexigenic and	**AGS Beers 2023: AVOID** — minimal weight effect, thrombotic risk and

improves appetite, weight and QoL	possible increased mortality
Protein requirement stated as 0.5 to >1.0 g/kg/day (conflicting data)	**1.0–1.2 g/kg/day** in healthy elders and **1.2–1.5 g/kg/day** in acute/chronic illness (ESPEN 2022); **ICMR-NIN 0.83 g/kg/day** is a population safe level, not a therapeutic target
Serum albumin used as a practical indicator of malnutrition	Albumin/prealbumin now regarded as **inflammatory (prognostic) markers**, not nutritional markers; **not part of GLIM**
Obesity managed with lifestyle; drug therapy data "insufficient" in elders	**GLP-1 RAs now widely used**, but carry a **lean-mass loss and weight-regain risk**; must be paired with protein + resistance exercise
WHO BMI cut-offs applied uniformly	**Indian LASI-derived age-specific BMI cut-offs (2024)** perform better in those ≥60
Dehydration diagnosed clinically	**ESPEN**: diagnose low-intake dehydration on **serum osmolality**; correct with **hypotonic fluids**

Examiner's Tip — Mark-Earning Lines

- "Malnutrition in the elderly is a **geriatric syndrome**, not a single disease — it requires comprehensive geriatric assessment, not simply calorie counting."
- "**Anorexia of ageing** is physiological; **protein-energy malnutrition** is pathological. The clinical skill lies in separating the two."
- "Diagnosis follows a **two-step approach: screen with MNA-SF, then diagnose with GLIM** (one phenotypic + one etiologic criterion)."
- "**Unintentional weight loss of 5% in 1 month, 7.5% in 3 months or 10% in 6 months** is clinically significant and mandates evaluation."
- "ONS reduce mortality only when given at **≥400 kcal/day for ≥35 days** to genuinely malnourished, unwell elders — dose and duration matter."
- "**PEG feeding in advanced dementia does not improve survival and does not reduce aspiration pneumonia** — the decision belongs in an advance care planning discussion."
- "**Resistance exercise, not nutrition alone**, is the primary intervention for sarcopenia; nutrition is permissive, exercise is the stimulus."
- "In India, **LASI shows roughly one in four elders is underweight**, while overweight is rising — the **double burden of malnutrition** defines Indian geriatric nutrition."

Question 2. Malnutrition in Older Adults — Causes, Approach to Diagnosis, Assessment and Management

15 marks · Source: Pathy's 6th ed. (Ch. 12, 13, 14, 16); Essentials of Clinical Geriatrics 7th ed. (Ch. 3, 10, 12)

20 High-Yield Points

DEFINITION AND CONCEPT

1. **Definition.** As per **WHO**, malnutrition means **deficiencies, excesses or imbalances** in energy and nutrient intake. In geriatric practice, "malnutrition" and **undernutrition (protein-energy malnutrition)** are used interchangeably.
2. **It is a geriatric syndrome, not a single disease.** Like falls or delirium, it is **multifactorial with poor outcomes** and needs a multi-domain approach. Three overlapping presentations exist: **starvation** (pure intake deficit, responds to refeeding), **cachexia** (inflammation-driven, does not respond to refeeding alone), and **sarcopenia** (loss of muscle mass and strength, may occur with normal weight).
3. **Epidemiology.** Using MNA, overall prevalence is **22.8%** — **rehabilitation 50.5%**, **hospital 38.7%**, **community 5.8%**. European meta-analysis: **long-term care 28.7%, rehabilitation 29.4%, hospital 22%, nursing home 17.5%, community 6%**. People aged >70 consume about **one-third fewer calories** than the young, and **16–18% of community elders eat <1000 kcal/day**.

CAUSES

4. **Physiological — the anorexia of ageing.** (a) **Loss of homeostatic regulation** — elders fail to regain weight after underfeeding; (b) **ageing gut** — impaired **fundic accommodation** (raised nitric oxide), **delayed gastric emptying**, early satiation; (c) **declining senses** — over **50% aged 65–80 and >75% aged over 80** have measurable olfactory decline, and **46% of those with olfactory dysfunction have a change in appetite or weight**; reduced **sensory-specific satiety** narrows dietary variety; (d) **hormonal** — raised **CCK (levels + sensitivity), leptin, insulin and PYY**.
5. **Medical and psychiatric causes. Depression** (2–10% of community elders, far more in institutions; the commonest reversible cause), **dementia** (≈2-fold risk of anorexia; up to **50% of institutionalised dementia patients have PEM**), **COPD**, **CHF** (anorexia in >40% of end-stage), **CKD/dialysis (30–40%)**, **cancer**, **chronic infection**, **hyperthyroidism**, **malabsorption**, **constipation and faecal impaction**, **H. pylori infection**.
6. **Oral, swallowing and GI causes. Edentulism and ill-fitting dentures** (denture wearers have poorer protein intake), **xerostomia**, **oral candidiasis**, **dysphagia** (cranial nerves V, VII, IX, X, XI; worsened by stroke and dementia), **atrophic gastritis with hypochlorhydria** impairing B12 and iron absorption.
7. **Functional and social causes. Immobility (stroke), tremor (Parkinson's), impaired vision** all limit shopping, cooking and self-feeding. **Poverty and food insecurity**, **living alone** (elders eat up to **50% more when eating in company**), **social isolation**, institutionalisation, failure of facilities to cater to **ethnic food preferences**, and unnecessary **restrictive therapeutic diets**.
8. **Iatrogenic causes — polypharmacy.** More than **250 prescribed drugs alter taste**. **Digoxin and chemotherapy** cause nausea and anorexia; **aluminium/magnesium hydroxide antacids deplete phosphate and potassium** causing weakness and anorexia; **penicillamine causes zinc depletion and loss of taste**; **high-fibre diets cause excess satiety**.
9. **Bedside mnemonic — "MEALS ON WHEELS"** (recommended by Pathy's to find treatable causes):
   Medications · **E**motional (depression) · **A**norexia tardive/alcoholism · **L**ate-life paranoia · **S**wallowing disorders · **O**ral factors · **N**o money · **W**andering/dementia · **H**yper/hypothyroidism, hyperparathyroidism, hypoadrenalism · **E**nteric (malabsorption) · **E**ating problems (unable to self-feed) · **L**ow-salt/low-cholesterol diets · **S**ocial isolation, **S**tones.
10. **Consequences (why it matters). ↓Muscle mass and strength → falls; ↓bone mass → fractures; immune dysfunction → infection and pneumonia; anaemia; poor wound healing and pressure ulcers** (prevalence up to **30% in long-term care, 19% in home care**); **cognitive impairment; delayed surgical recovery; longer hospital stay; more admissions; increased mortality**. **PEM is a strong independent predictor of mortality** in community, nursing home and hospital settings, and the risk is amplified by renal failure, cardiac failure and stroke.

APPROACH TO DIAGNOSIS — A THREE-STEP SEQUENCE

11. **Step 1 — SCREEN every older person.** Use **MNA-SF**: **12–14 normal, 8–11 at risk, 0–7 malnourished**; body mass entered as **BMI or calf circumference (<31 cm)**. MNA is **96% sensitive and 60% specific** for weight loss and is a **powerful independent prognostic factor**. Alternatives: **MUST, NRS-2002, SNAQ, SGA**. In nursing homes, **weigh monthly** — persistent weight loss must trigger a search for treatable causes.
12. **Step 2 — DIAGNOSE using the GLIM criteria** (need **one phenotypic + one etiologic** criterion):

Phenotypic (any one)	Etiologic (any one)

Weight loss >5% in 6 months, or >10% beyond 6 months	**Reduced food intake** — ≤50% of energy requirement for >1 week, any reduction for >2 weeks, or any chronic GI condition impairing assimilation
Low BMI — <20 if <70 y, <22 if >70 y; **Asia: <18.5 if <70 y, <20 if >70 y**	**Inflammation** — acute disease/injury, or chronic disease-related
Reduced muscle mass — by DXA, BIA, CT or MRI; where unavailable, **MAMC or calf circumference**; handgrip strength as a supportive measure	

Note: diagnosing malnutrition on low BMI alone, without an etiologic cause, overestimates prevalence.

13. **Step 3 — GRADE the mechanism using inflammation (CRP). CRP <3 mg/L → starvation** (responds to refeeding); **low–medium grade → inflammageing** (not itself malnutrition); **medium grade → cachexia** (organ failure, cancer, neurodegenerative disease); **CRP >100 mg/L → hypercatabolic malnutrition** (sepsis, trauma, ICU). This determines whether nutrition alone will work.
14. **Weight-loss thresholds worth quoting.** Loss of **5% in 1 month, 7.5% in 3 months or 10% in 6 months** is serious. **BMI <22 kg/m²** predicts higher one-year mortality and poorer function; **ideal BMI ranges are higher in older than younger adults**; **BMI below the 15th percentile** independently predicts 180-day mortality after hospitalisation.

ASSESSMENT

15. **Assessment is by comprehensive geriatric assessment (CGA).** Systematically cover — **cognition** (MMSE/MoCA), **depression** (GDS), **delirium** (CAM), **function** (Katz, Barthel, Lawton–Brody ADL/IADL), **falls and mobility** (TUG, unipodal stance, SPPB), **medication reconciliation and deprescribing**, **nutrition** (BMI, MNA, weight history, **swallowing test**, **constipation/faecal impaction screen**, **oral examination**), and **social and financial resources**. Each impaired domain has a matching action — meal-texture adaptation, dental treatment, housekeeper for shopping, meals-on-wheels, caregiver education.
16. **Anthropometry. Serial weight is the single most useful measurement** — kyphosis makes height and therefore BMI unreliable. Use **MUAC** when there is oedema (correlates with lean mass), **calf circumference**, **mid-arm muscle circumference**; estimate height by **knee-height formulae**; apply **percentage corrections in amputees**.
17. **Laboratory assessment. Serum albumin** is practical but confounded by **liver disease, proteinuria and protein-losing enteropathy**; **prealbumin and retinol-binding protein are more sensitive**. Add **CRP** (grades inflammation), **total lymphocyte count**, **haemoglobin** (**anaemia is one of the earliest manifestations of PEM**), **creatinine clearance**, **B12, folate, 25(OH)D, iron studies, calcium, TSH**, and **age-appropriate cancer screening** where weight loss is unexplained.
18. **Body composition and muscle function. DXA or BIA** for fat-free mass index; **CT at the L3 slice** for muscle index in cancer patients; where unavailable, **calf circumference is the accepted surrogate**. Function is measured by **handgrip strength, gait speed, chair-rise and SPPB** — these predict outcome better than mass alone.

MANAGEMENT

19. **Principles and targets.**
    - **First, treat reversible causes** — depression, drugs, dysphagia, dentition, pain, nausea, constipation, infection, thyroid disease. Treating depression alone produces weight gain.
    - **Set requirements:** approximately **30–35 kcal/kg/day**, **protein 1.0–1.5 g/kg/day**, **fluid ~1 mL/kcal (about 1.5 L/day)**.
    - **Avoid unnecessary therapeutic diets** (low-salt, low-fat, diabetic) in the frail underweight elder — the risk of undernutrition outweighs the benefit.
20. **Stepwise treatment ladder.**
    - **(a) Food-first and nursing measures** — oral and dental care, **texture modification**, **flavour enhancement** (improves intake and nutritional status in nursing-home residents), fortified/nutrient-dense foods, small frequent meals, **feeding assistance**, **eating in company**, meals-on-wheels and social support.
    - **(b) Oral nutritional supplements (ONS)** — meta-analysis of 42 trials showed **2.3% weight gain** and **reduced mortality (RR 0.74, 95% CI 0.59–0.92)**; benefit was consistent when **≥400 kcal/day was given for ≥35 days** to **unwell, malnourished patients aged ≥75** in hospital or nursing homes. ONS also **reduce pressure-ulcer incidence**. **No proven benefit on functional status or length of stay.**
    - **(c) Exercise — progressive resistance training 2–3 times/week** is the **primary intervention for sarcopenia**; essential amino acids stimulate muscle protein synthesis better than intact protein. Combined nutrition + early mobilisation in hospital **prevents loss of functional independence** even without weight gain.
    - **(d) Enteral nutrition** if oral intake remains inadequate and prognosis is favourable; **parenteral only if enteral is impossible** (higher morbidity than enteral). **PEG in nursing-home residents with severe cognitive**

impairment does not improve 24-month survival, and aspiration pneumonia occurs at the same rate with or without tube feeding — this is an advance-care-planning discussion, not a nutritional one.

- **(e) Orexigenic drugs — palliative only.** Megestrol acetate is the best studied (appetite, weight, quality of life; **oedema**); corticosteroids and cyproheptadine improve appetite without weight gain; cannabinoids and thalidomide have limited evidence.
- **(f) Monitor** weight, intake charts and function; **watch for refeeding syndrome** (falling phosphate, potassium, magnesium) when replenishing the severely depleted.

Latest Updates (2023–Present)

1. **Global burden quantified.** A meta-analysis of 98 studies (n = 79,976) found the global prevalence of malnutrition in older adults to be **18.6% (95% CI 16.4–21.1%)**, highest in Africa at 35.7% followed by the Americas at 20.3%; by instrument, the highest prevalence was 39.9% using NRS-2002. *(Salari N, et al. Public Health in Practice. 2025;9:100583.)*
2. **GLIM criteria — 5-year update.** A 43-member working group reviewed >400 publications (2019–2024). No revisions were made to the weight loss, low BMI or reduced food intake/assimilation criteria; assessment of low muscle mass should be guided by experience and available technological resources, and clinical judgement may suffice for the inflammation/disease burden criterion. Construct and predictive validity are now strong. *(Jensen GL, et al. Clin Nutr / JPEN. 2025.)*
3. **Bedside muscle-mass surrogates now validated — directly usable in Indian practice.** Applying calf circumference <31 cm in both sexes, or MUAC <23 cm in men and <22 cm in women, gave 84.6% and 83.4% accuracy against SGA, with sensitivity ~89% and ~84% and specificity ~81–83%. *(Shahraki Jazinaki M, et al. Front Nutr. 2024;11:1438158.)*
4. **ONS in hospitalised older people — Cochrane individual-participant network meta-analysis.** In older hospitalised people at risk of or with malnutrition, oral nutritional supplements *may* reduce mortality and serious adverse events compared with control at 30 days after randomisation, with little or no difference for other outcomes; the overall certainty of evidence was **low to very low**. This tempers the older, more confident textbook claim. *(Kiesswetter E, et al. Cochrane Database Syst Rev. 2026;CD015468.)*
5. **Individualised in-hospital nutrition works, but the effect does not persist.** Reaching ≥75% of estimated nutrition goals, versus lower achievement, significantly lowered the risk of adverse events and mortality; yet while individualised nutritional support during the hospital stay significantly reduced short-term mortality, there was **no legacy effect on longer-term outcomes**. **Implication: nutritional care must be continued after discharge.** *(Schuetz P, et al. Lancet. 2019; Clin Nutr. 2020; ESPEN practical guideline, polymorbid medical inpatients. Clin Nutr. 2024.)*
6. **ESPEN geriatric guideline — current management standard.** All older persons shall be routinely screened for malnutrition; oral intake is supported by nursing interventions, counselling, food modification and ONS; enteral nutrition is initiated if oral is insufficient and parenteral if enteral is insufficient or impossible, provided the general prognosis is favourable; dietary restrictions should generally be avoided. Low-intake dehydration should be diagnosed on serum osmolality and corrected rapidly with hypotonic fluids given as drinks, subcutaneously or intravenously depending on severity. A dedicated ESPEN update for dementia was published in 2024. *(Volkert D, et al. Clin Nutr. 2022;41:958–989; Volkert D, et al. Clin Nutr. 2024.)*
7. **Sarcopenia reframed — GLIS 2024.** Muscle mass, muscle strength and muscle-specific strength were all accepted as components of sarcopenia, whereas impaired physical performance was accepted as an outcome rather than a component. *(Kirk B, et al. Age Ageing. 2024;53:afae052.)*
8. **Megestrol is no longer acceptable in older adults.** The 2023 AGS Beers Criteria grades it **avoid** (strong recommendation) — minimal effect on weight, with increased risk of thrombotic events and possibly death in older adults. *(AGS Beers Criteria Update Expert Panel. J Am Geriatr Soc. 2023;71.)*
9. **Indian data.** In LASI, 28% of men and 25% of women aged 60 and above are underweight; the urban population is less likely to be underweight, and older adults from low economic and social strata are more likely to be underweight. Using a modified MUST, about 26% of respondents aged 45+ fell into the undernourished high-risk group, characterised by low BMI, recent hospital admission and food-insecure households. CART analysis of LASI further suggests age-specific BMI cut-offs — the underweight threshold falling from <18.5 to <17.4 for ages 60–74 and <13.3 for those 75 and above. *(Khan J, et al. Sci Rep. 2023;13:12820; BMC Public Health. 2023; Singh A, Chattopadhyay A. Sci Rep. 2024;14:15200.)*
10. **ICMR-NIN Dietary Guidelines for Indians 2024.** Seventeen guidelines addressing India's double burden of malnutrition, with the **"My Plate for the Day"** model and RDA 2020 values (**safe protein 0.83 g/kg/day**, EAR 0.66 g/kg/day, DIAAS replacing PDCAAS). The guidelines discourage protein supplements and high-protein concentrates for muscle building, citing bone mineral loss and kidney damage with prolonged intake. *(ICMR-NIN. Dietary Guidelines for Indians. 2024.)*

Current Practice Update

Textbook Position (Pathy's 2022 / Essentials 7e)	Current Evidence-Based Practice (2023-present)
GLIM described as newly established (2019)	**GLIM 5-year update 2025** — criteria unchanged and now validated across >400 studies; **any available method** for muscle mass, **clinical judgement** acceptable for inflammation
Muscle mass ideally by DXA/BIA/CT	**Calf circumference <31 cm** and **MUAC <23 cm (M) / <22 cm (F)** formally validated as accurate surrogates — the practical option in Indian wards
ONS reduce mortality (RR 0.74) based on 2009 Cochrane review of 42 trials	**Cochrane IPD network meta-analysis 2026**: ONS *may* reduce 30-day mortality and serious adverse events, but certainty is **low to very low** — benefit is real but weaker than previously stated
Nutritional support during admission improves outcomes	Benefit is **confined to the hospital episode** — **no legacy effect at 6 months**; current emphasis is on **continuing nutrition after discharge**
Megestrol acetate is the best-studied and most widely used orexigenic	**AGS Beers 2023: AVOID** — minimal weight effect, thrombotic risk, possible increased mortality
Protein requirement quoted as 0.5 to >1.0 g/kg/day (conflicting)	**1.0–1.2 g/kg/day** in healthy elders, **1.2–1.5 g/kg/day** in acute or chronic illness (ESPEN 2022); the **ICMR-NIN 0.83 g/kg/day** figure is a population safe intake, **not a therapeutic target**
Albumin presented as "a practical indicator of malnutrition"	Albumin and prealbumin are now regarded as **inflammatory and prognostic markers**, not nutritional markers, and are **not part of GLIM**
Sarcopenia screening based on low muscle performance (EWGSOP)	**GLIS 2024** — physical performance is an **outcome**, not a component; **muscle-specific strength** added as a component
WHO BMI cut-offs applied to all	**Asian GLIM cut-offs (<18.5 if <70 y, <20 if >70 y)** and **Indian LASI-derived cut-offs (2024)** perform better in our population

Examiner's Tip — Mark-Earning Lines

- "Malnutrition in the older adult is a **multifactorial geriatric syndrome with poor outcomes** — it demands comprehensive geriatric assessment, not calorie counting."
- "The approach is a **three-step sequence: SCREEN (MNA-SF) → DIAGNOSE (GLIM) → GRADE the mechanism by inflammation (CRP)**."
- "**GLIM requires one phenotypic plus one etiologic criterion**; for India, remember the **Asian BMI cut-offs — <18.5 below 70 years and <20 above 70 years**."
- "**Weight loss of 5% in 1 month, 7.5% in 3 months or 10% in 6 months** is clinically significant and mandates evaluation."
- "Use **MEALS ON WHEELS** to systematically uncover reversible causes — **depression and drugs are the two most commonly missed and most treatable**."
- "**Serial weight, not BMI**, is the most reliable anthropometric measure in the elderly because kyphosis makes height unreliable."
- "**Distinguish starvation from cachexia** — starvation responds to refeeding, cachexia does not; the discriminator at the bedside is **CRP**."
- "ONS work when the **dose and duration are right — ≥400 kcal/day for ≥35 days** in genuinely malnourished, unwell elders."
- "**Resistance exercise is the stimulus; nutrition is only permissive** — neither works well alone in sarcopenia."
- "**PEG feeding in advanced dementia does not prolong survival and does not prevent aspiration pneumonia.**"

Question 3. Nutritional Assessment in Older Persons

15 marks · Source: Pathy's 6th ed. (Ch. 12, 13, 14, 16 and Function Assessment Scales appendix); Essentials of Clinical Geriatrics 7th ed. (Ch. 3, Tables 3-11 to 3-14)

20 High-Yield Points

CONCEPT AND FRAMEWORK

1. **Purpose.** Nutritional assessment answers four questions (Essentials, Table 3-12): **Is there reason to suspect malnutrition? Of which nutrient(s) and to what extent? What is the pathophysiological mechanism? What is the underlying etiology?**
2. **Why assessment is different in the elderly.** No single parameter is adequate — **anthropometric variables are not highly correlated with biochemical or haematologic indicators**, and **the effect of ageing on lean body mass is so great that lean mass alone is a poor reflection of nutritional status**. Assessment must therefore be **multi-parameter and multi-domain**.
3. **The standard two-step (now three-step) approach. Step 1 — SCREEN** every older person with a validated tool. **Step 2 — DIAGNOSE** using **GLIM criteria**. **Step 3 — GRADE severity and identify the mechanism**, then plan intervention.

STEP 1 — SCREENING TOOLS

4. **Mini Nutritional Assessment (MNA) — the geriatric standard.** The full MNA is **multidimensional and identifies malnourished elders or those at risk, whatever the cause**. **MNA-SF** has equivalent performance: **12–14 = normal, 8–11 = at risk, 0–7 = malnourished**. Body mass is entered as **BMI (0 = <19, 1 = 19–20.9, 2 = 21–22.9, 3 = ≥23)** or, if BMI is unavailable, **calf circumference (0 = <31 cm, 3 = ≥31 cm)**.
5. **MNA components (six domains). (A)** decline in food intake over 3 months, **(B)** weight loss over 3 months, **(C)** mobility, **(D)** psychological stress or acute illness in 3 months, **(E)** neuropsychological problems (dementia/depression), **(F)** BMI or calf circumference.
6. **Performance and prognostic value.** MNA is **96% sensitive and 60% specific** for body weight loss. Subjects with a normal MNA had **lower mortality (RR 0.35, 95% CI 0.18–0.66)** than those with an abnormal score. Those "at risk" had **more acute illness, more assistance needs and more weight loss**. MNA is a **powerful independent prognostic factor**, especially in cancer, and can assess **cancer cachexia**. A **six-item version** has equivalent predictive value.
7. **Other validated tools. SGA** (predicts mortality but classifies more patients as well-nourished than MNA — those "well-nourished" by MNA had better 3-year survival), **MUST**, **NRS-2002**, and the **Geriatric Nutritional Risk Index (GNRI)**, which predicts healthcare costs and hospitalisation.
8. **SNAQ (Simplified Nutritional Appetite Questionnaire)** — four questions on **appetite, early satiety, taste of food, and number of meals per day**, each scored 1–5. **A score <14 indicates significant risk of weight loss.** Recommended by Pathy's as a rapid appetite screen.
9. **Screening question in routine geriatric review** (Pathy's Function Assessment Scales): ask **"Have you lost weight?"** and record **height and weight**; if positive, proceed to **BMI, MNA, SNAQ**, and use the **"MEALS ON WHEELS" mnemonic** to look for treatable causes.

STEP 2 — DIAGNOSIS BY GLIM

10. **GLIM criteria** — malnutrition requires **≥1 phenotypic + ≥1 etiologic** criterion:

Phenotypic	Etiologic
Weight loss **>5% in 6 months** or **>10% beyond 6 months**	Reduced food intake — **≤50% of energy requirement for >1 week**, any reduction for **>2 weeks**, or any **chronic GI condition** impairing assimilation/absorption
Low BMI — **<20 if <70 y, <22 if >70 y**; **Asia: <18.5 if <70 y, <20 if >70 y**	Inflammation — **acute disease/injury** or **chronic disease-related**
Reduced muscle mass — by validated body-composition technique; where unavailable, **mid-arm muscle or calf circumference**; **handgrip strength as a supportive measure**	

11. **Severity grading (GLIM Stage 1 vs Stage 2). Stage 1 (moderate):** weight loss **5–10% in 6 months or 10–20% beyond 6 months**; BMI **<20 if <70 y, <22 if ≥70 y**; mild–moderate muscle deficit. **Stage 2 (severe):** weight loss **>10% in 6 months or >20% beyond 6 months**; BMI **<18.5 if <70 y, <20 if ≥70 y**; severe muscle deficit.
12. **Caution. Diagnosing malnutrition on low BMI alone, without identifying a cause, overestimates prevalence.** Always pair a phenotypic with an etiologic criterion.

STEP 3 — COMPONENTS OF FULL ASSESSMENT

13. **History. Weight history** (magnitude and rate of loss — **5% in 1 month, 7.5% in 3 months, 10% in 6 months is serious**), **appetite** (SNAQ), chewing and swallowing difficulty, **dietary history**, food fads and restrictive diets, alcohol, **drug review** (>250 drugs alter taste), depression, dementia, **social and financial circumstances**, ability to shop and cook, living alone.
14. **Physical examination.** General appearance and muscle wasting; **oral examination** — dentition, ill-fitting dentures, xerostomia, candidiasis, dental plaque, **loss of occluding pairs**; **swallowing test**; abdominal exam for **faecal impaction**; skin for **pressure ulcers and poor wound healing**; **Braden scale** for pressure-ulcer risk; signs of specific deficiencies (glossitis, cheilosis, neuropathy, ataxia, ecchymoses, oedema).
    Pathy's dental screening tool: dry mouth (2), eating difficulty (1), no dental care within 2 years (1), tooth/mouth pain (2), change in food selection (1), oral lesions (2) — **≥3 points warrants dental referral.**
15. **Anthropometry — what each measure reflects** (Essentials, Table 3-11):

Measure	Reflects
Weight	Global nutritional status
BMI	Total body fat
Triceps and subscapular skinfolds	Percentage body fat
Waist–hip ratio	Central adiposity
Upper-arm (mid-arm) circumference	**Lean body mass** — particularly useful in **oedematous patients where weight misleads**
Calf circumference (<31 cm)	Low muscle mass surrogate

16. **Practical anthropometric pitfalls and corrections.**
    - **Serial weight is preferred to BMI** — kyphosis makes height unreliable; also consider oedema.
    - **Height from knee height:** Men = (2.02 × knee height cm) − (0.04 × age) + 64.19; Women = (1.83 × knee height cm) − (0.24 × age) + 84.88.
    - **Amputee weight corrections:** single below-knee **6%**, at knee **9%**, above knee **15%**, single arm **6.5%**, arm below elbow **3.6%**.
    - **BMI <22 kg/m²** predicts higher one-year mortality; **BMI below the 15th percentile** independently predicts 180-day post-hospitalisation mortality.
17. **Body composition and muscle assessment. DXA** (fat-free mass index, appendicular lean mass index), **BIA**, **CT at the L3 slice** (muscle index, used in cancer follow-up), **MRI**. GLIM/sarcopenia cut-offs: **FFMI <17 (men), <15 (women) kg/m²**; **ASMI <7.0 (men), <5.4–5.7 (women) kg/m²**. Where these are unavailable, **calf circumference or MAMC is acceptable**.
18. **Functional assessment (increasingly the anchor). Handgrip strength**, **gait speed**, **Timed Up and Go**, **unipodal stance**, **chair rise**, and **SPPB** (predicts disability). Function correlates with outcome better than mass alone.
19. **Biochemical assessment.**
    - **Serum albumin** — practical, but **liver disease, proteinuria and protein-losing enteropathy must be excluded**; a **normal or raised albumin does not exclude malnutrition**.
    - **Prealbumin (thyroxine-binding) and retinol-binding protein are more sensitive** than albumin and transferrin.
    - **Haemoglobin** — **anaemia is one of the earliest manifestations of protein-calorie malnutrition**.
    - **Total lymphocyte count** — a good marker of nutritional problems.
    - **CRP** — grades inflammation and separates **starvation (CRP <3 mg/L)** from **cachexia (medium grade)** and **hypercatabolism (CRP >100 mg/L)**.
    - **Micronutrients:** B12 with **MMA and homocysteine**, folate (**red-cell folate reflects the preceding 120 days**), **25(OH)D**, ferritin with CRP, calcium, TSH. **Zinc measurement is unreliable** — cytokines lower serum zinc; leukocyte or hair zinc may help.
    - Also: **creatinine clearance**, blood count, and **targeted cancer screening** where weight loss is unexplained.
20. **Dietary intake and multidomain assessment.**
    - **Intake:** 24-hour recall, food-frequency questionnaire, food diary, plate-waste/intake charts. Limitations in elders: **recall and misclassification bias**, worse with cognitive impairment — hence the value of **objective nutrient biomarkers**.
    - **CGA-based assessment (Pathy's Table 14.3)** — cognition (**MMSE/MoCA**), depression (**GDS**), delirium (**CAM**),

falls (**TUG, SPPB**), function (**Katz, Barthel, Lawton–Brody**), morbidity and drug review, nutrition (BMI, MNA, weight history, swallowing, constipation screen, oral exam), and **social/socioeconomic domains**.

- **Monitoring:** weigh nursing-home residents **monthly**; **persistent weight loss must prompt a search for treatable medical, psychiatric and functional conditions**.

Latest Updates (2023–Present)

1. **GLIM — 5-year update.** Following review of >400 publications from 2019–2024, no revisions were made to the weight loss, low BMI or reduced food intake/assimilation criteria; assessment of low muscle mass should be guided by experience and available technological resources, and clinical judgement may suffice for the inflammation/disease burden criterion. Severity-grading thresholds are unchanged, but a broad distinction between moderate and severe reduction of muscle mass is not yet possible because only some methods allow that level of differentiation, and further research is needed to secure consensus reference BMI data for Asian populations. *(Jensen GL, et al. Clin Nutr / JPEN. 2025.)*
2. **Bedside surrogates for muscle mass formally validated.** Applying calf circumference <31 cm in both sexes, or MUAC <23 cm in men and <22 cm in women, gave 84.6% and 83.4% accuracy against SGA, with sensitivity ~89% and ~84% and specificity ~81–83%. Directly applicable in Indian wards without DXA or BIA. *(Shahraki Jazinaki M, et al. Front Nutr. 2024;11:1438158.)*
3. **Choice of tool changes the answer.** A meta-analysis of 98 studies (n = 79,976) found a global malnutrition prevalence of 18.6% (95% CI 16.4–21.1%) in older adults, with the highest prevalence — 39.9% — obtained using NRS-2002. **Examination point: always state which instrument was used.** *(Salari N, et al. Public Health in Practice. 2025;9:100583.)*
4. **Sarcopenia assessment reframed — GLIS 2024.** Muscle mass, muscle strength and muscle-specific strength were all accepted as components of sarcopenia, whereas impaired physical performance was accepted as an outcome rather than a component. *(Kirk B, et al. Age Ageing. 2024;53:afae052.)*
5. **BIA phase angle and BIVA — emerging bedside tools.** Phase angle is a highly reliable indicator of skeletal muscle quality because it is calculated directly from reactance and resistance and does not depend on an estimation formula. In hospitalised older patients, standardised phase angle carried prognostic significance and BIVA usefully estimated body-composition/nutrition phenotypes; patients with low standardised phase angle had significantly lower BMI and fat mass. BIVA also discriminated sarcopenic from non-sarcopenic elderly in both sexes, supporting 50 kHz phase-sensitive measurements as biomarkers of hydration, catabolic state and nutritional status. *(Front Aging / Clin Nutr cohort studies. 2024–2025; Sensors. 2023;23:7090.)*
6. **Muscle ultrasound entering nutritional assessment.** In hospitalised older adults, ultrasound and bioelectrical impedance analysis are reliable, non-invasive and reproducible for quantitative and qualitative muscle assessment, with rectus femoris cross-sectional area, thickness, echogenicity and compressibility measured combinedly for sarcopenia diagnosis. *(Nutrients. 2024;16:2429.)*
7. **ESPEN — screening is mandatory, not optional.** All older persons shall routinely be screened for malnutrition in order to identify existing risk early. Low-intake dehydration should be diagnosed on the basis of serum osmolality, not clinical signs alone. A dedicated ESPEN update for dementia followed in 2024. *(Volkert D, et al. Clin Nutr. 2022;41:958–989; Volkert D, et al. Clin Nutr. 2024.)*
8. **Indian BMI cut-offs need adjustment.** CART analysis of LASI data lowered the underweight cut-off from <18.5 to <17.4 for ages 60–74 and <13.3 for those 75 and above, based on cardio-metabolic outcomes. Set against LASI prevalence — 28% of men and 25% of women aged 60 and above are underweight, with those from low economic and social strata more likely to be underweight. *(Singh A, Chattopadhyay A. Sci Rep. 2024;14:15200; Khan J, et al. Sci Rep. 2023;13:12820.)*
9. **Indian dietary reference values for interpreting intake.** ICMR-NIN Dietary Guidelines for Indians 2024 with the "My Plate for the Day" model and RDA 2020 (safe protein 0.83 g/kg/day, EAR 0.66 g/kg/day; DIAAS replacing PDCAAS for protein quality). The guidelines discourage protein supplements and high-protein concentrates, citing bone mineral loss and kidney damage with prolonged intake. *(ICMR-NIN. Dietary Guidelines for Indians. 2024.)*

Current Practice Update

Textbook Position (Pathy's 2022 / Essentials 7e)	Current Evidence-Based Practice (2023-present)
"**Serum albumin is a practical indicator of malnutrition**"; prealbumin/RBP more sensitive	Albumin and prealbumin are now regarded as **inflammatory and prognostic markers, not nutritional markers**, and are **excluded from GLIM**; **CRP** is used only to grade inflammation
Muscle mass measurement "difficult in routine practice"; calf circumference proposed as a surrogate	**Calf circumference <31 cm** and **MUAC <23 cm (M) / <22 cm (F)** now have **formal validation data** (accuracy >83% vs SGA); GLIM 2025 explicitly permits **whatever method is locally available**
GLIM presented as a newly published consensus (2019)	**GLIM 5-year update 2025** — criteria confirmed, **severity grading unchanged**, validity established across >400 studies; **Asian BMI reference data still awaited**

Sarcopenia assessed by muscle mass + performance (EWGSOP)	**GLIS 2024** — performance is an **outcome**, not a component; **muscle-specific strength** added as a third component
Body composition by DXA, BIA, CT, MRI	**BIA-derived phase angle and BIVA**, and **muscle ultrasound (rectus femoris CSA, thickness, echogenicity)** are emerging as bedside, radiation-free adjuncts with independent prognostic value
WHO BMI cut-offs applied to all older adults	**Asian GLIM cut-offs (<18.5 if <70 y; <20 if >70 y)** and **Indian LASI-derived cut-offs (2024)** perform better in our population
Screening described as good practice	**ESPEN: routine screening of all older persons "shall" be performed** — now a standard of care; and the **choice of tool materially changes prevalence** (NRS-2002 gives the highest estimates)

Examiner's Tip — Mark-Earning Lines

- "Nutritional assessment in the elderly follows a **three-step sequence: SCREEN (MNA-SF) → DIAGNOSE (GLIM) → GRADE severity and mechanism (CRP)**."
- "**No single parameter suffices** — anthropometric measures correlate poorly with biochemical indices, and ageing alters lean body mass so much that it is a poor marker on its own."
- "**MNA-SF: 12–14 normal, 8–11 at risk, 0–7 malnourished**; use **calf circumference <31 cm** when BMI cannot be obtained."
- "MNA is **96% sensitive and 60% specific** — a good screen, not a diagnostic test."
- "**Severity grading: Stage 1 (moderate) = 5–10% loss in 6 months; Stage 2 (severe) = >10% loss in 6 months.**"
- "**Serial weight, not BMI**, is the most reliable anthropometric measure — use **knee-height formulae** when height cannot be measured."
- "**Mid-arm circumference is the measure of choice in oedema**, where weight misleads."
- "**Albumin is an inflammatory marker, not a nutritional marker** — the single commonest error in ward practice."
- "**Anaemia is one of the earliest manifestations of protein-calorie malnutrition** — it should prompt, not follow, nutritional assessment."

Question 4. Anorexia of Ageing

15 marks · Source: Pathy's 6th ed. Chapter 13 "The Anorexia of Aging", with Chapters 12 and 14; Essentials of Clinical Geriatrics 7th ed. (Ch. 12)

20 High-Yield Points

DEFINITION AND MAGNITUDE

1. **Definition.** In many older adults the **decrease in energy intake is greater than the decrease in energy expenditure**, so body weight is lost. This **physiological, age-related reduction in energy intake is termed the anorexia of ageing** (Morley, 1997).
2. **It is a geriatric syndrome.** Pathy's depicts it as a **multifactorial geriatric syndrome with poor outcomes**, arising from four groups of factors — **physiological changes, changes due to chronic disease, social determinants/environment, and iatrogenic causes/polypharmacy**.
3. **Physiological vs pathological.** The **physiological** component is intrinsic to ageing; the **pathological** component (disease, drugs, depression, dementia) is superimposed and is **at least partly treatable — hence recognition matters**.
4. **Magnitude of intake decline. Energy intake falls by ~30% between ages 20 and 80.** NHANES III: decline of **1321 kcal/day in men and 629 kcal/day in women**. New Mexico longitudinal study: **−25.1 kcal/day/year in men, −19.3 in women**. Swedish cohort (ages 70→76): **−610 kcal/day in men, −440 in women**. Older people eat **smaller meals, more slowly, with fewer snacks**, and consistently report **less hunger**.
5. **Prevalence.** Anorexia occurs in **3.3% of community-dwelling elders rising to 33% in nursing homes**. About **16–18% of community elders consume <1000 kcal/day**. **Loss of appetite is the single key predictor of malnutrition in older adults.**

PATHOPHYSIOLOGY — FOUR PHYSIOLOGICAL MECHANISMS

6. **(i) Loss of homeostatic regulation.** In Roberts' classic experiment, young and old men were underfed by **~750 kcal/day for 21 days**; on refeeding *ad libitum*, **young men over-ate and regained weight, whereas old men returned only to baseline intake and never regained the lost weight**. Consequently, after an anorectic insult (e.g. surgery), older adults **stay undernourished longer** and are more susceptible to superimposed illness.
7. **(ii) The ageing gut. Cell loss in the oesophageal myenteric plexus** and slowed visceral nerve conduction reduce sensory feeding stimuli. **Impaired receptive relaxation of the gastric fundus** — possibly due to **increased fundic nitric oxide** — causes **more rapid antral filling and distension → earlier satiety**, and contributes to **slower gastric emptying**; postprandial hunger is inversely related to the rate of gastric emptying.
8. **(iii) Declining senses.** Smell declines markedly after age 50 — **over 60% aged 65–80 and over 80% aged ≥80 have major reductions** (thinning epithelium, reduced mucus, poor regeneration of olfactory receptor cells). **46% of older adults with olfactory dysfunction report a change in appetite or weight.** Ageing also reduces **sensory-specific satiety** (the normal decline in pleasantness of a food after eating it), favouring a **less varied, monotonous diet** and hence **micronutrient deficiency**.
9. **(iv) Central neurotransmitters. Noradrenaline stimulates, serotonin inhibits, and dopamine has region-specific effects** on feeding. Ageing is associated with **increased satiating effects of serotonin**, without clear changes in dopamine or noradrenaline.
10. **Opioids — reduced feeding drive.** Elderly patients with **idiopathic senile anorexia have lower plasma and CSF β-endorphin** than age-matched controls. Morphine increases food intake in young but **not old** mice; naloxone reduces intake in young but not old rats; older men are less sensitive to naloxone's inhibitory effect on fluid intake. The **stimulatory opioid drive declines with age**.
11. **Cholecystokinin (CCK) — the strongest candidate.** CCK is a **physiological satiety hormone** released by intestinal fat and protein, which also **slows gastric emptying**. **Plasma CCK is higher in healthy older than young adults**, and **significantly higher in elderly people with idiopathic anorexia**. Crucially, **IV CCK-8 suppressed food intake twice as much in older than young adults (31% vs 15%, p = 0.02)**. The combination of **higher levels plus enhanced sensitivity** suggests CCK may cause the anorexia of ageing and raises the possibility of **CCK antagonists** as therapy.
12. **Other gut peptides — largely negative evidence.**
    - **GLP-1:** increases fullness and reduces intake; ageing studies show **no change or increased levels** — unproven as a cause.
    - **PYY:** reduces intake by ~30% acutely, but **no difference in fasting or postprandial PYY between young and old** — no convincing role.
    - **Ghrelin:** the only orexigenic gut hormone; the **effect of healthy ageing on circulating ghrelin is unclarified**

but probably minimal; **ghrelin resistance may occur** (older subjects are less sensitive to its GH-releasing effect). Ghrelin is **positively associated with testosterone**, so falling androgens may lower it.

- **Insulin:** rises with ageing, but this is mainly due to **adiposity-related insulin resistance**; peripheral insulin infusion does not alter appetite — **unlikely to contribute**.

13. **NPY, galanin, orexins and CART. NPY** strongly stimulates feeding and falls with age in male rodents, but **human data show increased, not decreased, NPY** — hence no convincing human role. **Galanin** levels are unchanged. **Orexin A** levels actually *increase* with age. **CART** (inhibitory) rises in old male rats, mediated by the **age-related fall in testosterone** — but human data are absent.
14. **Leptin and testosterone.** Leptin suppresses appetite and rises with fat mass. **In men — but not women — ageing raises leptin even after adjusting for fat mass, because of the age-related fall in testosterone** (leptin is inversely related to testosterone). **Leptin resistance** may also develop with age. Testosterone therapy in frail older men improves **muscle strength, grip, function and rehabilitation outcomes** (Amory, Bakhshi, Srinivas-Shankar) and reduced hospitalisation when combined with a nutritional supplement (Chapman) — **but does not reliably increase appetite**.
15. **Cytokines — "ageing as a form of stress." IL-1, IL-6 and TNF-α** reduce food intake and body weight via central and peripheral pathways and are raised in cancer/AIDS cachexia. Ageing produces **stress-like hormonal changes — raised cortisol and catecholamines, falling sex hormones and growth hormone**. Cortisol and catecholamines **stimulate IL-6 and TNF-α**, while **sex hormones inhibit IL-6**. **Plasma IL-6 rises with normal ageing and correlates inversely with functional ability**; raised CRP and cytokine receptors independently predict physical dysfunction.

PATHOLOGICAL AND ENVIRONMENTAL CONTRIBUTORS

16. **Chronic disease. Depression** (2–10% in community, far more in institutions — the commonest treatable cause; treating it produces weight gain), **dementia** (**~2-fold risk of anorexia**; up to **50% of institutionalised dementia patients have PEM**; apraxia of swallowing, pocketing, spitting; **weight loss may precede dementia as a preclinical marker**), **COPD** (catabolic state, nicotine, opioids, dysmotility), **CHF** (**anorexia in >40% of end-stage**; raised catecholamines, cortisol, renin, aldosterone; hypoxia-driven TNF-α → raised leptin), **CKD** (**30–40% of dialysis patients**, with **four-fold increased mortality**; uraemic toxins; more frequent haemodialysis improves appetite), plus **cancer, rheumatoid arthritis, infection, hyperthyroidism and malabsorption**.
17. **Physical, social and iatrogenic factors. Poor dentition and ill-fitting dentures** (denture wearers had poorer protein intake), **dysphagia**, immobility (stroke), tremor (Parkinson's), poor vision; **poverty and food insecurity**; **~29.3% of non-institutionalised elders live alone**, and **older people eat up to 50% more in company than alone**; institutions often fail to cater to ethnic food preferences. **Iatrogenic: more than 250 prescribed drugs alter taste**; **digoxin and chemotherapy** cause nausea and anorexia; **aluminium/magnesium antacids deplete phosphate and potassium**; **penicillamine causes zinc depletion and loss of taste acuity**; **high-fibre diets cause early satiety**.

CONSEQUENCES, ASSESSMENT AND MANAGEMENT

18. **Consequences.** Anorexia of ageing leads to **impaired muscle function (↓mass, strength, relaxation), decreased bone mass and fracture risk, immune dysfunction and infection, anaemia, poor wound healing, cognitive impairment, delayed recovery from surgery, more falls, more admissions, longer stays and increased mortality**. **Protein-energy malnutrition is a strong independent predictor of mortality** in community, nursing-home, hospital and recently discharged elders, and the risk is amplified by renal failure, cardiac failure and cerebrovascular disease.
19. **Assessment.** Screen appetite with the **SNAQ** — four items on **appetite, early satiety, taste of food and number of meals**, scored 1–5; **a score <14 indicates significant risk of weight loss**. Also use **MNA/MNA-SF item A** (decline in food intake over 3 months). **Differentiate** from **cachexia** (inflammation-driven, CRP raised), **starvation**, **primary depression**, **malabsorption** and **hyperthyroidism**. Apply the **"MEALS ON WHEELS" mnemonic** to uncover treatable causes.
20. **Management.**
    - **First, treat the pathological component:** depression, dementia-related feeding problems, drugs, dentition, dysphagia, constipation, pain, infection, thyroid disease.
    - **Food-first measures: flavour enhancement** (improves intake and nutritional status in nursing-home residents), **eating in company**, small frequent **nutrient-dense** meals, texture modification, oral and dental care, feeding assistance, meals-on-wheels.
    - **Oral nutritional supplements** — mortality benefit when **≥400 kcal/day for ≥35 days** in malnourished, unwell elders aged ≥75.
    - **Resistance exercise** — the primary intervention for the associated sarcopenia.

- **Orexigenic drugs (palliative only): megestrol acetate** is the most studied (increases appetite, weight and quality of life; **oedema** the significant adverse effect); **corticosteroids** improve appetite **without weight gain**; **cyproheptadine** increases appetite without weight gain; **cannabinoids (dronabinol, nabilone)** and **thalidomide (anti-TNF)** have limited evidence. **CCK antagonists remain theoretical.**

Latest Updates (2023–Present)

1. **Prevalence now quantified globally.** A meta-analysis of 36 studies found the overall pooled prevalence of anorexia of ageing to be **22.7%** with an incidence of 3.78%, stable from 2011 to 2024; the highest prevalence was in inpatients at **34.7%**, the most-used tool was the SNAQ, and the authors concluded that a global consensus on diagnosis is needed. *(Maturitas. 2025.)*
2. **SNAQ-based estimates are higher still.** Across 62 studies from 24 countries (n = 80,176), the pooled prevalence of anorexia/appetite loss was **29% (95% CI 26–34%)**, ranging from 22% in South America to 55% in Africa; by setting it was **25% in the community, 38% in nursing homes and 42% in hospitals**, and higher rates were associated with **female sex, lower educational level, eating alone, oral problems and depression**. Note how the "eating alone" and "oral problems" findings validate the textbook's social and dental factors. *(Li W, et al. Age Ageing. 2025;54:afaf249.)*
3. **Formal link with frailty.** In a systematic review of 4,657 community-dwelling older adults, anorexia of ageing was present in 11.3% overall but in **20.5% of frail and pre-frail participants**; robust status was associated with a **63% lower probability** of concomitant anorexia of ageing (OR 0.37, 95% CI 0.21–0.65, p = 0.0005). *(Nutrients. 2023;15:2966.)*
4. **Ghrelin agonism has become a real therapy.** Anamorelin, an oral ghrelin receptor agonist, was approved in Japan in 2021 for cancer cachexia in lung, gastric, colorectal and pancreatic cancers, acting mainly through stimulation of growth hormone secretion and appetite enhancement. In several randomised, double-blind, placebo-controlled trials it significantly improved lean body mass and appetite in cancer cachexia. **Examination point: the textbook's proposed ghrelin/CCK pathways have now produced a licensed drug.** *(Anticancer Res. 2025;45:865; eClinicalMedicine. 2025.)*
5. **GDF-15 identified as a new driver of anorexia-cachexia.** In a phase 2 randomised double-blind 12-week trial, patients with cancer cachexia and elevated GDF-15 (≥1500 pg/mL) receiving ponsegromab, a humanised anti-GDF-15 monoclonal antibody, had significantly greater weight gain than placebo — a median between-group difference of **2.81 kg** in the 400-mg group — with improvements in appetite, cachexia symptoms and physical activity, confirming GDF-15 as a driver of cachexia. **This is a mechanism not present in the textbook at all.** *(Groarke JD, et al. N Engl J Med. 2024;391:2291–2303.)*
6. **Mirtazapine — promising in oncology, disappointing in geriatrics.** A randomised trial supported mirtazapine as an appetite stimulant in NSCLC-related anorexia (JAMA Oncol 2024), but in a multi-site propensity-matched cohort of adults ≥65 started on mirtazapine 7.5 mg, appetite improvement did not differ from controls (20% vs 23%; OR 0.89, 95% CI 0.43–1.82), mean weight change was −1.22 kg, and oversedation prompted discontinuation in 6.3%. *(JAMA Oncol. 2024;10:305–314; companion geriatric cohort. 2026.)*
7. **Cannabinoids should not be recommended.** Randomised controlled trials and meta-analyses in patients with cancer showed that medicinal cannabis did not improve appetite or weight gain, and in the absence of high-quality evidence cannabinoids cannot presently be recommended for cancer-associated cachexia. *(J Thorac Dis. 2025;17:3478–3483.)*
8. **Megestrol is now contraindicated in older adults.** The 2023 AGS Beers Criteria grades it **avoid** (strong recommendation, moderate quality) — minimal effect on weight, with increased risk of thrombotic events and possibly death in older adults. *(AGS Beers Criteria Update Expert Panel. J Am Geriatr Soc. 2023;71.)*
9. **Screening is now mandated.** ESPEN recommends that all older persons shall routinely be screened for malnutrition to identify existing risk early, with oral intake supported by nursing interventions, education, nutritional counselling, food modification and oral nutritional supplements. A dedicated ESPEN dementia update followed in 2024. *(Volkert D, et al. Clin Nutr. 2022;41:958–989; Volkert D, et al. Clin Nutr. 2024.)*
10. **A new iatrogenic cause — the GLP-1 era.** The textbook lists GLP-1 as an unproven endogenous contributor; **exogenous GLP-1 receptor agonists are now a common prescribed cause of anorexia and weight loss in older adults**, with concomitant muscle mass loss raising concerns about sarcopenia and sarcopenic obesity. *(Curr Nutr Rep. 2026.)*

Current Practice Update

Textbook Position (Pathy's 2022)	Current Evidence-Based Practice (2023-present)
Prevalence quoted as **3.3% (community) to 33% (nursing home)** from a single 2013 study	Pooled meta-analyses give **22.7% overall** (36 studies) and **29% by SNAQ** (62 studies) — **community 25%, nursing home 38%, hospital 42%**
Ghrelin's role "unclarified, probably minimal"; therapy	**Anamorelin**, an oral ghrelin receptor agonist, is **licensed in Japan**

theoretical	**(2021)** for cancer cachexia and improves lean mass, appetite and weight
Cytokine mechanism described as IL-1, IL-6, TNF-α	**GDF-15** is now established as a key driver; **ponsegromab (anti-GDF-15) gave up to 2.8 kg weight gain vs placebo** in phase 2 (NEJM 2024)
Megestrol acetate presented as the best-studied and effective orexigenic	**AGS Beers 2023: AVOID** — minimal weight benefit, thrombotic risk, possible increased mortality
Cannabinoids "have shown promise" in cancer and AIDS cachexia	RCTs and meta-analyses show **no improvement in appetite or weight**; not recommended
Antidepressants discussed only via treatment of depression	**Mirtazapine** helps in cancer-related anorexia but showed **no benefit over control in hospitalised older adults**, with **oversedation in 6.3%**
GLP-1 listed as a possible endogenous contributor needing further study	GLP-1 receptor agonists are now a major **iatrogenic** cause of anorexia and lean-mass loss in older adults
Anorexia of ageing described as a syndrome without a formal diagnostic standard	Still true — the 2025 meta-analysis explicitly **calls for a global consensus definition**; **SNAQ <14** remains the most widely used operational criterion
Frailty association implied	Now quantified: **anorexia in 20.5% of frail/pre-frail vs 11.3% overall; robust status carries 63% lower odds**

Examiner's Tip — Mark-Earning Lines

- "The anorexia of ageing is the **physiological, age-related decline in appetite in which the fall in energy intake exceeds the fall in energy expenditure**, resulting in weight loss."
- "**Energy intake falls by about 30% between ages 20 and 80** — anorexia of ageing is the physiological substrate on which pathological causes act."
- "The four physiological mechanisms are **loss of homeostatic regulation, the ageing gut, declining senses, and hormonal/neurotransmitter change**."
- "**Roberts' underfeeding experiment** is the cleanest demonstration of impaired homeostasis — young men regained lost weight, old men did not."
- "**CCK is the strongest single candidate** — levels are higher *and* sensitivity is enhanced; IV CCK-8 suppressed intake **31% in older versus 15% in young adults**."
- "**Impaired fundic accommodation with raised nitric oxide** produces rapid antral filling and early satiety — the mechanical basis of early satiation."
- "**Reduced sensory-specific satiety** narrows the diet, and a monotonous diet is how micronutrient deficiency develops."
- "**Ageing behaves as a chronic stress state** — raised cortisol and catecholamines drive IL-6 and TNF-α, while falling sex hormones remove IL-6 inhibition."
- "**Screen with SNAQ — a score below 14 signals significant risk of weight loss.**"
- "**Depression is the commonest treatable cause**; the simplest effective intervention is **having the older person eat in company — intake rises by up to 50%**."
- "Anorexia of ageing is physiological, but **its consequences are not inevitable** — the aim of management is to prevent protein-energy malnutrition and sarcopenia, not to abolish the appetite decline itself."

Question 5. Obesity in the Elderly and Its Management

15 marks · Source: Pathy's 6th ed. (Ch. 8, 12, 14); Essentials of Clinical Geriatrics 7th ed. (Ch. 12)

20 High-Yield Points

DEFINITION, BODY COMPOSITION AND EPIDEMIOLOGY

1. **Definition.** As per **WHO**, malnutrition includes **excesses** as well as deficiencies — **obesity is a form of malnutrition**. Conventionally defined as **BMI ≥30 kg/m²**; **overweight = 25–29.9 kg/m²**. **The prevalence of obesity among older persons is growing.**
2. **Body composition changes with ageing make BMI unreliable. Fat mass increases and redistributes centrally** (visceral and ectopic — liver, muscle), while **lean mass falls by up to 3 kg per decade after age 50**. Because BMI cannot separate fat from fat-free mass, and **kyphosis makes height measurement unreliable**, **change in weight is preferred to change in BMI** in older adults.
3. **The "obesity paradox."** A meta-analysis showed **BMI 25–29.9 is not associated with a significantly increased mortality risk in older adults**, while **BMI ≥30 carries only a modest (~10%) increase**, irrespective of sex, disease status or smoking. Proposed explanations: **selective survival, cohort effects, a ceiling effect of mortality rates, and the limitations of BMI as a measure of body composition**. In the US NHIS 1997–98, **only 47.2% of those ≥75 were overweight versus 63.5% of those aged 45–64**.
4. **Central adiposity is the better risk marker.** A **higher waist-to-hip ratio was associated with increased mortality independent of smoking, alcohol and oestrogen use** in 31,702 Iowan women followed 11–12 years. But **WHR is hard to interpret in old age** — narrow hips may reflect **gluteal muscle wasting** rather than low fat. The literature on **BMI, WHR and waist circumference in elders remains inconsistent**; waist circumference is nonetheless simple, inexpensive and captures both effects.
5. **Sarcopenic obesity.** Defined as the **coexistence of diminished lean mass and increased fat mass**. At a BMI cut-off of ~27 kg/m², **14% of men <70 and 29% of men >80** were sarcopenic-obese, and **5% of women <70 and 8% of women >80**. It is the phenotype that carries the worst functional prognosis.

PATHOPHYSIOLOGY AND CONSEQUENCES

6. **Visceral fat drives the metabolic syndrome.** While generalised obesity is linked to excess mortality, CVD, osteoarthritis, mobility impairment and disability, it is **predominantly excess visceral fat** that produces **dyslipidaemia, raised fibrinogen, hyperinsulinaemia, glucose intolerance or diabetes, vascular insulin resistance, hypertension and cardiovascular disease**. **All components of the metabolic syndrome are prevalent in older populations.**
7. **Adverse consequences in older adults.**
   - **Metabolic/cardiovascular:** hypertension, type 2 diabetes, dyslipidaemia, coronary artery disease.
   - **Musculoskeletal: high BMI increases knee osteoarthritis risk**; mechanical burden on ageing joints.
   - **Respiratory:** pulmonary function abnormalities, **obesity-hypoventilation syndrome, obstructive sleep apnoea**.
   - **Genitourinary/GI: urinary incontinence rises with BMI**; obesity is associated with **faecal incontinence in women**.
   - **Oncological: breast, colon, pancreas, renal, bladder and prostate cancer**.
   - **Functional: markedly reduced self-reported mobility**, exacerbation of age-related functional decline, **frailty**, **higher rate of nursing-home admission**, and **impaired quality of life**.
8. **Beneficial effects of obesity in old age. Increased bone mineral density with reduced osteoporosis and hip fracture** in older men and women, plus greater nutritional reserve during acute illness. This is why **obesity in older adults is a target for function and quality of life, not primarily for mortality**.

ASSESSMENT

9. **Clinical assessment.** Measure **weight, height (or knee-height estimate), BMI, waist circumference and waist-to-hip ratio**; use **DXA, BIA or CT** for body composition where available. **Assess muscle function — handgrip strength, gait speed, chair rise, SPPB — to detect sarcopenic obesity.** Screen for **metabolic syndrome components, OSA, osteoarthritis, incontinence and depression**; review **drugs causing weight gain (insulin, sulphonylureas, steroids, antipsychotics)**; and establish **the patient's function, goals and life expectancy** before setting a weight target.

MANAGEMENT

10. **The cardinal principle. Weight-loss therapy that minimises muscle and bone loss is recommended only for older persons who are obese AND have functional impairments or metabolic complications that can benefit from weight loss.** In **sarcopenic obesity, prevention of further muscle loss takes priority over weight**

reduction.

11. **Lifestyle change is the primary approach, with modest goals.** A **modest reduction in energy intake of 500–700 kcal/day** is recommended. Note that **short-term caloric restriction improves blood pressure, cholesterol and metabolic rate only when the reduction is at least 20%**.
12. **Exercise is not optional. Regular physical activity is particularly important in obese older persons to improve physical function and help preserve muscle and bone mass. Exercise plus diet is the most effective non-surgical treatment for obesity** and is uniformly advocated by international consensus panels. Adding exercise to diet gives **greater weight loss, preservation of fat-free mass and resting metabolic rate, improved fitness, correction of visceral-obesity-related metabolic abnormalities, and better long-term dietary adherence**.
13. **Choosing the exercise modality.**
    - **Aerobic training may be somewhat better for reducing abdominal fat**, and most robust studies used **moderate- to high-intensity aerobic exercise**; there is no evidence that low-intensity exercise reduces abdominal fat.
    - **Energy restriction causes significant loss of muscle and bone; adding resistance training prevents these adverse body-composition changes, whereas aerobic exercise alone does not.**
    - **Resistance training is more suitable for older obese people with cardiovascular disease, arthritis, osteoporosis or mobility disorders** who cannot tolerate sustained aerobic work.
    - **Combined aerobic plus resistance training is superior to aerobic alone** for trunk fat in older men.
14. **Small weight loss, large visceral benefit. Preferential visceral fat mobilisation** means that a loss of only **~5% of total body weight or fat mass may produce a 25% or greater reduction in visceral fat**, with important metabolic implications for insulin resistance.
15. **Many benefits are independent of weight loss. Blood pressure can be lowered by dietary change (DASH) without weight loss**, and this effect persists for **3–5 years despite weight regain**; coronary prevention effects and **improvements in physical function are also obtainable without weight loss**. **The improvement comes from lifestyle change, not necessarily from the weight falling.**
16. **Evidence from major trials.** In **Look AHEAD**, intensive lifestyle intervention in diabetes produced **sustained weight loss, improved fitness and better cardiovascular risk factors**, but **no reduction in cardiovascular events**, **no difference in objective neuropathy**, and **marginally greater cognitive decline in obese subjects at five years**. In older adults randomised to caloric restriction versus exercise, caloric restriction produced greater weight loss **but also greater loss of lean mass**, with **no long-term difference in physical performance** between the two.
17. **Protect nutrition during weight loss.** Provide **protein 1.0–1.5 g/kg/day**, adequate **calcium (1000–1200 mg) and vitamin D (800 IU)**, and avoid intakes **below ~1500 kcal/day**, since low energy intake **almost guarantees micronutrient inadequacy**. Emphasise **nutrient-dense foods**.
18. **Avoid weight cycling.** Repeated attempts at sustained weight loss cause **loss of lean tissue (muscle and bone) and a fall in metabolic rate**, worsening the energy-balance equation and making long-term dietary management progressively harder.
19. **Pharmacotherapy. The available data are insufficient to determine the efficacy and safety of pharmacotherapy for obesity in older persons.** In obese older adults with type 2 diabetes, weight-loss diets are central; **metformin may act as an appetite suppressant** and **acarbose reduces carbohydrate absorption**. Note that **insulin itself causes significant weight gain**, aggravating the underlying problem.
20. **Bariatric surgery and monitoring. Bariatric surgery is the most effective weight-loss therapy**, but in older patients **perioperative morbidity and mortality are greater, while relative weight loss and improvement in obesity-related complications are lower** than in younger patients. It should therefore be **reserved for a select group of older subjects with disabling obesity that can be ameliorated by weight loss and who meet standard criteria**. Throughout follow-up, **distinguish intentional from unintentional weight loss** — in SHEP, the **excess mortality associated with weight loss was present even in the heaviest subjects (BMI ≥31) and was independent of baseline weight**.

Latest Updates (2023–Present)

1. **Obesity has been formally redefined — clinical vs preclinical.** The Lancet Diabetes & Endocrinology Commission defines **preclinical obesity** as excess adiposity with preserved organ function and generally increased future risk, and **clinical obesity** as a chronic systemic illness with objective signs or symptoms of altered organ function or impaired ability to conduct daily activities directly caused by excess adiposity. Confirmation of excess adiposity requires either direct body fat measurement (DXA or bioimpedance), or at least one anthropometric criterion in addition to BMI, or at least two anthropometric criteria regardless of BMI — using waist circumference, waist-to-hip ratio or waist-to-height ratio with age-, sex- and ethnicity-appropriate cut-offs. Diagnosis of clinical obesity requires at

least one of 18 specified obesity-related conditions in adults. **This directly addresses the textbook's complaint that BMI performs poorly in older adults.** *(Rubino F, et al. Lancet Diabetes Endocrinol. 2025;13:221–262.)*

2. **India has its own revised definition — highly examinable.** In **Stage 1 obesity**, individuals have increased adiposity (**BMI >23 kg/m²**) without discernible effects on organ function or daily activities. **Stage 2 obesity** denotes a more advanced state with generalised and abdominal adiposity affecting physical and organ function and causing functional limitation, requiring **BMI >23 kg/m² plus abdominal adiposity (excess waist circumference or waist-to-height ratio) plus either a mechanical condition such as knee arthritis due to excess weight, or an obesity-associated disease such as type 2 diabetes**. This is "obesity with consequences" and mandates more intensive management. *(Misra A, Vikram NK, Ghosh A, et al. Diabetes Metab Syndr. 2025;19:102989.)*
3. **EASO's parallel framework.** The EASO report retains BMI ≥30 kg/m² but adds BMI >25 kg/m² with a waist-to-height ratio >0.5, with both criteria requiring evidence of medical, functional or mental limitation. *(EASO framework, 2024.)*
4. **Sarcopenic obesity now has consensus diagnostic criteria — but they need validation.** In 2022 ESPEN and EASO issued the first global consensus on diagnostic methods for sarcopenic obesity; applied for the first time in free-living older adults, the algorithm gave a prevalence of 9% and was 100% sensitive for diagnosis, with insulin resistance and an altered ghrelin profile emerging as risk factors. A Western Chinese community validation found a prevalence of 13.2% by ESPEN/EASO criteria, rising with age and BMI and associated with falls. However, the ESPEN/EASO consensus showed **poor diagnostic agreement** with other commonly used sarcopenic-obesity definitions in a 4,155-participant Chinese cohort. *(Donini LM, et al. Clin Nutr / Obes Facts. 2022; Clin Nutr. 2023; Arch Gerontol Geriatr. 2024;127:105557; Front Endocrinol. 2024.)*
5. **GLP-1 receptor agonists work as well in the elderly.** A post hoc analysis of the SURMOUNT and SUMMIT phase 3 trials in adults ≥65 years found that all tests for treatment-by-age-group interaction were non-significant for percent body weight change, waist circumference, systolic blood pressure and HbA1c, indicating comparable efficacy in older and younger participants. Head-to-head, SURMOUNT-5 demonstrated superior efficacy of tirzepatide over semaglutide for percentage weight change and for weight-reduction targets of ≥10%, ≥15%, ≥20% and ≥25% at 72 weeks, with greater improvements in blood pressure, HbA1c, fasting insulin, triglycerides and HDL. *(Alfaris N, et al. Diabetes Obes Metab. 2026; Aronne LJ, et al. N Engl J Med. 2025;393:26–36.)*
6. **But the geriatric caveats are serious.** GLP-1 RAs effectively induce weight loss, but concomitant muscle mass loss is common, raising concerns about sarcopenia and sarcopenic obesity in older adults. A meta-analysis of eight RCTs found participants regained **9.69 kg over 48–52 weeks after stopping** semaglutide or tirzepatide. Handgrip strength appears statistically preserved in short-to-mid-term trials, but longitudinal and retrospective research in older adults with type 2 diabetes has reported reduced handgrip strength and accelerated sarcopenia with prolonged semaglutide use. **Practical rule: never prescribe a GLP-1 RA to an older adult without concurrent adequate protein intake and resistance training.** *(Curr Nutr Rep. 2026; Maturitas. 2025.)*
7. **ESPEN sets the threshold for intervention.** Weight loss in old age should be considered only in cases of obesity with associated health problems, and should be achieved by moderate energy reduction in combination with exercise. Dietary restrictions should generally be avoided in older persons. *(Volkert D, et al. Clin Nutr. 2022;41:958–989.)*
8. **India's double burden in the elderly.** In LASI, about 25% of males and 37% of females aged 45–59 are overweight or obese, while among those aged 60 and above 28% of males and 25% of females are underweight; the urban population is more than twice as likely to be overweight and less likely to be underweight. Age-appropriate Indian BMI cut-offs derived from LASI differ substantially from WHO values in those aged 60 and above. *(Khan J, et al. Sci Rep. 2023;13:12820; Singh A, Chattopadhyay A. Sci Rep. 2024;14:15200.)*

Current Practice Update

Textbook Position (Pathy's 2022 / Essentials 7e)	Current Evidence-Based Practice (2023-present)
Obesity diagnosed by **BMI ≥30**, with acknowledgement that BMI is a poor index in elders	**Lancet Commission 2025**: BMI alone is insufficient — **confirm excess adiposity with a second anthropometric or body-composition measure**, then classify as **preclinical vs clinical obesity** based on organ dysfunction/functional limitation
WHO BMI cut-offs applied uniformly	**India-specific staging (2025): Stage 1 = BMI >23 without organ/functional effect; Stage 2 = BMI >23 + abdominal adiposity + mechanical or disease consequence**
Sarcopenic obesity defined loosely as "diminished lean mass with increased fat mass"	**ESPEN/EASO 2022 consensus algorithm** — screen (obesity + clinical suspicion) → confirm **low handgrip strength + low muscle mass by BIA** → stage by comorbidity; prevalence ~9–13% in older cohorts, though **agreement with other definitions is poor**
"Data are insufficient to determine the efficacy and safety of pharmacotherapy for obesity in older persons"	**GLP-1/GIP receptor agonists are now first-line pharmacotherapy**, with **post hoc analyses in adults ≥65 showing no age interaction** for weight loss or cardiometabolic benefit

Weight loss achieved by **diet + exercise**, with concern about muscle and bone loss	Same principle, but the concern is now **acute for GLP-1-induced weight loss — lean-mass loss, weight regain of ~9.7 kg after discontinuation, and reported grip-strength decline with prolonged use in older diabetics**
Bariatric surgery — greater perioperative risk, lesser benefit in the old; reserve for selected patients	Position broadly unchanged, but **pharmacotherapy now displaces surgery for many older candidates**, with surgery reserved for those failing or intolerant of medical therapy
Obesity in older adults "may not be an important clinical target for reducing mortality"	Still valid — but the **Lancet "clinical obesity"** construct reframes the target as **organ dysfunction and functional limitation**, which is exactly the geriatric endpoint

Examiner's Tip — Mark-Earning Lines

- "**Obesity is a form of malnutrition** — the WHO definition covers excesses as well as deficiencies."
- "**BMI is a poor index of adiposity in old age** because fat mass rises, lean mass falls by up to 3 kg per decade after 50, and kyphosis makes height unreliable — **use weight change and waist circumference instead**."
- "**The obesity paradox: overweight (BMI 25–29.9) carries no excess mortality in those over 65, and obesity only about a 10% increase.**"
- "**Obesity in the older adult is a target for function and quality of life, not for mortality.**"
- "Weight-loss therapy is indicated **only when there is functional impairment or metabolic complication**, and **only if it minimises muscle and bone loss**."
- "**A modest energy deficit of 500–700 kcal/day plus exercise** — never diet alone, because **energy restriction causes significant muscle and bone loss and only resistance training prevents it**."
- "**A 5% loss of body weight can produce a 25% or greater reduction in visceral fat** — preferential visceral mobilisation explains why modest goals still work."
- "**In sarcopenic obesity, protecting muscle takes priority over reducing weight.**"
- "**Blood pressure, lipids, insulin sensitivity and physical function all improve with lifestyle change even when weight does not fall** — the intervention matters more than the number on the scale."
- "**Avoid weight cycling** — repeated attempts lose lean tissue and bone and lower metabolic rate."
- "For India: **Stage 1 obesity is BMI >23 without consequence; Stage 2 is BMI >23 with abdominal adiposity plus a mechanical or disease consequence** — the definition examiners will expect after 2025."

Appendix — Rapid Revision Grid

Every number, cut-off, criterion, mnemonic and named trial from the five answers, compiled for night-before revision.

A. Diagnostic Criteria and Cut-offs

Item	Value to Quote
GLIM — requirement	**≥1 phenotypic + ≥1 etiologic** criterion
GLIM phenotypic	Weight loss >5% in 6 mo or >10% beyond 6 mo · Low BMI · Reduced muscle mass
GLIM etiologic	Reduced intake/assimilation (≤50% requirement >1 wk, any reduction >2 wk, chronic GI disease) · Inflammation/disease burden
GLIM BMI — Western	<20 if <70 y · <22 if >70 y
GLIM BMI — Asian	**<18.5 if <70 y · <20 if >70 y**
GLIM Stage 1 (moderate)	Loss 5–10% in 6 mo (or 10–20% beyond); BMI <20 (<70 y) / <22 (≥70 y)
GLIM Stage 2 (severe)	Loss >10% in 6 mo (or >20% beyond); BMI <18.5 (<70 y) / <20 (≥70 y)
Significant weight loss	**5% in 1 month · 7.5% in 3 months · 10% in 6 months**
MNA-SF score bands	**12–14 normal · 8–11 at risk · 0–7 malnourished**
MNA performance	96% sensitive, 60% specific; normal MNA → mortality RR 0.35 (0.18–0.66)
SNAQ	4 items (appetite, early satiety, taste, meals/day), each 1–5; **<14 = significant risk of weight loss**
Calf circumference	**<31 cm** = low muscle mass (both sexes)
MUAC (2024 validation)	**<23 cm men · <22 cm women**
FFMI cut-off	<17 kg/m² men · <15 kg/m² women
ASMI cut-off	<7.0 kg/m² men · <5.4–5.7 kg/m² women
CRP inflammation grades	<3 mg/L = starvation · medium = cachexia · >100 mg/L = hypercatabolic
Mortality-predictive BMI	<22 kg/m² → higher 1-y mortality; <15th percentile → 180-day post-hospital mortality
Obesity — India Stage 1 (2025)	BMI >23 kg/m², no organ/functional effect
Obesity — India Stage 2 (2025)	BMI >23 + abdominal adiposity (WC or WHtR) + mechanical condition *or* obesity-associated disease
Lancet 2025 obesity	**Preclinical** = excess adiposity, organs preserved · **Clinical** = excess adiposity + organ dysfunction / functional limitation (1 of 18 conditions)
Dental referral score	≥3 points (dry mouth 2, eating difficulty 1, no dental care 2 y 1, pain 2, food change 1, oral lesions 2)

B. Prevalence Figures

Population / Condition	Figure
Malnutrition by MNA — overall	**22.8%**
By setting (MNA)	Rehabilitation 50.5% · Hospital 38.7% · Community 5.8%
European meta-analysis	LTC 28.7% · Rehab 29.4% · Hospital 22% · Nursing home 17.5% · Community 6%
Global malnutrition (2025 meta-analysis, 98 studies)	**18.6%** (Africa 35.7%; highest by NRS-2002 39.9%)
Anorexia of ageing (textbook)	3.3% community → 33% nursing home
Anorexia of ageing (2025 pooled, 36 studies)	**22.7%**; incidence 3.78%; inpatients 34.7%
Appetite loss by SNAQ (2025, 62 studies)	**29%** — community 25%, nursing home 38%, hospital 42%
Anorexia in frail/pre-frail	20.5% (vs 11.3% overall); robust → OR 0.37
Involuntary weight loss	5–15% community · 27% frail ≥65 · 8% of adult outpatient visits
Sarcopenic obesity (ESPEN/EASO applied)	9% free-living Italian cohort · 13.2% Western China community
PEM in institutionalised dementia	up to 50%
Anorexia in end-stage CHF / dialysis	>40% / 30–40% (4-fold mortality)
India — LASI, age ≥60 underweight	28% men · 25% women

India — LASI, age 45–59 overweight/obese	25% men · 37% women
Indian age-specific underweight cut-off (2024)	<17.4 (60–74 y) · <13.3 (≥75 y)

C. Physiology Numbers — Anorexia of Ageing

Parameter	Figure
Fall in energy intake, age 20→80	**~30%**
NHANES III decline	1321 kcal/day men · 629 kcal/day women
New Mexico longitudinal	−25.1 kcal/day/year men · −19.3 women
Swedish cohort (70→76 y)	−610 kcal/day men · −440 women
Roberts' underfeeding experiment	~750 kcal/day deficit × 21 days; old men never regained weight
CCK-8 IV suppression of intake	**31% older vs 15% young (p = 0.02)**
Olfactory decline	>50% aged 65–80; >75–80% aged ≥80; 46% report appetite/weight change
Effect of eating in company	Intake rises up to **50%**
Elders living alone	~29.3% of non-institutionalised
Drugs altering taste	**>250** prescribed drugs
Lean mass loss after age 50	up to **3 kg per decade**
Total body water after 75	falls from 60% to **50%**

D. Therapeutic Targets

Target	Value
Energy requirement	30–35 kcal/kg/day
Protein — healthy elder (ESPEN)	1.0–1.2 g/kg/day
Protein — ill/malnourished (ESPEN)	1.2–1.5 g/kg/day
Protein — ICMR-NIN RDA 2020	Safe intake 0.83 g/kg/day (EAR 0.66)
Fluid	~1 mL/kcal, about **1.5 L/day**
Calcium / Vitamin D after 50	1000–1200 mg / 800 IU (ULs 2000 mg / 4000 IU)
25(OH)D adequacy / deficiency	≥20 ng/mL adequate; treat <10–12 ng/mL
ONS dose-duration rule	**≥400 kcal/day for ≥35 days** in unwell malnourished ≥75 y
ONS effect (2009 meta-analysis, 42 trials)	2.3% weight gain; mortality **RR 0.74 (0.59–0.92)**
Resistance training	Progressive, **2–3 times/week**
Obesity — energy deficit	**500–700 kcal/day** (metabolic benefit needs ≥20% reduction)
Visceral fat response	~5% weight/fat loss → **≥25% visceral fat reduction**
Sodium correction rates	Hypernatraemia <10 mEq/L/24 h; hyponatraemia 6–8 mEq/L/24 h
Folate replacement	1 mg/day
Minimum energy for micronutrient adequacy	~1500 kcal/day

E. Named Evidence — Trials, Guidelines, Consensus

Name	What It Says (one line)
GLIM 2019 / 5-year update 2025	Two-step diagnosis; criteria validated and unchanged; any available muscle-mass method acceptable
GLIS 2024	Sarcopenia = mass + strength + muscle-specific strength; performance is an *outcome*
ESPEN geriatrics 2022	82 recommendations; screen all; food → ONS → enteral → parenteral; avoid dietary restrictions
ESPEN dementia update 2024	Nutrition and hydration guideline specific to dementia
EFFORT trial	Reaching ≥75% of nutrition goals lowers adverse events and mortality; **no legacy effect at 6 months**
Cochrane IPD-NMA 2026	ONS *may* reduce 30-day mortality/SAEs in hospitalised elders; certainty **low to very low**
AGS Beers 2023	**Avoid megestrol** — minimal weight effect, thrombotic risk, possible increased mortality

Endocrine Society 2024 (vitamin D)	Empiric vitamin D at **≥75 years**; against routine 25(OH)D testing; daily > intermittent over 50
USPSTF 2024 draft (vitamin D)	**D grade** — no net benefit for falls/fractures in healthy community-dwelling elders
Ponsegromab (NEJM 2024)	Anti-GDF-15 mAb; up to **2.81 kg** weight gain vs placebo in cancer cachexia
Anamorelin	Oral ghrelin receptor agonist; licensed in Japan 2021; improves lean mass and appetite
Mirtazapine	Works in NSCLC anorexia (JAMA Oncol 2024); **no benefit in hospitalised elders**, 6.3% oversedation
Look AHEAD	Lifestyle intervention improved weight, fitness and risk factors but **no reduction in CV events**
SHEP	Weight loss ≥1.6 kg/yr → **4.9×** mortality; effect present even at BMI ≥31
Cardiovascular Health Study	5% loss → risk-adjusted mortality 1.67×, irrespective of intentionality
Lancet Commission 2025	Clinical vs preclinical obesity; BMI must be paired with a second adiposity measure
Misra et al. 2025 (India)	Stage 1 / Stage 2 obesity for Asian Indians; BMI >23 threshold
ESPEN/EASO 2022	First consensus algorithm for sarcopenic obesity (screen → strength + mass → stage)
SURMOUNT-5 / post hoc ≥65 y	Tirzepatide > semaglutide; **no age interaction** for weight or cardiometabolic benefit
ICMR-NIN 2024	17 guidelines; “My Plate for the Day”; DIAAS; discourages protein supplements
LASI Wave 1	Indian double burden — ~1 in 4 elders underweight, rising urban overweight

F. Mnemonics and One-Line Concepts

MEALS ON WHEELS — Treatable Causes of Weight Loss

Medications · **E**motional (depression) · **A**norexia tardive / alcoholism · **L**ate-life paranoia · **S**wallowing disorders · **O**ral factors · **N**o money · **W**andering / dementia · **H**yper- and hypothyroidism, hyperparathyroidism, hypoadrenalism · **E**nteric (malabsorption) · **E**ating problems (unable to self-feed) · **L**ow-salt / low-cholesterol diets · **S**ocial isolation, **S**tones

Ten Concepts That Earn Marks in Any Nutrition Question

- **Malnutrition is a geriatric syndrome** — multifactorial, poor outcomes, needs CGA.
- **Three-step approach:** SCREEN (MNA-SF) → DIAGNOSE (GLIM) → GRADE (CRP).
- **Starvation responds to refeeding; cachexia does not** — CRP is the bedside discriminator.
- **Serial weight beats BMI** — kyphosis makes height unreliable.
- **Albumin is an inflammatory marker, not a nutritional one** — excluded from GLIM.
- **Anaemia is one of the earliest manifestations of PEM.**
- **ONS need the right dose and duration** — ≥400 kcal/day for ≥35 days.
- **Exercise is the stimulus, nutrition is permissive** — neither works alone in sarcopenia.
- **PEG in advanced dementia neither prolongs survival nor prevents aspiration.**
- **Obesity in the elderly is a target for function, not mortality** — and in sarcopenic obesity, muscle comes before weight.

Three Places Where the Textbook Is Now Outdated — Know These Cold

- **Megestrol acetate:** the textbook calls it the best-studied orexigenic; **AGS Beers 2023 says AVOID**.
- **Albumin:** the textbook calls it a practical indicator of malnutrition; it is now an **inflammatory/prognostic marker excluded from GLIM**.
- **Obesity pharmacotherapy:** the textbook says data are insufficient in older persons; **GLP-1/GIP agonists now have positive ≥65-year post hoc data**, tempered by lean-mass loss and weight regain.

End of document · Textbook content from Pathy’s 6th ed. and Essentials of Clinical Geriatrics 7th ed. · Updates current to August 2026

MD GERIATRIC MEDICINE · EXAMINATION COMPILATION

Palliative Care, End-of-Life Care, Chronic Pain & Bereavement

Six compiled high-yield examination answers
with Latest Updates (2023–present) and Current Practice Updates

Primary sources: Pathy's Principles and Practice of Geriatric Medicine, 6th edition
Kane's Essentials of Clinical Geriatrics
Supplemented with current international and Indian guidelines

Contents

1 **Palliative care — principles, and interventions to relieve suffering in painful bone metastasis with nausea, vomiting, constipation and breathlessness; evolution in India**

WHO principles · total pain · WHO ladder · pathological fracture · cause-directed antiemetics · opioid-induced constipation · death rattle · NDPS Act and the Kerala model

2 **Management of a terminally ill patient and the issues that need to be addressed**

Diagnosing dying · GSF triggers · anticipatory prescribing · hydration vs nutrition · withholding vs withdrawing · DNACPR · palliative sedation · assisted dying

3 **Management of chronic / persistent pain in the elderly; classification and pathophysiology of pain; role of palliative care**

Nociceptive, neuropathic, nociplastic · wind-up and central sensitisation · gate control · ATMAN framework · Beers 2023 · WHO 2023 low back pain guideline

4 **End-of-life care issues**

Recognition · communication · ethics · futility · rationing and ageism · POLST/MOLST · seven legal myths · culture and spirituality · caregivers

5 **Hospice and respite care**

Hospice vs palliative care · Cicely Saunders · four Medicare levels · interdisciplinary team · caregiver burden · adult day services · Zarit Burden Interview

6 **Bereavement**

Grief, mourning and bereavement · complicated grief red flags · DSM-5 bereavement exclusion · Prolonged Grief Disorder in ICD-11 and DSM-5-TR · grief-focused CBT

HOW TO USE THIS COMPILATION

Each answer follows the same architecture: **high-yield numbered points** organised under labelled subheadings → **Latest Updates (2023–present)** with named trials and guidelines cited inline → **Current Practice Update** comparing textbook position with current evidence → **Exam One-Liners** for rapid recall. The one-liner boxes at the end of each answer are designed for night-before revision.

NOTE ON OVERLAP

Questions 1, 2, 4 and 5 overlap substantially in the syllabus. Where content recurs it has been **pivoted rather than repeated** — symptom-control pharmacology sits mainly in Q1 and Q3; the ethical, legal and service-delivery framework sits mainly in Q2 and Q4; hospice and caregiver structure sits in Q5. Read them as a set.

QUESTION 1 · 15 MARKS

Palliative care — principles, and interventions to relieve suffering in painful bone metastasis with nausea, vomiting, constipation and breathlessness; evolution in India

High-Yield Points

Definition and Principles

1. **WHO definition** — palliative care improves the **quality of life (QOL)** of patients and families facing a life-threatening illness by giving **pain and symptom relief, psychosocial and spiritual support**. It also covers prevention, family support and **bereavement counselling** after death.
2. It advocates **early identification of the dying process and impeccable assessment**. It **affirms life** and treats dying as a **normal process**.
3. Palliative care can be given **alongside curative treatment at any point in the disease trajectory, even from diagnosis**. It does not depend on prognosis, stage of disease, or the need for other therapies.
4. **Four fundamental elements**: (i) systematic control of pain, symptoms, psychosocial distress, spiritual and practical needs; (ii) culturally sensitive information with repeated reassessment of **benefits versus burdens**; (iii) genuine **coordination and continuity of care** across settings; (iv) preparation of patient and family for dying, with **hospice options and bereavement support**.
5. **Generic (primary) palliative care** is delivered by all clinicians; **specialist palliative care** is delivered by trained **interdisciplinary teams** (palliative physician, nurse specialist, physiotherapist, occupational therapist, dietician, pharmacist, social worker, spiritual and psychological support).
6. **Hospice** is a subset of palliative care for those near the end of life (in the US, certified prognosis **≤6 months**). Its focus is **caring, not curing**, and it may be delivered at home, in hospital, in a hospice, or in a nursing home.
7. Palliative care is appropriate not only in cancer but also in **chronic disease, heart failure, COPD, end-stage renal disease, dementia and frailty**. **Best outcomes occur when it is started early.**
8. Effective **communication and advance care planning** are core. Structured frameworks include **REMAP**, the **Serious Illness Conversation Guide**, and **Byock's "Five Things"** (thank you; forgive me; I forgive you; I love you; goodbye).

> **CICELY SAUNDERS' CONCEPT OF "TOTAL PAIN"**
>
> **Physical + psychological + social + spiritual suffering.** Treating only the physical component leaves the patient in pain. This single concept underpins the entire interdisciplinary structure of palliative care and is the most quotable framing device for any palliative care answer.

Painful Bone Metastasis

1. **Standard pain assessment** is essential: accurate pain history, a **validated assessment tool**, clinical examination, and appropriate laboratory and radiological investigation. **Numerical rating, pictorial and verbal descriptor scales are more reliable than visual analogue scales in the elderly**; the **same scale must be used at follow-up**.

2. **25–40% of elderly cancer patients have daily pain** and it is frequently **undertreated**. Risk factors for under-treatment are **age >75 years, minority race and cognitive impairment**. There is **no proven change in pain perception with ageing** — older patients simply complain less. In cognitive impairment, assess frequently and use **behavioural cues** (agitation, altered cognition, changed appearance or respiration).
3. Follow the **WHO Analgesic Ladder** — start with **non-opioids (paracetamol, aspirin)**, then add **opioids such as morphine**. Give drugs **"by the clock" every 2–6 hours, not on demand**, especially in long-term care. Use **sublingual, transdermal, rectal or subcutaneous** routes when the oral route fails.
4. **Opioid use in the elderly**: start low, titrate slowly. Prefer **short-acting immediate-release morphine, oxycodone or hydromorphone**. **Avoid meperidine, propoxyphene, fentanyl patches and methadone** (complex metabolism, active metabolites). Anticipate **delirium, falls, nausea and constipation**.
5. **Adjuvants for bone pain**: **NSAIDs and corticosteroids** are useful for bone and visceral pain (watch GI bleeding and renal failure); **topical lidocaine, NSAID gels, capsaicin**; neuropathic adjuvants with the **lowest anticholinergic burden**; plus massage, heat, cold and focused physiotherapy.
6. **Disease-directed therapy for malignant bone pain** — **surgery, chemotherapy, radiotherapy and/or bisphosphonates**. **Radiotherapy relieves pain in up to 80%** and promotes bone healing. **IV bisphosphonates (pamidronate, zoledronic acid, ibandronate)** reduce pain and **skeletal-related events** in breast cancer and myeloma; **caution if creatinine clearance <30 ml/min**.
7. **Pathological fracture** — bone pain is the commonest presenting feature. **About 50% follow breast cancer metastasis** and the **femur is the commonest site**. Surgery is considered first where a fracture has occurred; **long bones need rigid internal fixation before irradiation**. Radiotherapy alone is used for ribs, scapula and pelvis. Surgery is justified if survival is **≥3 months for non-weight-bearing bones** and even **about 1 month for weight-bearing bones**.

Nausea and Vomiting

1. **Identify the mechanism first** — drugs (opioids, chemotherapy), **constipation, bowel obstruction, electrolyte abnormality, hepatic or renal failure, radiotherapy, CNS/brain lesions and anxiety**. Receptors involved are **dopamine, serotonin, histamine and acetylcholine**, acting via the **chemoreceptor trigger zone, vomiting centre, gut and labyrinth**.
2. **Select the first-line antiemetic by cause**, give it **on a schedule** until controlled, then taper to as-needed. **Add a second agent** if symptoms persist, since nausea is often multifactorial. **Use side effects to advantage** — haloperidol when delirium coexists; **avoid ondansetron when constipation is the cause**.

Cause	First-line agent
Constipation	**Senna, PEG, lactulose, bisacodyl**
Delayed gastric emptying	**Metoclopramide 5–10 mg TDS–QDS**
Uraemia / liver metastases	**Haloperidol 1–2 mg PO or 0.5–1 mg IV/SC TDS**
Brain metastases	**Dexamethasone 2–4 mg QDS**
Vertigo / labyrinthine	Hyoscyamine, scopolamine patch, meclizine
Anxiety	Lorazepam 0.5–1 mg

Inoperable bowel obstruction	**Octreotide + dexamethasone + glycopyrrolate**; olanzapine 2.5–5 mg as needed
Non-drug measures	Small frequent meals, relaxation techniques, **acupuncture / acupoint stimulation** for chemotherapy-induced vomiting

Constipation

1. Defined as **fewer than three bowel movements per week**, hard stool and difficult passage. **Prevention is the main strategy** and **opioid-induced constipation must be anticipated and treated prophylactically** in every patient. Combine a **stimulant laxative (senna, bisacodyl)** with an **osmotic laxative (lactulose, PEG, sorbitol)**. **Stool softeners such as docusate are no better than placebo and are not recommended**; **bulking agents such as psyllium are dangerous at end of life** and can cause impaction. **Methylnaltrexone (SC)** is effective for refractory opioid-induced constipation — it **does not cross the blood–brain barrier** and does not precipitate withdrawal.

Breathlessness

1. **Opioids are the main therapy for dyspnoea** — patients and staff often need education about this indication. **Benzodiazepines** are added when anxiety coexists; the two act **synergistically**, allowing lower doses of each. **Oxygen only when hypoxia is present and the patient finds it helpful**. Non-drug measures: a **cool, well-ventilated room, a fan or open window, upright positioning**, glucocorticoids and bronchodilators for bronchospasm, antibiotics if infection is driving it.
2. **Death rattle** — **avoid deep suctioning** (uncomfortable, causes gagging). **Reposition for postural drainage**. **Antimuscarinics** (atropine, scopolamine, **glycopyrrolate — preferred as it does not cross the blood–brain barrier**) may be used, though **clinical benefit is unproven**. **Stop blood pressure monitoring; monitor only respiratory rate and pulse.**
3. **Watch for iatrogenic harm** — **metoclopramide and haloperidol** cause extrapyramidal and neuropsychiatric toxicity; opioids, anxiolytics, antidepressants, antihistamines, steroids, antipsychotics and fluoroquinolones cause **delirium**. For **terminal agitation**, treat the cause first, use behavioural measures, then **low-dose haloperidol** (chlorpromazine or quetiapine if sedation is required).
4. **Total care** must cover **psychological, social and spiritual domains**. **Caregivers with emotional stress have significantly increased mortality** — identify and refer them. Hospice programmes provide **bereavement follow-up for about one year**; screen for **complicated grief**.

Evolution of Palliative Care in India

Period	Landmark
1985	**NDPS Act** enacted — stiff penalties and 4–5 simultaneous licences; morphine consumption **fell about 92%** (roughly 600 kg to 48 kg in 13 years); India ranked **113 of 131** in per-capita morphine use by 1997
1986	**Shanti Avedna Sadan, Mumbai** — India's first hospice
1991	**Pain and Palliative Care Society, Calicut, Kerala** (Dr M R Rajagopal, Dr Suresh Kumar)
1994	**Indian Association of Palliative Care (IAPC)** founded
1998	**Delhi High Court order** (Ghooi case) triggered state-level simplification of narcotic rules

2001	**Neighbourhood Network in Palliative Care (NNPC)**, Kerala — the largest **community- and volunteer-owned** palliative care network in the world
2008	**Kerala** becomes the **first Indian state and the first in Asia** to declare a palliative care policy; home-based care as its cornerstone (revised 2019)
2012	**National Programme for Palliative Care (NPPC)** launched by MoHFW; later merged under **NP-NCD**
2014	**NDPS (Amendment) Act** — created the class of **"Essential Narcotic Drugs"**, reduced licences from **six to one**, and allowed **Recognised Medical Institutions (RMIs)** to stock and dispense morphine
2017–22	Palliative care included in the **National Health Policy 2017**; MD Palliative Medicine recognised; palliative care made one of the **12 expanded service packages of Ayushman Bharat Health & Wellness Centres**
2018	**Common Cause v Union of India** — Supreme Court recognised the **right to die with dignity (Article 21)**, legalised **passive euthanasia** and gave legal status to **Advance Medical Directives (living wills)**
2023	Supreme Court **simplified** the Advance Medical Directive procedure
2024	**MoHFW draft guidelines** for withdrawal of life support in terminally ill patients

Latest Updates (2023–Present)

Bone metastasis

1. The **ASTRO 2024 clinical practice guideline** (replacing the 2017 version, developed with ASCO and MSTS) confirms that **8 Gy in 1 fraction**, 20 Gy/5, 24 Gy/6 and 30 Gy/10 give **equivalent pain relief** for previously unirradiated bone metastases. **Retreatment is more frequent after single fraction**, but its convenience makes it the preferred option in **limited life expectancy**.
2. For **spinal cord or cauda equina compression** in patients unfit for surgery: 8 Gy/1, 16 Gy/2, 20 Gy/5 or 30 Gy/10. **Surgery plus post-operative radiotherapy is conditionally preferred over radiotherapy alone** where feasible.
3. **SBRT is conditionally recommended over conventional radiotherapy** in selected patients with **good performance status**, no neurological deficit and no surgical indication.
4. **Dexamethasone significantly reduces the incidence of pain flare** when given with single-fraction radiotherapy.
5. **Re-irradiation** is now explicitly endorsed: spine 8 Gy/1, 20 Gy/5, 24 Gy/6 or 20 Gy/8; non-spine 8 Gy/1, 20 Gy/5 or 24 Gy/6.
6. **Shared decision-making** received a **strong recommendation** — regimen choice must weigh prognosis, prior dose, normal-tissue risk, quality of life, cost and patient values.

Breathlessness

1. **BETTER-B trial (Lancet Respiratory Medicine, 2024)** — **mirtazapine 15–45 mg daily for 56 days did NOT improve severe breathlessness** in COPD or ILD and caused **more adverse effects and greater care use**. Mirtazapine is **not recommended** for breathlessness.
2. **MABEL trial (Lancet Respiratory Medicine, 2025)** — **morphine did not improve worst breathlessness intensity** in chronic breathlessness; the findings **do not support its routine use** in this setting.

3. **ABCD trial** — **dexamethasone did not improve dyspnoea in advanced cancer** and caused more toxicity, so routine steroid use for cancer dyspnoea is discouraged.
4. The **ERS 2024 clinical practice guideline** on symptom management in serious respiratory illness emphasises **non-pharmacological first-line measures** — **fan-to-face, breathing techniques, pulmonary rehabilitation, pacing and psychological support** — with opioids reserved for **carefully selected, closely monitored patients**.

Nausea, vomiting and constipation

1. **Olanzapine 5 mg** added to standard triple antiemetic therapy significantly improved control of **carboplatin-induced nausea** in a phase III trial (J Clin Oncol 2024), strengthening olanzapine's place as a **broad-spectrum antiemetic**.
2. The **MASCC/ESMO 2023 updated consensus** incorporates **olanzapine** into antiemetic pathways for moderately and highly emetogenic regimens; **low-dose olanzapine is increasingly used for chronic, non-chemotherapy nausea in advanced cancer**.
3. **Peripherally acting mu-opioid receptor antagonists (PAMORAs)** — **methylnaltrexone, naldemedine, naloxegol** — are now standard second-line therapy for **opioid-induced constipation** unresponsive to combined laxatives.

India

1. **Supreme Court order, 24 January 2023** (*Common Cause v Union of India*) — the **living will no longer needs a Judicial Magistrate's countersignature**; it can be **attested by a notary or gazetted officer**. **More than one guardian** may be named. Approval now rests on a **Primary Medical Board** (treating doctor plus two experts of at least 5 years' experience) and a **Secondary Medical Board** (including a CMO nominee), with defined **48-hour timelines**; courts intervene only in disputes.
2. **MoHFW draft guidelines (September 2024)** operationalise the 2018 and 2023 Supreme Court orders, defining **terminal illness** and the clinical pathway for **withholding or withdrawing life-sustaining treatment**.
3. **Access data (2025) — 7–10 million Indians need palliative care annually; fewer than 4% receive it.** India had **526 palliative care centres in 2022** (density **4 per 10 million**). **44.5% of all centres are in Kerala** (2.5% of the population, with about 95% population coverage). **Median travel time to the nearest centre is 118 minutes**; only **23.6% live within 30 minutes**. **Morphine is stocked in only about 66% of centres.** Rural access is markedly worse; Bihar, Odisha and Madhya Pradesh are the worst served.
4. **Essential medicines gap (2025) — no Indian national or state essential medicines list contains all recommended morphine formulations**, and several states omit **oral morphine** entirely, despite morphine consumption being the **NP-NCD indicator of palliative care coverage**.

Current Practice Update

Area	Textbook position	Current evidence (2023–present)
Bone metastasis RT	Radiotherapy relieves pain in up to 80%; fractionation not specified	**8 Gy single fraction is the default** for uncomplicated painful bone metastases; equivalent to multifraction; **SBRT conditionally added** for selected good-performance-status patients
Pain flare after RT	Not addressed	**Prophylactic dexamethasone** reduces post-radiotherapy pain flare

Bone-modifying agents	IV bisphosphonates emphasised	**Denosumab and bisphosphonates** both standard; denosumab preferred in renal impairment (no dose adjustment); **dental assessment before starting** to prevent osteonecrosis of the jaw
Breathlessness — opioids	Opioids are "the main therapy" for dyspnoea	Benefit is **more modest than previously believed**; **MABEL (2025) was negative**. Opioids are now **individualised, trial-based and monitored**, with **non-drug measures first-line**
Breathlessness — antidepressants	Not discussed	**Mirtazapine should not be used** (BETTER-B, 2024)
Breathlessness — steroids	Steroids for bronchospasm	**Dexamethasone is not effective for cancer dyspnoea** and adds toxicity
Nausea and vomiting	Cause-directed single agent, then add a second agent	**Olanzapine has moved to the front line** as a broad-spectrum agent for both chemotherapy-induced and chronic advanced-cancer nausea
Opioid-induced constipation	Methylnaltrexone as rescue	**PAMORA class** (naldemedine, naloxegol, methylnaltrexone) now established second-line; docusate confirmed ineffective
End-of-life decision-making	Framed around UK, US and Australian law	**India now has its own legally operative framework** — 2023 Supreme Court order plus 2024 MoHFW draft guidelines
India-specific service model	Not covered	**Kerala's community NNPC model** is the WHO-cited exemplar for low-resource settings; national coverage remains **under 4%**

Exam One-Liners

Total pain (Cicely Saunders) = physical + psychological + social + spiritual suffering.

Analgesics by the clock, by the mouth, by the ladder — WHO principle.

8 Gy in a single fraction = standard palliative dose for uncomplicated painful bone metastasis.

Commonest site of pathological fracture = femur; commonest primary = breast.

Glycopyrrolate is preferred for death rattle — it does not cross the blood–brain barrier.

Docusate is no better than placebo; never use bulking agents at end of life.

Ondansetron is the wrong choice when constipation is the cause of nausea.

NDPS Amendment 2014 — the single most important Indian reform for morphine access.

Kerala 2008 — first state in Asia with a palliative care policy.

Supreme Court 2023 — notary or gazetted officer, not a magistrate, for a living will.

QUESTION 2 · 15 MARKS

Management of a terminally ill patient and the issues that need to be addressed

High-Yield Points

Definitions and Recognising the Dying Patient

1. **End of life** refers to the **last 12 months of life**; the **terminal phase** refers to the **last few days or hours**. Not every patient has a recognisable terminal phase — some die suddenly, as in **massive pulmonary embolism or terminal haemorrhage**.
2. **Clinicians often fail to consider that the patient is dying** when the condition deteriorates. Unless this possibility is actively considered, dying patients continue to receive **resuscitation** and are **removed from their homes and admitted to hospital for their last hours and days**.
3. **Gold Standards Framework triggers** to identify a patient needing a palliative approach:
    - The **"surprise" question** — would you be surprised if this patient died in the next 6–12 months?
    - **Choice or need** — the patient chooses comfort care only, or has special supportive care needs
    - **Clinical indicators** of advanced disease
4. **Prognostication** should be expressed as **time, function, or unpredictability**. Use **ePrognosis, the Palliative Performance Scale, Karnofsky and ECOG** plus clinical experience. Always give estimates as **ranges** — hours to days, days to weeks, weeks to months — acknowledging inherent uncertainty.

Assessment, Communication and Care Planning

1. Assessment must be **multidimensional** — physical, psychological, social, spiritual, cultural, informational and practical needs of **both patient and family**, taking account of health literacy and capacity to participate.
2. **Communication is the core intervention.** Use structured frameworks — **REMAP** and the **Serious Illness Conversation Guide**. Contrary to clinician fears, serious illness conversations **reduce patient anxiety and depression** and do not damage the doctor–patient relationship.
3. A **documented care plan** based on the patient's wishes must be shared with family and all providers. It should account for **functional and cognitive status, disease trajectory, cultural and spiritual preferences, family structure, finances, caregiver availability and home support**, and must be revised as the condition changes.
4. Decide **whether and when to refer to specialist palliative care**. Not everyone needs referral; many are well cared for by their usual team. Clinicians must **never say "there is nothing more that can be done"** — specialist palliative care works on the principle that there is **always something, however small**, that can improve comfort and quality of life.

Symptom Control in the Terminal Phase

1. **Anticipatory prescribing is essential** — prescribe in advance for the symptoms likely to develop (**pain, breathlessness, nausea, agitation, secretions**), ensure the drugs are physically available, and **establish a parenteral (subcutaneous) route** before the oral route fails.

2. **Pain** — follow the **WHO ladder**, give opioids **"by the clock" every 2–6 hours, not on demand**, use **sublingual, transdermal, rectal or subcutaneous** routes when swallowing fails, and always co-prescribe a **bowel regimen**.
3. **Breathlessness** — **opioids are the main therapy**; add a **benzodiazepine** if anxiety coexists; use a **cool, well-ventilated room and a fan**; **oxygen only if hypoxic and the patient finds it helpful**.
4. **Respiratory secretions ("death rattle")** — **avoid deep suctioning** (uncomfortable, causes gagging); **reposition for postural drainage**; **glycopyrrolate** is preferred (does not cross the blood–brain barrier, less delirium), though **clinical benefit is unproven**. Stop measuring blood pressure; monitor only **respiratory rate and pulse** to avoid disturbing patient and family.
5. **Terminal agitation and delirium** — first look for **reversible causes** (pain, urinary retention, faecal impaction, drugs, hypercalcaemia, infection). Use **behavioural measures** — limit visitor numbers, music therapy. If these fail, use **low-dose haloperidol** (chlorpromazine or quetiapine if sedation is needed). Remember that many symptom-control drugs themselves cause delirium.
6. **Deprescribe.** Stop **ineffective and burdensome medicines**. Some patients actually **improve** when unnecessary drugs are stopped — showing in retrospect that the drugs, not the disease, caused the deterioration.
7. **Basic comfort care is non-negotiable** — meticulous **mouth care**, pressure-area care, bowel and bladder care, positioning, and a quiet, dignified environment.

Hydration and Nutrition

1. **Consider hydration and nutrition as separate decisions.** When the patient cannot swallow, **maintaining some hydration is an important comfort measure** and a key adjunct to mouth care. **Subcutaneous fluid infusion (hypodermoclysis)** into the abdominal wall can be set up almost anywhere, has a **very low complication rate**, is **as effective as intravenous hydration** for maintenance, yet is **rarely used**. Avoid **fluid overload**, especially in cardiac decompensation.
2. **Nutrition is emotionally charged.** Dying patients have **anorexia, distorted taste and disease-related cachexia**. **Anorexia may be the first sign of subclinical nausea** — exclude **hypercalcaemia, hyperglycaemia, renal failure and infection (especially urinary)**. **Corticosteroids** give only a short-lived appetite effect and cause **hyperglycaemia, muscle wasting, GI erosion and bleeding, labile mood and poor sleep**; **megestrol has no advantage**. **Artificial nutrition rarely improves survival or quality of life**; nasogastric feeding is uncomfortable, disfiguring and risks **aspiration pneumonia**.

Ethical and Legal Issues

1. **Withholding and withdrawing** an ineffective, unduly burdensome treatment are **morally and legally equivalent**. This permits a **time-limited trial** of any treatment of doubtful benefit. In almost all cases the **underlying disease causes death**, not the withdrawal — this must be explained to relatives and team members *in advance*, especially where death follows withdrawal quickly (for example, ventilator withdrawal).
2. **Cardiopulmonary resuscitation (CPR)** is a medical treatment like any other. Two **separate** decisions are involved:
    - **Can CPR work physiologically?** — a clinical judgement. The chance is **markedly reduced in advanced terminal disease**, end-stage organ failure and degenerative neurological disease; in one series **no terminally ill patient survived CPR**.
    - **Is the resulting quality of life worth having?** — **only the patient can decide this**; check first for an advance directive.

    A major harm of unsuccessful CPR is **denying a peaceful and dignified death**. Public expectations are distorted by **television dramas** that grossly overstate success and never show the harms. Where the team judges CPR futile, a discussion that *appears to offer a choice* is inappropriate and misleading

— but the clinician still has a duty to ensure patient and family understand the **terminal nature of the illness** and that care is now aimed at comfort.

3. **Advance care planning (ACP)** — a process of discussing and recording values, goals and wishes in an **advance care directive (ACD)**. An advance decision to refuse treatment (**"living will"**) is legally binding where it is (i) **specific to the situation**, (ii) made **with capacity**, (iii) **voluntary and informed**, and (iv) **not subsequently invalidated by the patient's actions**. It only takes effect **once capacity is lost**. **Values rarely change over a lifetime, but care decisions can** — review the ACD **yearly, on deterioration, and before admission to a care home**. Where no ACD exists, decision-making reverts to the **"best interests" principle**, with a **proxy, enduring power of attorney or durable power of attorney for health care**.
4. **Palliative (terminal) sedation** should **rarely be necessary**. It is indicated where a patient is **exhausted by complex pain**, profoundly distressed after a neurological insult, or has **refractory delirium**. It **requires consent** as a therapeutic intervention; if capacity is lost, proceed via the **legally appointed attorney or proxy** in the patient's best interests. Use a **short-acting agent such as midazolam, titrated upward in increments**, with **regular review so the dose can be reduced**. Small anxiolytic doses of a benzodiazepine usually suffice for terminal restlessness — **deliberate deep unconsciousness is not required**.

> **EXAMINER TRAP — SEDATION VERSUS EUTHANASIA**
>
> **Palliative sedation is titrated to the symptom**, with the aim of symptom relief, and **death results from the underlying disease**. Giving high doses by **continuous infusion without titration** and **without considering hydration** suggests an intent to shorten life. State the distinction in exactly these terms — intent, titration and cause of death.

1. **Assisted dying** — **assisted suicide** = supplying the means (for example, prescribing a lethal dose); **euthanasia** = deliberately administering a lethal drug. Arguments **for** centre on **autonomy, existential distress and futility**; arguments **against** on **public safety, coercion of the vulnerable, absence of watertight safeguards, and respect for life**. Diagnosis and prognosis are imprecise; **3 of 18 Oregon patients** given lethal prescriptions had **unrecognised, untreated depression**; **internal coercion** ("I have a duty to die") is very hard to detect. When a patient requests it, first explore **what they are actually asking** — a confirmed wish to die, or a question about whether life is still worth living.

Place of Care, Family and Bereavement

1. **Preferred place of care and death must be asked about and planned for.** In the UK, **49–100% of cancer patients wish to die at home**, yet only about **26% of cancer deaths and 20% of all deaths** occurred at home. Where a preference had been **documented**, the place of death matched it in **73%**. Before discharge: arrange **beds, oxygen, commodes, shower chairs**, confirm **who will prescribe opioids** and that the pharmacy stocks them, and ensure someone is at home to receive deliveries.
2. **Integrated care pathways** (for example, the **Liverpool Care Pathway**) improved symptom control, communication with relatives and nurses' confidence, but were **phased out in the UK after complaints** — the fault lay in **"tick-box" use rather than the pathway itself**. Replaced by the **Five Priorities for Care of the Dying Person** and a **personalised** approach. **If the patient improves, discontinue the pathway, review, and reconsider active interventions.**
3. **Family and bereavement** — grief **begins at diagnosis**, not at death. Relatives must be told what is happening, encouraged to be involved, and allowed at the bedside — including frail elderly spouses and children. **Warning signs of complicated grief**: sudden or traumatic death, loss of a child, loss of a partner with an **ambivalent relationship**. Bereaved relatives have increased **depression, cardiac events, intercurrent infection and mortality**; **caregivers reporting emotional stress have significantly increased mortality**. Hospices provide bereavement follow-up for about **one**

year. Offer families **Byock's "Five Things"** — thank you; forgive me; I forgive you; I love you; goodbye. As Cicely Saunders said, *the way people die remains in the memory of those who live on*.

Framework: "Issues That Need to Be Addressed"

A revision checklist — if the question says "issues", answer in domains, not in a narrative.

Domain	Key issues
Physical	Pain, breathlessness, nausea and vomiting, constipation, secretions, agitation, fatigue, mouth care, pressure areas
Recognition	Diagnosing dying; prognostication; when to refer to specialist palliative care
Psychological	Fear, anxiety, depression, loss of control, dignity, unfinished business
Social	Caregiver burden, finances, dependants, isolation, place of care
Spiritual and cultural	Meaning, faith, rituals, culturally sensitive disclosure and decision-making
Ethical and legal	Capacity, consent, ACP/ACD, proxy, DNACPR, withholding versus withdrawing, futility, sedation, requests for assisted dying
Practical and logistic	Anticipatory drugs, subcutaneous route, equipment at home, 24-hour access, opioid supply
Therapeutic review	Deprescribing, artificial nutrition and hydration decisions, stopping observations and blood tests
Family and bereavement	Information, involvement, presence at the bedside, children, complicated grief, follow-up
Team	Staff moral distress, documentation, handover, training, avoiding "tick-box" care

Latest Updates (2023–Present)

Palliative sedation

1. The **revised EAPC framework on palliative sedation (Palliative Medicine, 2024)**, developed by international Delphi consensus with **42 statements**, redefines the practice:
    - **Proportionality** is now built into the definition — sedation is the *monitored, proportional* use of medication to reduce consciousness.
    - **"Suffering"** now explicitly includes **physical, psychological and existential** distress; **"refractory"** means **untreatable** (clinician view) *and* **intolerable** (patient view).
    - **No fixed remaining life expectancy is specified** as a criterion — the trigger is refractoriness and proportionality.
    - **Hydration is decided independently** of the decision to sedate.
    - **Patient autonomy is strongly emphasised**, with a **stepwise pharmacological approach** provided.

Hydration at the end of life

1. **NICE guidance (NG31; quality standard QS144)** clarifies that there is **no evidence that clinically assisted hydration prolongs dying, nor that withholding it hastens death**.
    - **Assess hydration status daily** and support **oral drinking** wherever it is safe.
    - Consider a **therapeutic trial** of clinically assisted hydration; **monitor at least 12-hourly**.
    - **Continue if benefit**, **reduce or stop if harm** (fluid overload, infusion-site pain or swelling) or if the patient no longer wants it.
    - **"One-size-fits-all" prescribing proformas are discouraged** — they contributed to the over-sedation concerns raised in the Neuberger review.

End-of-life decision-making in India

1. **Supreme Court of India, 24 January 2023** (*Common Cause v Union of India*) simplified the 2018 framework: the **advance medical directive no longer needs a Judicial Magistrate's countersignature** — a **notary or gazetted officer** suffices; **more than one guardian** may be named; approval runs through a **Primary Medical Board** and a **Secondary Medical Board** with defined timelines.
2. The **ISCCM–IAPC Expert Consensus and Position Statements for End-of-Life and Palliative Care in the ICU (Indian Journal of Critical Care Medicine, 2024)** is now the operative Indian bedside document. It combines **contemporary evidence, ethics and law**, incorporates the January 2023 Supreme Court safeguards, and provides a structured process for **foregoing life-sustaining treatment (FLST)** based on **physician prognostic assessment, patient- and family-centred shared decision-making, documented due process, and integration of palliative care into the ICU**.
3. The **MoHFW draft guidelines for withdrawal of life support in terminally ill patients (September 2024)** operationalise the 2018 and 2023 Supreme Court orders, defining **terminal illness** and the **two-tier medical board pathway**.

Advance care planning

1. Systematic reviews and meta-reviews (2023–2025) show that ACP reliably improves **process outcomes** — documentation rates, patient and surrogate engagement, and concordance between stated preferences and recorded care — but has **limited or inconsistent effects on hospitalisation, health status and survival**.
2. The field has therefore shifted from **"completing a document"** to **iterative goals-of-care communication**, **surrogate preparation**, and **system-level implementation** (trained facilitators, embedded processes, staff education), rather than one-off form-filling.

Symptom management evidence

1. **BETTER-B (2024)** — **mirtazapine 15–45 mg daily for 56 days did not improve severe breathlessness** in COPD or ILD and caused **more adverse effects and greater care use**; it is **not recommended**.
2. **MABEL (2025)** — **morphine did not improve worst breathlessness intensity** in chronic breathlessness; the findings **do not support routine use**.
3. The **ERS 2024 clinical practice guideline** on symptom management in serious respiratory illness places **non-pharmacological measures first** — **fan-to-face, breathing techniques, pacing, rehabilitation, psychological support** — with opioids reserved for carefully selected and monitored patients.

Current Practice Update

Area	Textbook position	Current evidence and practice (2023–present)
Care of the dying	Integrated care pathways (LCP) described, then withdrawn; Five Priorities introduced	Fully **individualised care plans**; **no blanket prescribing proformas**; daily reassessment of hydration, symptoms and goals
Palliative sedation	"Rarely necessary"; consent required; titrated midazolam	**Proportionality** is now central; "refractory suffering" **explicitly includes psychological and existential distress**; **no fixed prognostic window**; **hydration decided separately**; stepwise drug algorithm provided
Clinically assisted hydration	Comfort measure; subcutaneous route under-used	Explicitly stated that clinically assisted hydration **neither prolongs dying nor does withholding hasten death**; **therapeutic trial with 12-hourly monitoring**, stop if harm
Advance care planning	Emphasis on **completing a valid ACD**	Documents alone change little; emphasis on **repeated goals-of-care conversations**, surrogate preparation and organisational implementation
DNACPR communication	UK position: **no legal or moral requirement** to tell a patient that a DNACPR decision was made	Practice has moved decisively toward **transparent discussion and documentation**; the ISCCM–IAPC statement requires **documented shared decision-making**
Legal framework	UK and Australia-centric (Mental Capacity Act, Victorian VAD Act)	**India now has its own operative framework** — Supreme Court 2023 order + MoHFW 2024 draft guidelines + ISCCM–IAPC 2024 position statement
Breathlessness drugs	Opioids are "the main therapy"	Benefit is **more modest than believed** (MABEL negative); **mirtazapine not recommended** (BETTER-B); **non-pharmacological measures first-line**
Terminal delirium	Low-dose haloperidol after treating causes	**Non-pharmacological measures first**; antipsychotics reserved for severe distress, at the lowest dose for the shortest time, recognising they may not improve — and can worsen — delirium
Artificial nutrition	Rarely beneficial; NG feeding uncomfortable and risks aspiration	Reinforced — **"comfort feeding only"** is the preferred humane alternative; tube feeding does not improve survival or quality of life in advanced dementia

Exam One-Liners

End of life = last 12 months; terminal phase = last days to hours.

Surprise question: "Would I be surprised if this patient died within 6–12 months?"

Prognosis in ranges — hours-to-days, days-to-weeks, weeks-to-months.

Withholding = withdrawing, morally and legally — this permits a **time-limited trial**.

In advanced terminal disease **CPR has essentially no meaningful success**; in one series, none survived.

An advance directive only takes effect once capacity is lost.

Values rarely change; care decisions do — review the ACD yearly and on deterioration.

Palliative sedation is titrated to the symptom — that is what separates it from euthanasia.

Hypodermoclysis = simple, safe, as effective as IV, and grossly under-used.

Never say "nothing more can be done" — refer to specialist palliative care.

Death rattle distresses the family, not the patient — reposition, do not suction.

India's operative documents: Supreme Court January 2023 order + ISCCM–IAPC 2024 + MoHFW 2024 draft guidelines.

QUESTION 3 · 15 MARKS

Management of chronic / persistent pain in the elderly; classification and pathophysiology of pain; role of palliative care

High-Yield Points

Definition and Epidemiology

1. **Chronic (persistent) pain** is pain that persists or recurs for **more than 3 months**, beyond the expected period of tissue healing. It is regarded as a **disease in its own right**, not merely a symptom.
2. Prevalence rises steeply with age — roughly **25–50% of community-dwelling older adults** and **up to 80% of long-term care residents**. Commonest causes: **osteoarthritis, degenerative spine disease, osteoporotic vertebral fracture, diabetic neuropathy, post-herpetic neuralgia, peripheral arterial disease and cancer**.
3. Pain in older people is **under-reported, under-diagnosed and under-treated**. Barriers include the belief that pain is a **normal part of ageing**, fear of being labelled "difficult", fear of what the pain signifies, fear of addiction, cognitive and sensory impairment, and the clinician assumption that older patients feel less pain — for which there is **very little evidence**.

Classification

1. **Mechanistic (IASP) classification — three types:**
   - **Nociceptive** — nociceptor activation by actual or threatened tissue damage.
     - *Somatic* (skin, bone, joint, muscle, connective tissue) → **well localised, constant; aching, throbbing, gnawing, stabbing**. Examples: arthritis, fracture, bone metastases.
     - *Visceral* (internal organs) → **diffuse, poorly localised, referred, intermittent; colicky, cramping, squeezing**, often with nausea, vomiting and sweating. Examples: renal colic, bowel obstruction.
   - **Neuropathic** — lesion or disease of the somatosensory nervous system, with **abnormal processing of sensory input → burning, tingling, shooting, electric-shock-like**, with **paraesthesia, dysaesthesia, allodynia, hyperalgesia**, sensory loss, weakness or altered reflexes. Examples: diabetic neuropathy, post-herpetic neuralgia, trigeminal neuralgia, radiculopathy, post-stroke central pain.
   - **Nociplastic** — pain from **altered nociception without demonstrable tissue damage or somatosensory lesion**. Examples: fibromyalgia, myofascial pain syndrome, complex regional pain syndrome.
2. **Other axes of classification** — by **duration** (acute <1 month, subacute, chronic >3 months); by **aetiology** (cancer versus non-cancer); by **ICD-11** (chronic *primary* pain versus chronic *secondary* pain). Most persistent pain in older people is **mixed**.
3. Special categories to recognise: **breakthrough (incident) pain**, **procedural pain**, and **"total pain"** — physical + psychological + social + spiritual suffering.

Pathophysiology

1. **Four processes of nociception:**
   - **Transduction** — noxious stimulus converted into an electrical impulse at peripheral nociceptors. **A-delta fibres** carry fast, sharp, localised pain; **C fibres** carry slow, dull, burning pain. Mediators: **prostaglandins, bradykinin, substance P, histamine, serotonin, hydrogen ions and ATP** (the "inflammatory soup").
   - **Transmission** — impulse passes via the **dorsal root ganglion → dorsal horn (laminae I, II, V) → spinothalamic tract → thalamus → somatosensory cortex**. Key dorsal-horn transmitters are **glutamate (NMDA receptor)** and **substance P (NK1 receptor)**.
   - **Perception** — conscious awareness in cortex and limbic system; explains the **affective and emotional dimension** of pain.
   - **Modulation** — **descending inhibitory pathways** from the **periaqueductal grey and rostral ventromedial medulla**, using **endogenous opioids, noradrenaline, serotonin and GABA**. The **gate control theory** (Melzack and Wall) explains how large A-beta fibre input inhibits nociceptive transmission at the dorsal horn — the basis of **TENS, rubbing and massage**.
2. **Transition from acute to chronic pain:**
   - **Peripheral sensitisation** — inflammatory mediators lower the nociceptor threshold → **primary hyperalgesia**.
   - **Central sensitisation** — repetitive C-fibre input produces **"wind-up"** via NMDA receptor activation, increasing dorsal-horn excitability and expanding receptive fields → **secondary hyperalgesia and allodynia**.
   - **Loss of descending inhibition**, **glial activation**, and **maladaptive cortical reorganisation** then maintain pain independently of the original injury.
3. **Age-related changes in the nociceptive system**: reduced density of **myelinated and unmyelinated peripheral fibres**, slowed conduction, reduced **substance P and CGRP**, and **impaired descending inhibitory modulation with reduced endogenous opioid function**. The net effect is that pain **threshold may be marginally raised but tolerance is reduced**, sensitisation occurs more readily, and established pain resolves more slowly. Older people are therefore **more vulnerable to persistent pain, not less sensitive to it**.
4. **Consequences of untreated pain** — depression, anxiety, sleep disturbance, anorexia and weight loss, **deconditioning, immobility, falls and fractures**, functional decline, social isolation, delirium, increased healthcare use and caregiver burden. **Pain is a key reversible cause of agitation in dementia.**

Assessment

1. AGS recommends that **every older person be screened for persistent pain** on initial presentation or admission to any healthcare service; anyone whose pain affects **physical function, psychosocial function or quality of life** needs a **comprehensive pain assessment**. Pain is treated as the **"fifth vital sign"**.
2. **History** — site and radiation, character, timing, aggravating and relieving factors; **relationship of pain to physical and social function and sleep**; **complete analgesic history** (prescribed, over-the-counter, alternative remedies, alcohol, side effects); **patient's attitudes and beliefs about pain and its treatment**; effectiveness of and satisfaction with current management; **social support and access to healthcare**.
   Examination — the painful site and common referral zones; focused **musculoskeletal** and **neurological** examination; **observation of physical function**; **psychological and cognitive assessment**.

3. **Measurement tools:**
    - **Cognitively intact — self-report is the gold standard.** The **numerical rating scale (0–10)** is the most suitable in older people; verbal descriptor scales, pain thermometer and faces scales are also validated. **Visual analogue scales are the least reliable. Always use the same scale on follow-up.**
    - **Cognitively impaired or non-verbal** — use observational tools: **Abbey Pain Scale, PAINAD, DOLOPLUS-2**, plus caregiver report. Assess **during movement**, assess **frequently** (poor recall), and treat **any unexplained behavioural change in advanced dementia as possible pain** until proved otherwise.

Management — General Principles

1. Set **realistic, function-based goals** (better sleep, mobility and participation — not zero pain). Use a **multimodal, multidisciplinary, biopsychosocial approach**. **Treat the underlying cause where possible.** Follow the geriatric rule of the **fewest agents, by the least invasive route, at the lowest effective dose — start low, go slow, but go.** Give analgesia **regularly ("by the clock") for constant pain**, with breakthrough doses. **Review, titrate, and deprescribe what does not work.**
2. **Non-pharmacological measures are first-line and are always combined with drugs**: patient and caregiver **education**; **structured exercise and physiotherapy** (strengthening muscles around the joint, aerobic and balance training); **weight reduction**; **occupational therapy** — walking aids, appropriate footwear, joint protection, environmental modification; **heat and cold**; **massage, TENS, acupuncture**; **relaxation, guided imagery, distraction, music**; and **psychological therapy — cognitive behavioural therapy (CBT)**, mindfulness and support groups.

Pharmacological Management

> **MNEMONIC — THE ATMAN STEPPED FRAMEWORK FOR CHRONIC NON-CANCER PAIN**
>
> **A** — **A**cetaminophen (paracetamol) and **a**ntidepressants (SNRIs)
> **T** — **T**opical analgesics (diclofenac, lidocaine, capsaicin, methyl salicylate, menthol, cannabidiol)
> **M** — **M**uscle relaxants (cyclobenzaprine, baclofen)
> **A** — **A**nticonvulsants (gabapentin, pregabalin, carbamazepine) and **a**nti-inflammatory drugs (NSAIDs)
> **N** — **N**on-drug interventions (CBT, physiotherapy, mindfulness, support groups, hot and cold remedies)
>
> **Step 1** paracetamol + topicals + non-drug measures → **Step 2** duloxetine, gabapentin/pregabalin, muscle relaxants, interventional therapies → **Step 3** nortriptyline, tramadol, NSAIDs → **Step 4** opioids.

1. **Non-opioids:**
    - **Paracetamol** — first-line for mild-to-moderate musculoskeletal pain. Maximum **4 g/24 h**; reduce to **2–3 g/day** in frailty, low body weight, hepatic impairment or regular alcohol use.
    - **NSAIDs** — effective for **inflammatory, bone and visceral pain**, but hazardous in the elderly: **GI bleeding and peptic ulcer, acute kidney injury, fluid retention, heart failure, hypertension**, and multiple drug interactions. Use the **lowest dose for the shortest time**, prefer **topical formulations**, co-prescribe a **PPI** if systemic use is unavoidable.
    - **Topical agents** — capsaicin, **lidocaine 5% patch**, diclofenac gel, ketamine or combination gels; minimal systemic absorption makes these particularly valuable in old age. **Intra-articular corticosteroid injection** for localised joint pain.

2. **Opioids** — reserved for **moderate-to-severe pain limiting function and quality of life** after non-opioid measures have failed.
    - **Start low and titrate slowly**; prefer **short-acting immediate-release morphine, oxycodone or hydromorphone**; convert to a long-acting preparation with breakthrough cover once requirements are stable.
    - In older adults expect a **higher peak effect** (fewer receptor sites) and a **longer duration of action** (slower elimination).
    - **Avoid meperidine (pethidine) and propoxyphene** (ineffective, neurotoxic); use caution with **fentanyl patches and methadone**, and **never initiate a patch in an opioid-naive patient**.
    - **Anticipate every side effect**: **constipation (never tolerated — always co-prescribe a stimulant plus osmotic laxative)**, nausea, sedation, **delirium, falls and fractures**, respiratory depression, myoclonus, urinary retention. **Methylnaltrexone and other PAMORAs** for refractory opioid-induced constipation.
    - Assess misuse risk (**Opioid Risk Tool**) — risk factors are personal or family history of substance abuse, PTSD or past abuse, and psychiatric comorbidity.
3. **Adjuvants:**
    - **Neuropathic pain** — **gabapentin and pregabalin** (dose-reduce in renal impairment; cause sedation, dizziness, oedema, falls); **duloxetine and venlafaxine (SNRIs)**; **topical lidocaine and capsaicin**; **carbamazepine for trigeminal neuralgia**. **Tricyclic antidepressants should generally be avoided in older adults** because of anticholinergic effects — dry mouth, urinary retention, constipation, delirium, tachycardia, blurred vision and falls; if unavoidable, use **low-dose nortriptyline** (least anticholinergic).
    - **Corticosteroids** — useful for **inflammatory, bone and nerve-compression pain**; watch for delirium, hyperglycaemia, fluid retention and immunosuppression.
    - **Bisphosphonates or denosumab** for metastatic bone pain.
    - Always review **anticholinergic burden**, **Beers and STOPP-START** criteria, and **drug–drug and drug–disease interactions**.
4. **Procedural and interventional modalities** when drugs and rehabilitation fail — **trigger-point and intra-articular injections, epidural steroid injection, medial branch block and radiofrequency ablation, sympathetic and coeliac plexus block, intrathecal drug delivery, spinal cord stimulation, vertebroplasty and kyphoplasty**, and **palliative radiotherapy for bone metastases**. Refer to a **specialist pain clinic** if pain remains uncontrolled.

Role of Palliative Care

1. **Palliative care is not restricted to cancer or to the last days of life.** It can be delivered **alongside disease-modifying treatment from the time of diagnosis**, in any life-limiting illness — **cancer, heart failure, COPD, end-stage renal disease, dementia and frailty** — and the **best outcomes occur when it is started early**.
2. It addresses **"total pain"** — physical, psychological, social and spiritual — through an **interdisciplinary team** (physician, clinical nurse specialist, physiotherapist, occupational therapist, social worker, pharmacist, chaplain, counsellor). It **reduces unnecessary treatment and polypharmacy**, improves quality of life, smooths care transitions and averts avoidable admissions.
3. **What palliative care specifically adds to persistent pain management in old age:**
    - Application of the **WHO analgesic ladder** with adjuvants and interventional techniques
    - Expertise in **opioid titration, opioid rotation** and **alternative routes** (sublingual, transdermal, rectal, subcutaneous) when the oral route fails
    - Systematic prevention and management of **predictable opioid side effects**

- Correction of patient and family **misconceptions about addiction, somnolence and hastened death**
- **Goals-of-care discussion and advance care planning**
- **Caregiver support and bereavement follow-up**
- The guiding principle: a clinician should **never say "there is nothing more that can be done"** — that is the point at which specialist palliative care should be involved

> **INDIAN CONTEXT**
>
> LASI wave-1 documented a heavy burden of **arthritis and musculoskeletal disease** in Indians aged 45 and above. Persistent pain in Indian elders is compounded by **limited access to physiotherapy and rehabilitation**, low awareness of pain services, and **restricted opioid availability** despite the 2014 NDPS amendment, with **fewer than 4%** of those needing palliative care actually receiving it.

Latest Updates (2023–Present)

1. WHO guideline on non-surgical management of chronic primary low back pain (December 2023)

The first WHO guideline in this field, developed explicitly **for adults including older people** in primary and community care. 37 interventions were assessed, yielding 24 recommendations.

- **May be offered**: structured **education and advice**; **structured exercise therapy**; **needling therapies such as acupuncture**; **spinal manipulative therapy**; **massage** (warn about short-term increase in pain); **CBT and operant therapy**; **NSAIDs**; **topical capsaicin**; **multicomponent biopsychosocial care by a multidisciplinary team**; and **quality, affordable mobility assistive products** based on person-centred assessment.
- **Should NOT be used as routine care**: **opioid analgesics**, **SNRI antidepressants**, **tricyclic antidepressants**, **anticonvulsants**, **skeletal muscle relaxants**, **glucocorticoids**, **injectable local anaesthetics**, **traction**, **therapeutic ultrasound**, **TENS**, **lumbar braces, belts and supports**, and **pharmacological weight loss**.
- **Good practice statement**: initiate any new medicine at the **lowest effective dose for the shortest duration**, with **close monitoring for benefit and harm — especially in older people**.
- **No recommendation possible** for **paracetamol, benzodiazepines or cannabis-related preparations** — no eligible trials were identified.

2. ICD-11 chronic pain classification (in force since 2022)

The first systematic coding framework for chronic pain, adding a second axis to the mechanistic classification:

- **Chronic primary pain** — pain is the disease itself (fibromyalgia, **chronic primary low back pain, ICD-11 code MG30.02**, chronic widespread pain).
- **Chronic secondary pain** — six groups: **cancer-related, post-surgical/post-traumatic, neuropathic, headache and orofacial, visceral, and musculoskeletal**.

3. Cochrane network meta-analysis of antidepressants for chronic pain (2023)

176 trials, **over 28,000 participants**, 25 antidepressants compared.

- **Duloxetine was consistently the highest-ranked antidepressant**, with moderate-to-high certainty evidence for both substantial pain relief and reduced pain intensity.
- **Standard-dose duloxetine 60 mg was as effective as higher doses** for most outcomes.

- **Milnacipran** ranked next, with lower certainty.
- There was **insufficient evidence to draw robust conclusions for any other antidepressant**, including **amitriptyline** — despite its long-standing first-line status in neuropathic pain guidelines.

4. OPAL trial (Lancet 2023)

347 adults with acute or subacute low back and/or neck pain, randomised to **modified-release oxycodone up to 20 mg/day (with naloxone)** or placebo for up to 6 weeks, alongside guideline care.

- **No difference in pain severity at 6 weeks.**
- **Pain and quality of life at long-term follow-up were better in the placebo group.**
- **Significantly higher risk of opioid misuse at 12 months** in the opioid arm.
- Conclusion: **opioids should not be recommended for acute non-specific low back or neck pain.**

5. AGS Beers Criteria 2023 update — implications for pain prescribing

- **Avoid non-COX-2-selective NSAIDs for chronic use**, and avoid short-term scheduled NSAIDs combined with **systemic corticosteroids, anticoagulants or antiplatelets**.
- **Avoid indomethacin** (most adverse effects of all NSAIDs, including CNS effects) and **ketorolac** (oral and parenteral).
- **Avoid meperidine** — ineffective at usual doses and higher neurotoxicity and delirium risk than other opioids.
- **Avoid all skeletal muscle relaxants** — carisoprodol, chlorzoxazone, cyclobenzaprine, metaxalone, methocarbamol, orphenadrine — poorly tolerated because of anticholinergic effects, sedation and fracture risk.
- Rationale for anticholinergic drugs expanded to explicitly recognise **cumulative anticholinergic burden**.
- The accompanying **"Alternative Treatments" resource (2025)** states that for **first-line management of chronic pain, non-pharmacological approaches should be used alone or with medication**, and suggests **short-course NSAIDs, topical NSAIDs or COX-2 selective agents** instead of chronic NSAIDs or muscle relaxants.

6–8. Other developments

- **NICE chronic pain guidance (NG193)** remains the reference position for **chronic primary pain** — offer **supervised group exercise, CBT or acceptance and commitment therapy, and acupuncture**, and consider **an antidepressant**; **do not initiate paracetamol, NSAIDs, opioids, gabapentinoids, benzodiazepines, corticosteroid injections or antipsychotics**.
- **Gabapentinoid deprescribing** — accumulating evidence of harm in older adults (sedation, dizziness, **falls and fractures**, peripheral oedema, and **respiratory depression when combined with opioids**, with mandatory dose reduction in renal impairment). Prescribe **only for a defined neuropathic indication, with a planned review and stopping date**.
- **Palliative care evidence** — **early integrated palliative care** continues to show improved symptom burden and quality of life across both cancer and non-cancer illness. In parallel, the negative **BETTER-B (mirtazapine, 2024)** and **MABEL (morphine, 2025)** trials for chronic breathlessness have shifted palliative practice toward **non-pharmacological measures first** — the same direction of travel as in persistent pain.

Current Practice Update

Area	Textbook position	Current evidence and practice
Classification	Nociceptive (somatic/visceral), neuropathic, "undetermined"; nociplastic introduced	**Nociplastic pain** is now a formal third mechanistic descriptor, and **ICD-11 adds the chronic primary versus chronic secondary axis** as a parallel classification
Paracetamol	First-line agent for persistent musculoskeletal pain	Effect size is **small and often not clinically important** in osteoarthritis and back pain; **WHO could make no recommendation** for it in chronic primary low back pain. **Non-drug therapy is the true first line**
NSAIDs	Avoid, or use rarely and with great caution	Position unchanged for **chronic systemic use** (Beers 2023 reinforces this), but **topical NSAIDs are now preferred**, and a **short course of oral NSAID may be offered** for chronic primary low back pain
Antidepressants	TCAs contraindicated; SSRIs and SNRIs "more effective" for pain	**Duloxetine is the only antidepressant with reliable evidence** (60 mg as good as higher doses); evidence for amitriptyline is weak. **But indication matters — WHO advises against both SNRIs and TCAs specifically for chronic primary low back pain**
Opioids	Add when pain limits function and quality of life	Markedly more restrictive: **no benefit in acute back or neck pain (OPAL)**, **WHO advises against routine use in chronic primary low back pain**. Reserve for **cancer pain, end-of-life care, and highly selected, monitored non-cancer pain** with explicit function-based goals and an exit strategy
Muscle relaxants	Listed within the ATMAN framework (step 2)	**All skeletal muscle relaxants are on the 2023 Beers "avoid" list**; WHO also advises against them in chronic primary low back pain
TENS, traction, ultrasound, braces	Included among physical and complementary therapies	**WHO advises against routine use** for chronic primary low back pain; **exercise, education, manual therapy, massage and acupuncture are retained**
Gabapentinoids	Effective for neuropathic pain with fewer side effects than older anticonvulsants	Growing recognition of **falls, fractures, oedema and respiratory depression with opioids**; **not recommended for non-neuropathic chronic primary pain**; needs renal dose adjustment and planned review
Palliative care	Framed largely around cancer and end-of-life care	Now positioned as an **early, concurrent service across all life-limiting illness** — including frailty, dementia, heart failure, COPD and end-stage renal disease

Exam One-Liners

Chronic pain = pain >3 months, persisting beyond expected healing; it is **a disease, not just a symptom**.

Three mechanisms: nociceptive (somatic/visceral) — neuropathic — **nociplastic**.

A-delta = fast, sharp, localised; C = slow, dull, burning.

Wind-up = NMDA-mediated central sensitisation → allodynia and secondary hyperalgesia.

Gate control theory explains why TENS, rubbing and massage work.

Ageing **impairs descending inhibition** — older people are **more vulnerable to persistent pain, not less sensitive**.

Numerical rating scale is the most suitable tool in the elderly; **VAS is the least reliable**.

In advanced dementia use **Abbey, PAINAD or DOLOPLUS-2** — and treat **unexplained behaviour change as pain until proved otherwise**.

ATMAN = Acetaminophen/antidepressants, Topicals, Muscle relaxants, Anticonvulsants/anti-inflammatories, Non-drug.

Avoid meperidine, propoxyphene, indomethacin, ketorolac and all skeletal muscle relaxants in older adults.

Opioids always need a bowel regimen — constipation is never tolerated.

Duloxetine 60 mg is the antidepressant with the best evidence for chronic pain.

OPAL 2023: opioids are no better than placebo for acute back and neck pain.

WHO 2023: for chronic primary low back pain — exercise, education, CBT, acupuncture and NSAIDs **in**; opioids, antidepressants, anticonvulsants, muscle relaxants, TENS, traction and braces **out**.

Never say "nothing more can be done" — that is the cue to involve specialist palliative care.

QUESTION 4 · 15 MARKS

End-of-life care issues

Angled at the issues themselves; the symptom-control detail sits in Question 2 and is only summarised here.

High-Yield Points

I. Recognition and Prognostic Issues

1. **Failure to diagnose dying is the commonest single failure in end-of-life care.** Unless the possibility is actively considered, dying patients continue to receive **resuscitation, futile investigations and transfer out of their homes to hospital for their last hours and days**.
2. Use the **Gold Standards Framework triggers**: the **"surprise" question**, **patient choice or need** for comfort care only, and **clinical indicators** of advanced disease.
3. **Prognostic uncertainty is unavoidable.** Use **ePrognosis, Palliative Performance Scale, Karnofsky and ECOG** with clinical experience, and communicate in terms of **time, function or unpredictability**, always as **ranges**.
4. **Non-cancer trajectories are far less predictable than cancer.** Patients with **organ failure, frailty and dementia** deteriorate in a stuttering, unpredictable way and are therefore **systematically under-referred** to palliative services.

II. Communication and Decision-Making Issues

1. **Goals-of-care conversations occur far too infrequently and far too late.** Barriers cited by clinicians: **uncertainty about prognosis, fear of provoking distress, and fear of jeopardising a long-standing relationship**. The evidence is the opposite of the fear — **serious illness conversations reduce patient anxiety and depression and do not harm the doctor–patient relationship**.
2. **Structured frameworks help**: **REMAP**, the **Serious Illness Conversation Guide**, and the **five-step approach** — (i) initiate the discussion, (ii) clarify the prognosis, (iii) evaluate the patient and family, (iv) identify end-of-life goals, (v) develop a plan of care.
3. **Never say "there is nothing more we can do."** Reframe the discussion around **what will be provided** — analgesia, positioning, non-drug comfort measures, presence.
4. **Do not shift the decisional burden onto relatives.** If families are given the (incorrect) impression that the decision to withhold or withdraw treatment is **theirs to make**, they suffer **significant and lasting distress from the emotional burden**, even when consensus is reached. The decision is a **clinical recommendation made in the patient's best interests**, shared with and explained to the family.

III. Ethical Issues

1. The **four principles — autonomy, beneficence, non-maleficence and justice —** are frequently in conflict at the end of life.
2. **Withholding and withdrawing** an ineffective or unduly burdensome treatment are **morally and legally equivalent**. This permits a **time-limited trial**, and it must be explained that **death results from the underlying disease, not the withdrawal**.
3. **Futility** — clinicians are ethically empowered to decline care that is **medically futile**, but the futility principle is itself contested. Where clinician and patient or proxy disagree, options include **transfer to**

another clinician, the institutional ethics committee, social services, or patient-advocacy agencies.

4. **Palliative sedation versus euthanasia** — sedation is **titrated to the symptom** with regular review, and death results from disease. Administering high doses by continuous infusion **without titration and without considering hydration** implies a different intent.
5. **Assisted dying** — **assisted suicide** = supplying the means; **euthanasia** = administering the lethal drug. Arguments **for**: autonomy, existential distress, futility. Arguments **against**: coercion of the vulnerable, absence of watertight safeguards, imprecision of prognosis. **Three of 18 Oregon patients** given lethal prescriptions had **unrecognised, untreated depression**; **internal coercion** ("I have a duty to die") is very difficult to detect. When a patient asks, first explore **what they are actually asking**.

> **RATIONING AND AGEISM — A HIGH-YIELD ETHICAL POINT EXAMINERS LIKE**
>
> Older adults are **prime targets for rationing** because they consume disproportionate resources and are seen as having "already lived their lives". Outcome measures based on **quality-adjusted life-years or active life expectancy are inherently biased against the elderly**, and treating dependency as the primary outcome implies that **disability is equivalent to death**. Geriatricians must be **vigilant advocates** against this.

IV. Legal and Documentation Issues

1. **Advance care planning and advance directives (ACD)** — an advance decision to refuse treatment (**living will**) is binding where it is **specific to the situation, made with capacity, voluntary, informed, and not subsequently invalidated**. It takes effect **only once capacity is lost**. Review **yearly, on deterioration, and before care-home admission**. **Values rarely change over a lifetime; care decisions can.**
2. **Surrogate decision-making** — a **durable power of attorney for health care or proxy** covers situations a living will cannot (for example, whether an antibiotic is life-supporting or comfort care). Where none exists, the **best-interests standard** and, if necessary, **court-appointed guardianship** apply.
3. **POLST / MOLST** (Physician or Medical Orders for Life-Sustaining Treatment) convert the patient's advance directive into an **actionable, transferable medical order** valid across settings. Especially critical for those who **want to avoid some or all life-sustaining treatment, reside in or need long-term care, or might die within the next year**.
4. **Legislation alone changes little.** The US **Patient Self-Determination Act (1991)** requires institutions to inform patients of their rights to participate in decisions, accept or refuse treatment and prepare an advance directive — but **does not require an advance directive to be made**, and has had **minimal impact on completion rates**. Hence the shift toward **POLST/MOLST and electronic records that travel with the patient**.

SEVEN LEGAL MYTHS THAT OBSTRUCT GOOD END-OF-LIFE CARE (ACP/ASIM) — ALL FALSE

1. Forgoing life-sustaining treatment in an incapacitated patient requires proof of their actual wish
2. Withholding or withdrawing artificial fluids and nutrition is illegal
3. Risk management must be consulted before stopping life-sustaining treatment
4. Advance directives must use specific forms, are not transferable between states, and oral directives are unenforceable
5. A doctor giving high-dose analgesia that results in death will be prosecuted
6. When suffering is overwhelming despite palliative care, no legally permissible options exist
7. The 1997 US Supreme Court decisions outlawed physician-assisted suicide

1. **DNACPR** — two **separate** questions: (i) **can CPR work physiologically?** (a clinical judgement — in advanced terminal disease the chance is markedly reduced, and in one series **no terminally ill patient survived**); (ii) **is the resulting quality of life worth having?** (only the patient can answer). A discussion that implies a genuine choice where CPR is futile is **misleading**. Public expectations are distorted by **television dramas** that grossly overstate success.

V. Clinical and Therapeutic Issues

1. **Symptom burden** — pain, breathlessness, nausea and vomiting, constipation, terminal agitation and delirium, and respiratory secretions. Requires **anticipatory prescribing**, a **subcutaneous route established before swallowing fails**, **deprescribing of burdensome drugs**, and meticulous comfort care (mouth, pressure areas, bowel and bladder). Watch **iatrogenic harm** from the very drugs used — opioids, steroids, anticholinergics, metoclopramide and haloperidol.
2. **Nutrition and hydration must be considered separately. Hydration is a comfort measure** and adjunct to mouth care — **subcutaneous hypodermoclysis** is simple, very low-risk, as effective as IV for maintenance, and **grossly under-used**; avoid fluid overload. **Nutrition is emotionally charged**: exclude reversible causes of anorexia (**hypercalcaemia, hyperglycaemia, renal failure, infection**); **corticosteroids** give only a short-lived appetite effect at significant cost, **megestrol has no advantage**, and **artificial nutrition rarely improves survival or quality of life** while nasogastric feeding is uncomfortable and risks aspiration.

VI. Place of Care and Service-Delivery Issues

1. **Preferred place of death is often not achieved** — **49–100% of cancer patients wish to die at home**, yet only about **26% of cancer deaths and 20% of all deaths** occur there. Where preference had been **documented**, place of death matched it in **73%**. Arrange **beds, oxygen, commodes, drugs, a named prescriber and carers before discharge**.
2. **Care homes** — there is a **strong case for collaborative future care planning for every resident**. Residents are by definition frail with significant disease burden and often cognitive impairment; on deterioration, hospital admission offers **minimal gain in length or quality of life with substantial harm and risk**, and carries an **opportunity cost** for other patients. Document which interventions are **not** to be used (hospital admission, CPR, ventilation, artificial nutrition in advanced dementia) while retaining those that remain appropriate (for example, **antibiotics for a urinary infection**).
3. **Care pathways** — the **Liverpool Care Pathway** improved symptom control, communication with relatives and nurses' confidence, but was **withdrawn in the UK after complaints**; the fault lay in **"tick-box" application, not the pathway concept**. It was replaced by the **Five Priorities for Care of the Dying Person** and personalised plans. **If the patient improves, stop the pathway, reassess and reconsider active treatment.**

VII. Cultural and Spiritual Issues

1. **Culture defines how patients and families understand illness, suffering and dying** — including how pain is experienced and expressed. **Use of life-prolonging technology, the locus of decision-making, and truth-telling are all culturally shaped.** Where adult children ask that a diagnosis be withheld, the practical solution is to ask the patient whether **they are the kind of person who wants all the details, or would prefer a family member to handle the decisions** — this respects culture **without assuming the patient conforms to it**. Practise **cultural humility**, acknowledging one's own unrecognised biases.
2. **Spiritual concerns must be addressed explicitly, not dismissed.** Where a family holds a strong belief in a miraculous cure, a chaplain can explore it and negotiate a plan that is **respectful of the belief yet realistic** — for example, that forgoing further chemotherapy does not change whether a miracle is possible.

VIII. Family, Caregiver and Bereavement Issues

1. **Informal caregivers deliver most end-of-life care with very limited resources**, and **caregivers reporting emotional stress have significantly increased mortality**. **Grief begins at diagnosis, not at death.** Warning signs of **complicated grief**: sudden or traumatic death, loss of a child, and an **ambivalent relationship**. Bereaved relatives have increased **depression, cardiac events, intercurrent infection and mortality**; hospices provide bereavement follow-up for about **one year**. Offer families **Byock's "Five Things"**.

Latest Updates (2023–Present)

Palliative sedation

1. The **revised EAPC framework on palliative sedation (Palliative Medicine, 2024)**, an international Delphi consensus of 42 statements, reframes practice: **proportionality is built into the definition**; **"suffering" now explicitly includes physical, psychological and existential distress**; **"refractory" means untreatable (clinician view) and intolerable (patient view)**; **no fixed remaining life expectancy** is set as a criterion; **hydration is decided independently** of sedation; and **patient autonomy** is strongly emphasised, with a stepwise pharmacological approach.

Hydration at the end of life

1. **NICE guidance (NG31, quality standard QS144)** states clearly that there is **no evidence that clinically assisted hydration prolongs dying, nor that withholding it hastens death**. Practice: **assess hydration daily**, support safe oral intake, consider a **therapeutic trial** with **monitoring at least 12-hourly**, continue if benefit, and **reduce or stop if harm** or if the patient no longer wants it. **"One-size-fits-all" prescribing proformas are explicitly discouraged.**

Advance care planning

1. Systematic reviews and meta-reviews (2023–2025) show ACP reliably improves **process outcomes** — documentation, patient and surrogate engagement, and concordance between stated preferences and delivered care — but has **limited or inconsistent effect on hospitalisation, health status and survival**.
2. The field has consequently shifted from **"completing a document"** to **iterative goals-of-care communication, surrogate preparation and system-level implementation**.

End-of-life decision-making in India

1. **Supreme Court of India, 24 January 2023** (*Common Cause v Union of India*) simplified the 2018 framework: the **advance medical directive no longer requires a Judicial Magistrate's countersignature** — a **notary or gazetted officer** suffices; **more than one guardian** may be

named; approval runs through a **Primary Medical Board** and a **Secondary Medical Board** with defined **48-hour timelines**; courts intervene only in disputes.

2. The **ISCCM–IAPC Expert Consensus and Position Statement for End-of-Life and Palliative Care in the ICU (Indian Journal of Critical Care Medicine, 2024)** is now the operative Indian bedside document, providing a structured process for **foregoing life-sustaining treatment (FLST)** based on physician prognostic assessment, **documented shared decision-making**, due process consistent with the 2023 Supreme Court safeguards, and **integration of palliative care into the ICU**.
3. The **MoHFW draft guidelines for withdrawal of life support in terminally ill patients (September 2024)** operationalise the 2018 and 2023 orders, defining **terminal illness** and the **two-tier medical board pathway**.

Assisted dying — the international legal landscape is moving fast

1. In **England and Wales**, the **Terminally Ill Adults (End of Life) Bill** — which would have permitted assisted dying for mentally competent adults with a prognosis under **six months** — passed the **House of Commons on 20 June 2025 (314 to 291)** but **fell on 24 April 2026** after more than **1,200 amendments** stalled it in the House of Lords and the session was prorogued. A **successor bill was reintroduced in the Commons on 17 June 2026**, with second reading scheduled for **11 September 2026**.
2. **Jersey passed its Assisted Dying Law on 26 February 2026** — eligibility requires terminal illness with unbearable physical suffering and expected death within **6 months (12 months for neurodegenerative disease such as Parkinson's or motor neurone disease)**, assessment by **two independent doctors**, and — uniquely among jurisdictions — permits a **doctor or nurse to administer** the substance. A separate assisted dying bill is before the **Scottish Parliament**.
3. **India's position is unchanged**: **passive euthanasia and withdrawal of life support are lawful** under the Supreme Court framework; **active euthanasia and assisted suicide remain illegal**.

Access and equity

1. Indian access data (2025): **7–10 million people need palliative care annually and fewer than 4% receive it**; **44.5% of all centres are in Kerala**; **morphine is stocked in only about 66% of centres**; **median travel time to the nearest centre is 118 minutes**; and **no Indian national or state essential medicines list contains all recommended morphine formulations**.

Current Practice Update

Issue	Textbook position	Current position (2023–present)
Care of the dying	Integrated pathways (LCP) described, then withdrawn; Five Priorities introduced	Fully **individualised care plans**; **no blanket prescribing proformas**; daily reassessment of symptoms, hydration and goals
Palliative sedation	"Rarely necessary"; consent required; titrated midazolam	**Proportionality now central**; refractory suffering **explicitly includes psychological and existential distress**; **no prognostic window** required; **hydration decided separately**
Clinically assisted hydration	Comfort measure; subcutaneous route under-used	Explicit: it **neither prolongs dying nor does withholding hasten death**; **therapeutic trial with 12-hourly review**, stop if harm

Advance care planning	Emphasis on **completing a valid ACD**; POLST/MOLST as the operational fix	Documents alone change little; emphasis on **repeated goals-of-care conversations**, surrogate preparation and organisational implementation
DNACPR communication	UK position: **no legal or moral requirement** to inform the patient of a DNACPR decision	Practice has moved decisively to **transparent discussion and documentation**; ISCCM–IAPC **requires documented shared decision-making**
Legal framework	UK, US and Australia-centric (Mental Capacity Act, PSDA, POLST, Victorian VAD Act)	**India now has an operative framework** — Supreme Court 2023 order + ISCCM–IAPC 2024 + MoHFW 2024 draft guidelines
Assisted dying	Debate framed around Oregon, the Netherlands and Victoria	Rapidly evolving — **UK bill fell April 2026 and has been reintroduced; Jersey legalised February 2026; Scotland's bill pending**. India: passive euthanasia lawful, active euthanasia and assisted suicide **not**
Artificial nutrition	Rarely beneficial; NG feeding uncomfortable and risks aspiration	Reinforced — **"comfort feeding only"** is the preferred humane alternative; tube feeding does not improve survival or quality of life in advanced dementia
Terminal delirium	Low-dose haloperidol after treating reversible causes	**Non-pharmacological measures first**; antipsychotics reserved for severe distress, lowest dose, shortest time
Breathlessness at EOL	Opioids are "the main therapy"	Benefit more modest than believed (**MABEL 2025 negative**); **mirtazapine not recommended** (BETTER-B 2024); non-drug measures first-line

Exam One-Liners

> **End of life = last 12 months; terminal phase = last days to hours.**

> **Withholding = withdrawing** — this equivalence is what permits a **time-limited trial**.

> **An advance directive takes effect only after capacity is lost.**

> **POLST/MOLST** turns an advance directive into an **actionable, transferable medical order**.

> **The Patient Self-Determination Act obliges institutions to inform, not patients to complete.**

> Do not tell relatives the decision is theirs — that transfers a **lasting emotional burden**.

> **Hypodermoclysis** — simple, safe, as effective as IV, and grossly under-used.

> **Palliative sedation is titrated to the symptom** — that is what separates it from euthanasia.

> **QALY-based rationing is inherently biased against older people** — disability is not death.

> **Cultural humility** — ask the patient how much they want to know, rather than assuming.

> **Care-home residents deserve future care planning by default**, not only when they deteriorate.

> **India's operative documents**: Supreme Court January 2023 order + ISCCM–IAPC 2024 + MoHFW 2024 draft guidelines.

> **Never say "nothing more can be done"** — that is the cue for specialist palliative care.

QUESTION 5 · 15 MARKS

Hospice and respite care

Part 1 — Hospice Care

Definition and Concept

1. **Hospice is a comprehensive care system for patients with limited life expectancy**, living at home or in institutional settings. It is simultaneously a **philosophy of care** and an **organised, highly structured system for delivering care**, whose defining focus is **caring, not curing**.
2. **Hospice is a subset of palliative care** offered to patients **approaching the end of their lives**. Palliative care ideally begins **from the onset of serious life-threatening illness**, alongside disease-modifying treatment; **hospice enrolment is the final piece of that continuum**.

Feature	Palliative care	Hospice
Timing	Any stage, from diagnosis onward	Final phase of illness
Prognosis required	No	Yes — **physician-certified ≤6 months (US)**
Disease-modifying treatment	Continued concurrently	Generally relinquished
Setting	Any care setting	Home, hospice facility, hospital, nursing home, assisted living
Funding (US)	Usual insurance	Defined **Medicare/Medicaid hospice benefit**
Team	Interdisciplinary	Interdisciplinary + **mandatory trained volunteers**

Historical Evolution

1. The term **hospice** was coined by the British physician **Dame Cicely Saunders in 1967** (St Christopher's Hospice, London). **Florence Wald**, Dean of the Yale School of Nursing, brought the model to the United States. In **1979 the Health Care Financing Administration funded 26 hospices as a demonstration programme**, and in **1982 hospice was added as a benefit under Medicare and Medicaid**; it is now a standard benefit of all US health insurance plans. India's first hospice was **Shanti Avedna Sadan, Mumbai (1986)**.

Eligibility and Election

1. Two conditions must be met: (i) the patient's **primary clinician certifies a remaining life expectancy of approximately six months or less**; and (ii) the patient **elects hospice** and **agrees to accept care from an identified hospice team**. The patient's own physician may continue to provide care and work alongside the hospice team.

Services Provided

1. **The hospice service package:**
   - **Nursing** — registered nurses coordinate all care, provide direct care, monitor symptoms and medication, educate patient and family, and act as the **link between the family and the physician**
   - **Social services** — advice and counselling during the crisis period, interpretation of family dynamics for the team, and **advocacy** in accessing community resources
   - **Physician services** — the patient's own physician approves the plan of care; a **hospice medical director** serves as consultant and resource
   - **Spiritual care and counselling** — clergy and counsellors visit at home; churches and congregations engaged as requested
   - **Home health aide and homemaker** — bathing, shampooing, shaving, nail care; light housekeeping and meal preparation
   - **Continuous home health care** — hospice staff provide care in **8- to 24-hour periods on a short-term basis** when the patient's needs escalate or the family can no longer manage round-the-clock care
   - **Therapy and rehabilitation** — helping the patient find new ways to walk, dress or feed themselves
   - **On-call team support** — a team member available **24 hours a day, 7 days a week**, by phone or visit
   - **Respite care** — the hospice arranges a **brief period of inpatient care to give relief to family members**
   - **Bereavement counselling** — support through grieving, with a trained volunteer or counsellor visiting at set intervals **during the first year**, plus telephone and letter contact, support groups, and onward referral if needed
   - **Supplies and medications** are covered (for example, a bedside commode)
2. **The interdisciplinary team (IDT)** comprises the patient's personal physician, the hospice physician or medical director, nurses, hospice aides, social workers, bereavement counsellors, spiritual care providers, and **trained volunteers**. Hospice is unique in that **volunteers are required to provide at least 5% of total patient-care hours**.

Settings, Utilisation and Limitations

1. **Most hospice care is delivered wherever the patient calls home**, but it is also provided in **freestanding hospice facilities, hospitals, nursing homes, assisted living and other long-term care facilities**. Hospice is available to patients with **any terminal illness, of any age, religion or race**.
2. **Utilisation data (2017, US)** — over **4,515 hospice agencies** served **1.49 million Medicare beneficiaries**; **40% were served at home, 19% in long-term care**; **58% were female**; **48% of all Medicare decedents were enrolled in hospice at the time of death**, with an average length of stay of **76 days**. This indicates hospice remains **substantially under-utilised and entered too late**.
3. **The diagnostic mix has shifted** — cancer is the principal hospice diagnosis in only about **30%** of enrolees, having fallen as hospice use for **non-malignant terminal illness such as heart disease and dementia** has risen.
4. **Key practical limitation** — hospice agencies provide nursing, therapy, social services and family support in the home, but are **not capable of providing continuous 24-hour personal care**. Personal-care aides are **not paid for by Medicare or by most private insurance plans**.

5. **Referral is often too late.** Barriers include prognostic uncertainty, clinician reluctance to discuss dying, and the perception among patients and families that enrolment means "giving up". Clinicians must **never say "there is nothing more that can be done"** — that is precisely the moment to introduce hospice.

Part 2 — Respite Care

Definition and Rationale

1. **Respite care is the temporary — planned or emergency — provision of care for a dependent older person, in order to give the usual informal caregiver a break.** Its primary target is **the caregiver**, although the care recipient also benefits.
2. **The caregiver burden that justifies it:**
   - Caring for a relative with dementia or chronic illness is **physically, emotionally and financially stressful**
   - Approximately **35% of those caring for a person aged 50 or over report moderate-to-high financial strain**; about **25% provide more than 40 hours of care per week**
   - Caregivers develop **depression, anxiety, musculoskeletal pain worsened by caregiving tasks, social isolation and loneliness**
   - Many **exit the workforce early**, lose pension and retirement funds to medical costs, and find it hard to re-enter employment
   - **Caregivers reporting emotional stress have significantly increased mortality**
   - Distress **does not end at institutionalisation** — anxiolytic use by caregivers increases, and **nearly half are at risk of disabling depression after placing a relative in long-term care**; almost half of spouses visit daily and continue to provide physical and emotional care
3. **Assess the caregiver formally**, not just impressionistically:
   - **Zarit Burden Interview (ZBI)** — original 29-item, commonly used 22-item, with **12-, 4- and 3-item short versions**; developed for dementia caregivers, now broadened, and validated across many languages and cultures
   - **Caregiver Self-Assessment Questionnaire (CSAQ)** — 18 items, developed by the AMA; effective at detecting **depressive symptoms**
   - **Rapid Screen for Caregiver Burden** — 7 items (from the 25-item original)
   - **Caregiver Well-Being Scale** — a **strengths-based** 43-item tool, with 16-item and 6-item rapid versions

Types of Respite

1. **Forms of respite care:**
   - **In-home respite** — a paid aide or trained volunteer attends the home for a few hours
   - **Adult day care** — supervised recreational and restorative activity for people with **limited functional ability**; some programmes are specifically for the **cognitively impaired**. Improves basic **ADL and social skills**. Most programmes serve a given client **2–3 days a week rather than 5**, to serve more families
   - **Adult day health centre** — an intermediate model combining day care with **nursing, physiotherapy and social work**; may also host periodic ambulatory clinics
   - **Day hospital** — larger medical component, with virtually all hospital services on an ambulatory basis; emphasis on **rehabilitation, physiotherapy and occupational therapy**

- **Institutional / short-stay respite** — planned admission to a care home, community hospital or hospice bed for days to weeks
- **Hospice inpatient respite** — a brief inpatient stay arranged by the hospice expressly to relieve the family
- **Emergency or crisis respite** — when the carer becomes ill or breaks down

Benefits and Place in Care

1. **Respite provides important relief for the primary caregiver and may make the critical difference in allowing an impaired older person to remain at home** rather than be institutionalised. Where **carer stress is high**, **intensive home support or admission to a respite or step-down placement is a better option than hospital admission**, which is disorientating for people with dementia and **carries a higher risk of unnecessary long-term institutionalisation**. Less restrictive options should always be considered first.
2. **Respite is one component of a multidimensional caregiver-support package.** Effective programmes combine **education and psychoeducation, counselling, support groups, problem-solving and behaviour-management training, cognitive behavioural therapy, mindfulness-based stress reduction, telephone support, practical skills training, contingency planning, and respite or adult day care**. A **support group is hard to attend unless care for the recipient is provided at the same time** — a practical argument for pairing the two.
3. **Service context.** Respite sits within the community long-term care continuum alongside **home nursing and homemaking, adult foster care, assisted living, geriatric assessment, hospice, telephone reassurance, caregiver support, congregate housing, home repairs, meals services and emergency alarms**. In the UK, **day hospitals for older people have largely been decommissioned** because the supporting literature was sparse — one of their primary roles had been **carer support and respite for people with dementia and challenging behaviour**. **Voluntary, charitable and private-sector day care and respite services have expanded** as day hospitals closed.
4. **Barriers and practical advice** — services vary widely by community, budgets are constrained, and families often do not know what exists. **Families should be actively encouraged to seek respite periodically, at regular intervals, rather than only at the point of crisis. Where no formal respite programme exists, informal arrangements can usually be made to relieve the primary caregiver for short periods** — and such relief materially helps the caregiver cope. Signposting to the **Alzheimer's Association local chapter and the Area Agency on Aging** (or their national equivalents) is a core clinical task.

> **INDIAN CONTEXT**
>
> Care of dependent elders is overwhelmingly familial, and there is **no organised national respite system**. Day care centres and short-stay facilities are largely **NGO, charitable and urban**. **Kerala's Neighbourhood Network in Palliative Care** provides de facto community respite through trained volunteers. The policy backdrop is the **National Programme for Health Care of the Elderly (NPHCE)** and the **Maintenance and Welfare of Parents and Senior Citizens Act 2007 (amended 2019)**, with palliative and hospice services reaching **fewer than 4%** of those who need them.

Latest Updates (2023–Present)

Hospice utilisation

1. **Facts and Figures 2025** (National Alliance for Care at Home, successor to NHPCO), reflecting **calendar year 2024** data:
   - **1.91 million Medicare beneficiaries** enrolled in hospice for one or more days — a **4.4% increase on 2023**; **1.3 million died while in hospice**
   - **Historic milestone: 53.1% of all Medicare decedents received hospice care and were enrolled at the time of death** — the **first time a majority**, the third consecutive annual rise, and above pre-pandemic levels
   - **Principal diagnoses**: **circulatory 29.8%, neurovascular 25.4%, cancer only 22.3%, respiratory 10.1%, kidney disease 3.0%**
   - (The 2024 edition, reporting CY2022, recorded 1.72 million enrolees and **49.1% of decedents** — the first increase since 2019.)

> **THE FOUR LEVELS OF THE MEDICARE HOSPICE BENEFIT — HIGH-YIELD**
>
> **1. Routine home care (RHC)** — the commonest level, roughly **52% of all patient-days**
> **2. Continuous home care** — for symptom crisis managed at home
> **3. Inpatient respite care** — **maximum 5 consecutive days**, expressly to relieve the caregiver
> **4. General inpatient care (GIP)** — for symptom crises that cannot be managed elsewhere
>
> Payment at each level covers **all care related to the terminal illness** — interdisciplinary team, medication, equipment and supplies.

Hospice ownership, quality and oversight

1. **Commercialisation** — in **1992 about 5% of Medicare-certified hospices were for-profit; by 2024 approximately 73%** were. A two-part **HHS Office of Inspector General report** found that around **20% of hospice agencies had deficiencies posing serious safety risks to patients**.
2. **Hospice Special Focus Program (SFP)** — mandated by the Consolidated Appropriations Act 2021 and finalised in the CY2024 rule, the SFP used a **data-driven algorithm** (survey findings, complaint data and quality measures) to identify the poorest-performing roughly 10% of hospices for **more frequent survey, mandatory quality improvement, loss of accreditor "deemed status", and possible termination from Medicare**. After an initial list of 50 hospices was released and a **multi-state provider lawsuit** was filed in January 2025, **CMS ceased implementation on 14 February 2025** to re-evaluate the methodology, and confirmed it would not resume during 2025. **Hospice quality oversight is now an actively contested policy area.**

Respite care evidence

1. **Updated systematic review of adult day services as respite (2015–2025, more than 25 studies)**:
   - Regular use is associated with **reduced caregiver stress, improved mood, and measurable reduction in physiological stress markers**; on day-service days caregivers report **lower negative affect and higher positive affect**
   - Participation is linked to **delayed nursing home placement**, translating into **cost savings**
   - **Earlier Cochrane reviews were inconclusive** because of limited randomised data; **recent observational and longitudinal studies provide stronger evidence of benefit**
   - **Utilisation remains suboptimal** because of **financial constraints, limited awareness, caregiver reluctance and service availability**; facilitators are **caregiver education, culturally tailored programming, financial assistance and high-quality staff engagement**

2. **Caregiver-intervention meta-analyses** confirm that **multicomponent interventions and respite delay institutionalisation**, but that effect sizes on caregiver outcomes are generally **small to very small**. This has produced a proposed **"tripartite scaffolding" model of caregiver support**: (a) **care coordination and case management**, (b) **psychoeducation**, and (c) **skills training** — with respite embedded rather than standing alone.
3. **Service innovation** — nature-based, community-linked and technology-supported respite models are now being formally evaluated using **DEMQOL** (quality of life) and the **Zarit Burden Interview**, with implementation frameworks such as RE-AIM.

Current Practice Update

Area	Textbook position	Current evidence and practice
Hospice utilisation	48% of Medicare decedents; 1.49 million enrolees (2017); mean length of stay 76 days	**53.1% of Medicare decedents (2024)** — a majority for the first time; **1.91 million enrolled**. Stays remain short, so **late referral persists as the core problem**
Principal hospice diagnosis	Cancer about 30%, falling as heart disease and dementia rise	**Cancer now only 22.3%**; circulatory (29.8%) and neurovascular (25.4%) lead — hospice is now **predominantly a non-cancer service**
Ownership and oversight	Not addressed	**About 73% for-profit by 2024**; OIG found serious deficiencies in roughly 20% of agencies; **the Special Focus Program was created and then suspended in February 2025** amid litigation over its algorithm
Hospice respite	Described qualitatively as "a brief period of inpatient care to relieve the family"	Codified as a **distinct payment level — inpatient respite care, maximum 5 consecutive days per episode**
Respite evidence base	Presented as clearly beneficial for caregiver relief and for delaying institutionalisation	**Direction of benefit confirmed, but effect sizes are small**. Strongest current data are for **adult day services** — reduced stress, improved affect, delayed placement, cost savings. Respite works best **embedded in a multicomponent package**, not alone
Day hospitals	Widely available in the UK, then decommissioned for want of supporting literature	Replaced by **voluntary, charitable and private day care and respite services**, plus crisis resolution and home treatment teams
Caregiver assessment	ZBI, CSAQ, Rapid Screen for Caregiver Burden	**Strengths-based tools (Caregiver Well-Being Scale)** used alongside burden scales; **3-item ZBI** now validated for rapid clinic screening
Access	Framed around US Medicare and UK NHS models	In India, hospice and respite remain **largely NGO-based, urban and severely limited** — under 4% of those needing palliative care receive it

Exam One-Liners

Hospice = caring, not curing — a **subset of palliative care** for patients approaching death.

Cicely Saunders coined "hospice" in 1967; **Florence Wald** brought it to the US; **Medicare hospice benefit 1982**; **India's first hospice — Shanti Avedna Sadan, Mumbai, 1986**.

US eligibility = **physician-certified prognosis ≤6 months + the patient's own election**.

Four Medicare levels: routine home care — continuous home care — **inpatient respite (max 5 days)** — general inpatient care.

Hospice is the **only Medicare provider required to have volunteers deliver at least 5% of patient-care hours**.

Hospice cannot provide continuous 24-hour personal care, and Medicare does not pay for personal-care aides.

Bereavement follow-up for about one year is a core hospice service.

Cancer is now a minority of hospice diagnoses — heart disease and dementia dominate.

Respite care targets the caregiver, not the patient.

Respite may be **the single intervention that keeps a dependent older person at home**.

Where carer stress is high, respite or step-down placement beats hospital admission — admission risks permanent institutionalisation.

Zarit Burden Interview is the standard caregiver burden tool (22-, 12-, 4- and 3-item versions).

Caregiver distress does not end at institutionalisation — nearly half remain at risk of disabling depression.

If no formal respite programme exists, arrange informal relief at regular intervals — before the crisis, not after.

QUESTION 6 · 15 MARKS

Bereavement

High-Yield Points

Definitions and Concept

1. **Bereavement** is the objective state of having lost someone significant through death. **Grief** is the personal emotional, cognitive, physical and behavioural reaction to that loss. **Mourning** is the publicly and culturally patterned expression of grief — rituals, funerals, observances.
2. **The process of grief begins from the time a life-limiting illness is diagnosed** or health starts to deteriorate — not at the moment of death. For elderly couples who have lived together many years this marks **the beginning of a very painful parting**, and for the surviving partner **the loss of a lifelong companion is equivalent to part of their own personal dying**.
3. **Bereavement is formally the final stage of the palliative care continuum** — palliative care runs from diagnosis, through illness progression and end-of-life or hospice care, and culminates in **bereavement support for family and caregivers** (as staged by the Palliative Care Outcome Collaboration, Australia). Such relatives themselves require a **care plan that includes bereavement**.

Epidemiology and Significance in Old Age

1. Bereavement is near-universal in later life — spouse, siblings, friends, sometimes children. **Losses of family and friends** are listed among the **social factors predisposing older people to depression**, alongside isolation, loss of job and loss of income.
2. **Bereavement is associated with declines in health, increased utilisation of healthcare resources, and an increased risk of death.** Grieving relatives have increased rates of **depression, cardiac events, intercurrent infections and mortality**.
3. **Recent death of a spouse and widowhood are major suicide risk factors in older people.** The high-risk profile is: **male, white, widowed or divorced, recently bereaved, living alone, isolated, recently moved, retired, poor physical health, terminal illness, pain, depression, alcohol use and loneliness.** Older adults **give fewer warnings, use deadlier methods, and have an attempt-to-completion ratio of about 4:1 (versus 200:1 in adolescents)** — and **20–50% have seen their primary care physician in the week before suicide**.

Normal versus Complicated Grief

1. **Normal (uncomplicated) grief** presents as waves of yearning, sadness, tearfulness, sleep and appetite disturbance, transient hallucinations of the deceased, guilt and anger — gradually attenuating over months as the person re-engages with life. **Most bereaved people recover with the support of friends and family and need no professional intervention.**
2. **Complicated (prolonged) bereavement may be distinct from major depression**, and formal criteria have been proposed. Its features are:
    - extreme levels of **"traumatic distress"**
    - **numbness**
    - a feeling that **part of oneself has died**
    - **assuming the symptoms of the deceased**
    - **disbelief or bitterness**

- **symptoms enduring beyond six months**

It is listed among the **depressive disorders** in geriatric psychiatry and is one of the conditions in older adults that **does not fit the conventional paradigm of disease**.

3. **The DSM-5 "bereavement exclusion" (criterion E of DSM-IV) has been removed.** The presence of depression during a period of mourning is now judged **entirely by the clinician, regardless of the period of bereavement**. This is of great importance in geriatrics because **a large proportion of older people are dealing with recent loss** and were previously denied both diagnosis and treatment.

Distinguishing the three entities:

Feature	Normal grief	Prolonged / complicated grief	Major depression
Core affect	Yearning for the deceased, in waves	**Persistent yearning and preoccupation with the deceased**	Pervasive low mood and anhedonia
Self-view	Preserved	Identity disruption — "part of me has died"	**Global worthlessness and guilt**
Pleasure	Intermittently retained	Retained outside grief triggers	**Pervasive anhedonia**
Course	Attenuates over months	Persists **beyond 6–12 months** and disabling	Sustained; may be independent of the loss
Suicidality	Passive wish to join the deceased may occur	Wish to join the deceased	**Active ideation with plan**
Treatment	Support and normalisation	**Grief-focused psychotherapy**	Antidepressant with or without psychotherapy or ECT

Risk Factors

FOUR DOCUMENTED WARNING SIGNALS FOR COMPLICATED GRIEF

1. Sudden unexpected death · 2. Traumatic death · 3. Loss of a child · 4. Loss of a partner with an ambivalent relationship ("can't live with, can't live without").

Additional risks: pre-existing depression or anxiety, poor social support, prior caregiver strain, multiple concurrent losses, financial insecurity, and the person's **own previous experience of being bereaved**.

1. **How a person dies shapes how the survivor grieves.** As Dame Cicely Saunders said, "*The way people die remains in the memory of those who live on.*" Until about 20 years ago morphine was rarely used in dying patients because of a mistaken belief that it hastened death; many were left with unrelieved pain and distress, and **those memories still colour older people's perception of death and their fears about their own dying**.

Prevention — Care Before the Death

1. **The most effective bereavement intervention is good care of the dying person.** Relatives must be **made aware of what is happening, encouraged to be involved in care, and have their concerns listened to**. Even when frail and elderly, or a child, they may want to be at the

bedside of the person they love — **to deny them the ability to be present does them a great disservice**.

2. **Identify vulnerable caregivers before the death. Caregivers reporting emotional stress have a significantly increased mortality rate**; physician identification of vulnerable caregivers and referral to social workers and community resources **can be lifesaving**.
3. **The period immediately after hospice referral is one of particular vulnerability** — patients and families feel **abandoned by their physicians**. Make follow-up appointments, telephone the family, **express thanks and appreciation for the privilege of caring for the patient**, and say goodbye to patient and family where appropriate. Clinicians must **be aware of both normal and complicated grief and screen for them appropriately**.

Assessment

1. At follow-up, screen for sleep and appetite change, weight loss, alcohol use, functional decline, financial and social change, and **suicidal thoughts**. Use standard instruments — **Geriatric Depression Scale (GDS-15/30), PHQ-2 and PHQ-9, Cornell Scale for Depression in Dementia** — and for grief specifically the **Inventory of Complicated Grief**. Also screen for **loneliness and social isolation**, which are highly prevalent in older adults and independently associated with **increased mortality and functional decline**; **routine screening with individual and community-based interventions is recommended**.

Management

1. **Most bereaved people need support, not treatment.** Blanket counselling for everyone is neither necessary nor effective — **target intervention at those with risk factors or established prolonged grief**.
2. **Psychological therapies significantly reduce grief symptoms** in complicated bereavement: **brief dynamic psychotherapy, traumatic grief therapy, crisis intervention, and support groups**. Grief-specific therapy combines elements of interpersonal therapy and CBT — revisiting the loss, cognitive restructuring, and restoring relationships and personal goals.
3. **Antidepressants may also be considered** to treat complicated bereavement, and are indicated where **major depression** is present. Apply late-life prescribing rules — SSRIs first, **start low and go slow**, review regularly. **Avoid benzodiazepines**, which do not treat grief and add falls, delirium and dependence.
4. **Spiritual and religious support matters.** Religiosity and spirituality **provide a sense of meaning that buffers stress and assists coping**, and **a network of like-minded people who serve as social resources**, promoting self-esteem and personal growth. Referral to a chaplain or the person's own clergy should be routinely considered. Healthcare professionals have **a moral obligation to address spiritual concerns** — doing so signals concern for the whole person.
5. **Hospice bereavement services** — hospice programmes provide bereavement follow-up for **approximately one year** after the death: a trained volunteer or counsellor visits the survivors at specific intervals, plus telephone and letter contact, the opportunity to join **support groups**, and **onward referral to medical or professional care** where necessary.
6. **Byock's "Five Things"** — *thank you; forgive me; I forgive you; I love you; goodbye* — offered to families before the death, ease the grief that follows.

Special Groups and Context

1. **Bereaved grandchildren.** For many children a grandparent is **the most secure adult in their life**, as social disintegration and mobility may leave the child very dependent on them. On average, **in every UK school class about two children are bereaved of someone close such as a grandparent, and one of a parent or sibling**. The way clinicians care for the dying patient and their family therefore **influences the next generation's ability to cope with illness and death**.

2. **Bereavement and sexuality** — **depression, bereavement and grief reactions** are recognised **psychological inhibitors of sexual activity** in older people, alongside poor self-image, cultural beliefs and social isolation.

> **INDIAN AND CULTURAL CONTEXT**
>
> Mourning in India is highly ritualised and time-structured (10–13 day rites, annual observances), and these rituals are **protective** — providing social scaffolding and explicit permission to grieve. Grief must be judged against **the person's own societal, cultural and religious norms**, not a Western template. Practically, **widowhood in India carries social and economic disadvantage**, family ties are strong so depression in one member strongly affects the rest, and **formal bereavement services are almost entirely absent outside hospice and NGO programmes**.

Latest Updates (2023–Present)

Prolonged Grief Disorder is now a formal diagnosis

1. **Prolonged Grief Disorder (PGD)** has been added to both **ICD-11** and **DSM-5-TR (2022)** — the single most important change in this field. Core features: **yearning for, or persistent preoccupation with, the deceased**, accompanied by **intense emotional pain** (sadness, anger, bitterness, disbelief, **identity disruption**, avoidance of reminders, emotional numbness, **loss of meaning and purpose**, difficulty re-engaging socially), causing **clinically significant functional impairment**, and persisting **beyond what is expected given the person's social, cultural or religious norms**.
2. **The two systems differ in their time criterion — a favourite exam point:**
    - **ICD-11** — the reaction must persist for an atypically long period, **minimum 6 months**
    - **DSM-5-TR** — the death must have occurred **at least 12 months ago** (6 months for children and adolescents), with symptoms present nearly every day for at least the past month
    - Consequently the **prevalence differs**: point prevalence among the bereaved ranges roughly **4.7–6.8%**, with **DSM-5-TR yielding significantly lower rates than ICD-11**. Raising the ICD-11 threshold to 12 months brings the two into near-perfect agreement.
3. **The time thresholds are now actively contested.** A cross-national study (UK, Ireland, Ontario, 2025) found grief symptoms were **not markedly higher in those bereaved within the previous 6 months**, and that bereaved people themselves **do not regard 6 months as the normative point at which one should have come to terms with a loss**. The argument advanced is that **grief symptoms are pathological by their nature, not by their chronicity** — so rigid time thresholds are unlikely to prevent false-positive diagnoses but will **increase false negatives**, with under-treatment as the practical consequence.
4. **Criterion validity favours ICD-11.** In longitudinal analysis controlling for depression and insomnia, **ICD-11 prolonged grief symptoms predicted quality of life at 6-month follow-up, whereas DSM-5-TR symptoms did not**.
5. A **2025 Lancet Review** endorses the current conceptualisation: PGD is a **diagnosable mental health condition** with core symptoms of **yearning and/or preoccupation**, associated with **emotional pain, identity disturbance, loss of meaning and purpose, and functional impairment**, with a **typical onset 6 to 12 months after the death**.

Treatment

1. **Psychotherapy is first-line, and it must be grief-specific.** Grief-focused CBT and **Prolonged Grief Disorder Therapy (PGDT, formerly Complicated Grief Therapy)** — combining **exposure and revisiting the loss, cognitive restructuring, behavioural activation and restoration of**

personal goals — have the strongest evidence. Internet-delivered CBT is effective, with **more sessions producing greater benefit**.

2. **Bryant et al., JAMA Psychiatry 2024** — head-to-head randomised trial of **grief-focused CBT versus mindfulness-based cognitive therapy** in 100 adults with PGD:
    - **Both treatments produced substantial symptom reduction** (large effect size)
    - **Grief-focused CBT was superior at 6-month follow-up** for **core PGD symptoms, depression and grief-related cognitions** — though not for anxiety
    - Interpretation: mindfulness works, but **grief-specific exposure and cognitive work works better**
3. **Pharmacotherapy is secondary.** The landmark four-arm randomised trial (**citalopram + clinical management, citalopram + PGDT, placebo + PGDT, placebo + clinical management**; 333 patients aged 18–95) established that **PGDT drives the benefit — an antidepressant alone is not sufficient**. A 2025 machine-learning re-analysis found that **higher baseline depression severity, greater grief-related functional impairment, and not receiving PGDT** each predicted poorer response. **Tricyclic antidepressants and benzodiazepines have not proved effective for PGD.**
4. **New neurobiological hypothesis** — PGD may involve the **reward and attachment circuitry** rather than classical depression pathways, i.e. it may be conceptually closer to a **disorder of attachment or addiction**. This underpins randomised, triple-blinded, placebo-controlled trials of **oral naltrexone** (opioid receptor antagonist, 8-week course) for PGD. The evidence remains **preliminary and not yet practice-changing**.
5. **Validated diagnostic instruments now exist** for both criteria sets — notably the **Aarhus Structured Clinical Interview for Prolonged Grief Disorder (A-PGDi, 2024)**, covering both ICD-11 and DSM-5-TR.

Current Practice Update

Area	Textbook position	Current position (2023–present)
Nomenclature	"Complicated bereavement"; formal criteria "have been proposed"	**Prolonged Grief Disorder is now a formal, coded diagnosis** in both ICD-11 and DSM-5-TR (2022)
Time criterion	Symptoms enduring for **six months**	**ICD-11 at least 6 months; DSM-5-TR at least 12 months** — and both thresholds are now openly challenged as arbitrary, risking **under-diagnosis**
Prevalence	Not quantified	Roughly **4.7–6.8%** of bereaved adults, varying with the algorithm; DSM-5-TR yields substantially lower rates
Relation to depression	"may be distinct from major depression"	**Confirmed as a distinct disorder** with its own criteria, trajectory, treatment response and probable neurobiology
First-line treatment	Brief dynamic psychotherapy, traumatic grief therapy, crisis intervention, support groups; "antidepressants may also be considered"	**Grief-focused CBT and PGDT are first-line**, and are **superior to non-specific therapy**; **antidepressants alone are not sufficient** — the psychotherapy drives the benefit

Mindfulness-based therapy	Not addressed	Effective, but **significantly less effective than grief-focused CBT** (Bryant 2024)
Pharmacology	Antidepressants may be considered	SSRIs only where depression coexists; **TCAs and benzodiazepines ineffective for grief itself**; **naltrexone under investigation** on a reward-circuitry hypothesis
Diagnosis in practice	Clinical judgement, Inventory of Complicated Grief	**Validated structured interviews now available** (for example A-PGDi) for both criteria sets
Cultural framing	Implicit	Now **explicit in the criteria** — the reaction must exceed the person's own **social, cultural and religious norms**; guidance warns against applying Western-normed timeframes
DSM bereavement exclusion	Noted as removed, with impact "yet to be settled"	Settled — **depression during mourning is diagnosed and treated on clinical grounds**, and prolonged grief is coded separately alongside it

Exam One-Liners

Bereavement = the fact of loss; grief = the reaction; mourning = the culturally patterned expression.

Grief begins at diagnosis, not at death.

Bereavement is the **final stage of the palliative care continuum**.

Bereavement raises **healthcare use, cardiac events, infection risk and mortality**.

Widowhood and recent spousal death are major suicide risk factors, especially in older men.

Older adults' **suicide attempt-to-completion ratio is about 4:1** (versus 200:1 in adolescents).

Four red flags for complicated grief: sudden death — traumatic death — loss of a child — ambivalent relationship with the deceased.

Complicated grief hallmark: **feeling that part of oneself has died** and **assuming the symptoms of the deceased**.

The DSM-5 bereavement exclusion has been removed — judge depression during mourning clinically.

PGD time criterion: ICD-11 at least 6 months, DSM-5-TR at least 12 months; core symptom is **yearning or preoccupation**.

Grief-focused CBT / PGDT beats antidepressants and beats mindfulness.

> **Benzodiazepines have no role in grief.**

> **Hospice bereavement follow-up runs for about one year.**

> **"The way people die remains in the memory of those who live on"** — Cicely Saunders.

> How we care for the dying patient shapes **the next generation's** ability to cope with death.

MD GERIATRIC MEDICINE

High-Yield Examination Answers

Perioperative Medicine · Periprocedural Care · Constipation
Viral Hepatitis · Liver Transplantation · Eye Donation · Sleep Protocol

Primary sources: **Pathy's Principles and Practice of Geriatric Medicine, 6th edition** | **Essentials of Clinical Geriatrics, 7th edition**
Supplemented with current guidelines and peer-reviewed literature (2023 – 2026)

Eight questions · 15-mark format · Structured for night-before revision

Contents

1.	Perioperative / Pre-operative Care and Assessment of the Elderly
2.	Checklist for Older Adults Undergoing Diagnostic and Therapeutic Procedures — Periprocedural Medical Issues (including Antiplatelet Management)
3.	Constipation in the Elderly — Prevention and Management
4.	Recent Trends in the Treatment of Hepatitis C Infection
5.	Treatment of Chronic Hepatitis B Infection — Older and Newer Modalities
6.	Liver Transplantation
7.	Eye Donation
8.	Sleep Protocol

How to use this document

- Each answer runs: **Question → High-Yield Points → Latest Updates (2023–Present) → Current Practice Update → Examiner's Tip**.
- Textbook-derived content sits in the numbered High-Yield Points. Content added from guidelines and journals is confined to the **Latest Updates** and **Current Practice Update** sections, so the two are never mixed.
- Where the uploaded textbooks do not cover a topic, this is stated explicitly in an italic note beneath the question heading.
- Laboratory values are given in conventional/Indian units throughout.

Q1. Perioperative / Pre-operative Care and Assessment of the Elderly

High-Yield Points

Principles and Risk

1. Perioperative care of an older person means three things — **pre-operative risk assessment and optimisation, intra-operative safety, and post-operative recovery**. It is a team job involving the physician, surgeon and anaesthetist.
2. **Age above 70 raises the risk** of complications and death after major non-cardiac surgery. But the actual mortality is low even beyond 80 years if the surgery is planned.
3. **Comorbid conditions matter far more than age itself.** The type of operation, the local complication rate, and whether surgery is emergency or elective decide the outcome.
4. **Emergency surgery carries a much higher risk** than elective surgery. Wherever possible, stabilise the patient and operate electively.
5. Always ask first whether the operation is appropriate. The **risk–benefit ratio** must be weighed against the patient's goals, life expectancy and functional status.

What to Assess and Optimise Before Surgery

6. Optimise underlying disease before non-emergency surgery: **cardiovascular disease (especially heart failure), lung function, kidney function, diabetes, thyroid disease (often occult), anaemia, nutrition, and hydration/volume status** — the last is especially important in patients on diuretics.
7. **Cardiac conditions that greatly increase risk** are: myocardial infarction within 6 months, pulmonary oedema, angina (especially unstable), and severe aortic stenosis.
8. The **Revised Cardiac Risk Index (Lee index)** uses six factors — ischaemic heart disease, heart failure, cerebrovascular disease, insulin-treated diabetes, renal insufficiency (creatinine >2.0 mg/dL), and high-risk surgery.
9. **Assess exercise tolerance.** The classical bedside test is the ability to climb one flight of stairs. Poor exercise capacity predicts poor outcome.
10. Order tests only if the result will change management. **Pre-operative pulmonary function tests and arterial blood gases are rarely of prognostic value.** Non-invasive cardiac testing is reserved for intermediate- and high-risk patients.
11. **Coronary revascularisation (CABG or PCI) should be done only if it is indicated independently of the planned surgery** — not merely to 'get the patient through' the operation.

Medications and Prophylaxis

12. Plan the drug chart carefully — **some drugs are continued, some withheld, and some need dose adjustment**.
13. Three prophylactic measures have documented value: **beta-blockade in appropriately selected patients, antithrombotic (VTE) prophylaxis, and antimicrobial prophylaxis**.

Frailty, Cognition and Delirium

14. **Frailty independently predicts post-operative complications, longer hospital stay and discharge to a nursing facility** (Makary). It is assessed by weight loss, weak grip strength, exhaustion, low physical activity and slow walking speed.
15. **Pre-operative impaired cognition, recent falls, low albumin, greater anaemia, functional dependence and multiple comorbidities** predict higher mortality at 6 months (Robinson).
16. Four items independently predict **post-operative delirium** — prior stroke or TIA, low MMSE score, abnormal serum albumin, and a high Geriatric Depression Scale score (Rudolph). Preventing delirium is a core aim of perioperative care.
17. In cardiac surgery, frailty is scored by **Katz ADL ≤4 of 6, low grip strength, 5-metre walk ≥6 seconds, 6-minute walk <150 metres, timed get-up-and-go ≥15 seconds, and cognitive impairment**. Frailty gives a **two- to four-fold rise in operative mortality**.

Anaesthesia, Cardiac Surgery and Outcome

18. **Regional anaesthesia is not clearly superior to general anaesthesia** in older people. It may need added sedation, can cause significant cardiovascular changes, and does not clearly reduce DVT or blood loss. The choice must be individualised.

19. After cardiac surgery the most feared complication is **stroke**, usually embolic. Risk factors are cerebral atrophy, reduced cerebrovascular reserve, and atherosclerosis of the aorta or neck vessels. Screen with **carotid duplex**; severe ascending aortic atheroma raises neurological events to 10%.

20. **Confusion of uncertain cause or early dementia is a relative contraindication to cardiac surgery**, as open-heart surgery may worsen it. **Late referral independently worsens both operative risk and late outcome.**

21. In the elderly the aim of cardiac surgery is **relief of symptoms and improvement in quality of life**, not longevity. After cardiac surgery 84–94% of octogenarian survivors continue living independently and 83–98% would undergo the operation again.

22. A **comprehensive geriatric consultation** completes the assessment — medication review, functional assessment, environmental and caregiver assessment, and a **discussion of advance directives**.

Latest Updates (2023 – Present)

- **2024 AHA/ACC/Multisociety Perioperative Guideline (JACC 2024; Circulation 2024)** — the fourth major revision, superseding the 2014 guideline. It uses a **stepwise algorithm** and stresses a multidisciplinary, shared decision-making approach.
- **Timing definitions were redefined:** emergency = less than 2 hours (was 6 hours); urgent = 2–24 hours; time-sensitive = up to 3 months (was 1–6 weeks); elective = can be delayed indefinitely.
- **'Active cardiac conditions' are back in the algorithm** — acute coronary syndrome, decompensated heart failure and unstable arrhythmia. Elective surgery must be postponed until these are treated.
- A new step called **'risk modifiers'** was added: severe valvular disease, pulmonary hypertension, congenital heart disease, prior PCI/CABG, recent stroke, cardiac implantable electronic device, and **frailty**. Frailty is now formally part of the cardiac risk pathway.
- **Functional capacity should be measured, not estimated.** The METS study showed that clinician-estimated exercise capacity does not predict complications; the **Duke Activity Status Index (DASI)** does. DASI >34 indicates low risk. Basel-PMI and MET-REPAIR showed that **inability to climb two flights of stairs** predicts cardiac complications and death.
- **Cardiac biomarkers are a new step.** BNP or NT-proBNP (ACC/AHA preference) or troponin (ESC preference) may be measured in elevated-risk patients with poor exercise capacity. **BNP <92 ng/L or NT-proBNP <300 ng/L means low risk and no further testing.**
- **Post-operative troponin surveillance may now be considered in high-risk patients**, even without symptoms — a change from 2014 (myocardial injury after non-cardiac surgery, MINS).
- **Revascularisation before surgery is recommended only for acute coronary syndrome or left main stenosis >50%.** Coronary CT angiography is accepted as an alternative to pharmacological stress testing.
- **Medication guidance (2024):** continue beta-blockers if already on them but never start on the day of surgery (start ≥8 days before); continue statins; do not start clonidine; withhold ACE inhibitors/ARBs 24 hours before if taken for hypertension but continue for HFrEF; **withhold SGLT2 inhibitors 3–4 days before surgery** to prevent euglycaemic ketoacidosis.
- **Blood pressure:** consider delaying surgery if BP ≥180/110 mmHg; maintain intra-operative mean arterial pressure ≥60–65 mmHg and treat post-operative hypotension promptly.
- **GLP-1 receptor agonists:** the 2023 ASA advice to hold them was replaced by **2024 multisociety guidance (AGA, ASMBS, ASA, ISPCOP, SAGES)** — a risk-based, shared decision-making approach; most patients can continue, with a 24-hour clear-liquid diet and gastric point-of-care ultrasound if aspiration risk is a concern.
- **Prehabilitation** (structured exercise, nutrition and psychological preparation for 4–6 weeks before major elective surgery) reduces complications and length of stay in frail older adults; combined with **ERAS** protocols and the American College of Surgeons **Geriatric Surgery Verification** standards, which mandate routine screening for frailty, cognition, delirium risk, nutrition and goals of care.

- **Indian context:** around 10.5% of India's population is now 60 years or older (LASI/NSO projections). Pre-operative anaemia, undiagnosed diabetes and malnutrition are highly prevalent and correctable; surgical packages are covered under **PM-JAY**, and NP-NCD/NPHCE services support pre-operative optimisation at the primary level.

Current Practice Update

Textbook Position	Current Evidence / Guideline Position
Perioperative beta-blockade is recommended for high- and intermediate-risk patients with negative non-invasive tests.	Beta-blockers are **continued if already prescribed** but are **never started on the day of surgery**; if newly indicated, start at least 8 days before to allow titration (POISE trial showed excess stroke and death with day-of-surgery initiation).
Exercise tolerance is judged by the ability to climb one flight of stairs.	A **structured questionnaire (DASI >34)** or the ability to climb **two flights** is preferred; clinician-estimated METs do not predict outcome (METS study).
Risk is estimated clinically, with non-invasive cardiac testing for intermediate/high-risk patients.	Risk is calculated with a validated tool (RCRI, MICA, ACS-SRC, AUB-HAS2) and refined with **BNP/NT-proBNP or troponin**; stress testing only if the result will change management.
Emergency surgery is defined as surgery needed within 6 hours.	Emergency is now defined as **less than 2 hours**; urgent 2–24 hours; time-sensitive up to **3 months**.
Frailty is described as a predictor of complications in research studies.	Frailty is a **formal risk modifier** in the 2024 algorithm and a mandatory screening domain under ACS Geriatric Surgery Verification standards.
Textbook lists beta-blockade, antithrombotic and antimicrobial prophylaxis as the prophylactic measures.	The list now also includes **SGLT2 inhibitor and GLP-1 agonist management, delirium prevention bundles, prehabilitation and ERAS pathways**.
Post-operative troponin is checked only if there are signs or symptoms of ischaemia.	**Routine troponin surveillance may be considered in high-risk patients** to detect myocardial injury after non-cardiac surgery (MINS), which is usually silent.

Examiner's Tip — verbatim mark-earning lines

- "In the older surgical patient, **comorbidity and urgency, not chronological age, determine outcome**."
- "The four cardiac conditions that substantially raise risk are **myocardial infarction within 6 months, pulmonary oedema, unstable angina and severe aortic stenosis**."
- "**Frailty confers a two- to four-fold increase in operative mortality** and is now a formal risk modifier in the 2024 ACC/AHA perioperative guideline."
- "Pre-operative pulmonary function tests and arterial blood gases are **rarely of prognostic value** — order a test only if it will change management."
- "**Prophylactic revascularisation is indicated only when it would be indicated in the non-surgical setting** — ACS or left main >50%."
- "Quote the four delirium predictors of Rudolph: **prior stroke/TIA, low MMSE, abnormal albumin and high GDS score**."
- "Withhold **SGLT2 inhibitors 3–4 days** before surgery; withhold ACE inhibitors/ARBs 24 hours before if given for hypertension."
- "In the elderly, the goal of cardiac surgery is **symptom relief and quality of life, not longevity** — and late referral independently worsens outcome."

Q2. Checklist for Older Adults Undergoing Diagnostic and Therapeutic Procedures — Periprocedural Medical Issues (including Antiplatelet Management)

High-Yield Points

Framework of the Checklist

1. A periprocedural checklist for an older adult has five headings — **appropriateness, antithrombotic management, organ-system optimisation, geriatric syndromes, and post-procedure care**.
2. **Appropriateness first:** confirm the indication, the risk–benefit balance, life expectancy and lag time to benefit, capacity and informed consent, and advance directives. Ask whether the result will change management.

Antiplatelet and Anticoagulant Management

3. **Anticoagulants should be stopped before most invasive procedures**, but they may be continued for low-risk procedures where bleeding would be very minor or clinically insignificant.
4. **Heparin bridging is only for patients with high thromboembolic risk.** In a patient on a vitamin K antagonist, start LMWH or unfractionated heparin when the INR falls below 2, or after two to three doses have been omitted.
5. **Bridging is never needed for DOACs**, because their half-lives are short and the anticoagulant-free interval is brief.
6. **Restart anticoagulation about 24 hours after the procedure.** Delay to 48–72 hours only when the risk of post-operative bleeding is high.
7. **Mechanical prosthetic valves:** do not stop the VKA for minor surgical, superficial, dental or ophthalmic procedures — use local haemostasis and check that the INR on the day is not more than 0.5 above target. Major surgery needs an INR below 1.5.
8. For mechanical valves, stop warfarin **3–5 days** before surgery and bridge once the INR falls more than 0.5 below target. **Intravenous unfractionated heparin is preferred** because the effect of LMWH cannot be fully reversed. Restart oral anticoagulation the day after surgery.
9. Target INR for a mechanical prosthesis is **2.5–4.0** depending on prosthesis thrombogenicity and patient factors; **mitral or tricuspid position needs a higher target of 3.0**. **DOACs are contraindicated with mechanical valves.**
10. For a **surgical aortic bioprosthesis**, low-dose aspirin 75–100 mg daily for three months is now favoured over oral anticoagulation, provided the patient is in sinus rhythm.
11. **Duration of DAPT after PCI:** aspirin 75–100 mg plus clopidogrel 75 mg — one month after a bare-metal stent, at least six months after a drug-eluting stent, and 12 months after ACS irrespective of revascularisation strategy. Thereafter lifelong single antiplatelet therapy.
12. In older people **bleeding is commoner than recurrent thrombosis**, so a **short DAPT** course is appropriate — 6 months for stable IHD and 12 months for ACS, and as short as 1 and 6 months respectively with newer drug-eluting stents. **The evidence no longer supports bare-metal stents in the elderly.**
13. **Prasugrel is not recommended in patients aged 75 or above** — it was not superior to clopidogrel and increased major and fatal bleeding. **Ticagrelor** may be used; its bleeding risk is similar to clopidogrel.
14. **Add a proton pump inhibitor** to reduce gastrointestinal bleeding in older patients on DAPT, particularly with a previous GI bleed.
15. **Use the trans-radial approach** for PCI. Bleeding is the commonest non-cardiac complication of PCI and predicts death, MI, stroke, longer stay and higher cost in the elderly.
16. After **intracranial bleeding**, restarting anticoagulation is associated with fewer thromboembolic events and a similar risk of recurrent haemorrhage; the optimal timing is not established and the type of bleed matters (recurrence risk is higher after subarachnoid than intraparenchymal haemorrhage).

Organ-System and Drug Optimisation

17. **Renal protection:** estimate creatinine clearance, hydrate before contrast, withhold nephrotoxics and metformin, and dose-adjust all renally excreted drugs. Acute heparin dosing must be adjusted to renal function.
18. **Medication reconciliation and deprescribing:** stop or reduce anticholinergics, benzodiazepines and sedating antihistamines before the procedure; continue essential cardiac and antiepileptic drugs; plan insulin and oral hypoglycaemic adjustment; avoid prolonged fasting.
19. **Bowel preparation in elderly patients:** avoid sodium phosphate preparations (hyperphosphataemia, acute phosphate nephropathy) and avoid phosphate enemas in renal impairment; use PEG-based split-dose preparation with careful attention to volume status.

Geriatric Syndromes and Post-procedure Care

20. Screen for and address **frailty, cognitive impairment and delirium risk, falls, malnutrition, pressure-injury risk, sensory impairment (hearing aids, spectacles, dentures returned promptly), and continence**. Minimise sedation depth and duration.
21. Provide **VTE prophylaxis and antimicrobial prophylaxis** where indicated, ensure normothermia, and use short-acting anaesthetic and sedative agents.
22. After the procedure: **early mobilisation, early oral intake, removal of catheters and lines, multimodal opioid-sparing analgesia, delirium screening (4AT or CAM), and a clear plan for restarting antithrombotics**.

Latest Updates (2023 – Present)

- **2024 AHA/ACC Perioperative Guideline — antiplatelet recommendations: continue aspirin perioperatively where possible in all patients with stents**, regardless of stent type or timing. Continue DAPT if surgery is required within 30 days of a bare-metal stent or 3 months of a drug-eluting stent.
- **Elective non-cardiac surgery requiring interruption of antiplatelet therapy within 30 days of stent placement is classified as harmful** (risk of stent thrombosis).
- **Stop times before surgery (2024):** prasugrel 7 days, clopidogrel 5 days, ticagrelor 3 days, aspirin 4–5 days. **Ticagrelor is the only reversible platelet inhibitor** of this group.
- **Timing of surgery after PCI:** at least 14 days after balloon angioplasty; at least **12 months after a drug-eluting stent placed for ACS** or complex PCI; at least **6 months after a drug-eluting stent for chronic coronary disease**; time-sensitive surgery may proceed at **3 months** (ESC allows 1 month).
- **Aspirin for primary prevention should be stopped before surgery** (except carotid surgery), and should not be newly started pre-operatively. Aspirin for secondary prevention may reasonably be continued if the cardiac risk outweighs bleeding risk (based on POISE-2).
- If a patient after PCI is on **oral anticoagulant monotherapy** that must be stopped, **substitute aspirin** until the anticoagulant can be resumed.
- **Bridging:** the BRIDGE trial and the **PAUSE** study established that **routine bridging is not indicated** — a standardised, interval-based DOAC interruption without bridging or routine coagulation testing gives low rates of both thromboembolism and major bleeding. Bridging is reserved for high thrombotic risk on a vitamin K antagonist.
- **Endoscopy-specific guidance (ASGE/ESGE/BSG):** for **low-risk endoscopic procedures** (diagnostic endoscopy ± biopsy, ERCP with stent placement without sphincterotomy) antiplatelets and anticoagulants are **continued**. For high-risk procedures (polypectomy, EMR/ESD, sphincterotomy, PEG insertion, EUS-FNA) they are interrupted; aspirin is usually continued throughout.
- **GLP-1 receptor agonists:** the 2024 multisociety guidance (AGA/ASMBS/ASA/ISPCOP/SAGES) replaced blanket withholding with a **risk-stratified approach** — continue in most patients, use a 24-hour clear-liquid diet, and consider gastric point-of-care ultrasound or rapid-sequence induction if there are GI symptoms or high aspiration risk.
- **SGLT2 inhibitors** should be withheld **3–4 days** before an elective procedure (4 days for ertugliflozin) to prevent euglycaemic ketoacidosis.
- **Left atrial appendage occlusion** is an accepted alternative for patients with non-valvular AF who cannot tolerate long-term anticoagulation, and increasingly allows procedures without antithrombotic interruption.

- **Indian practice point:** generic clopidogrel and ticagrelor are widely used; warfarin remains common because of cost, so **INR-based planning, patient literacy and reliable follow-up** must be built into the checklist. Andexanet alfa and idarucizumab availability is limited outside large centres, which favours interval-based DOAC interruption over reliance on reversal agents.

Current Practice Update

Textbook Position	Current Evidence / Guideline Position
Anticoagulation should be stopped before the vast majority of invasive procedures.	Many **low-risk procedures (including diagnostic endoscopy with biopsy, cataract surgery, dental extraction, pacemaker implantation)** are now performed **without interrupting** anticoagulation, using local haemostasis.
Bridging with heparin is used for high thromboembolic risk.	**PAUSE and BRIDGE** confirmed that **routine bridging is unnecessary and increases bleeding**; it is confined to mechanical valves and very high-risk VKA patients.
DAPT is 1 month after a bare-metal stent and at least 6 months after a drug-eluting stent.	**Bare-metal stents are obsolete.** With current drug-eluting stents, DAPT of **1–3 months followed by single antiplatelet monotherapy** is safe and reduces bleeding in older people (MASTER-DAPT, TWILIGHT).
The textbook does not give specific pre-operative stop intervals for oral antiplatelets.	**Prasugrel 7 days, clopidogrel 5 days, ticagrelor 3 days, aspirin 4–5 days** (2024 ACC/AHA).
Aspirin is broadly continued for patients on antiplatelet therapy.	Aspirin taken for **primary prevention should be stopped**; aspirin for **secondary prevention and in all stented patients is continued** wherever bleeding risk allows.
No guidance is given on newer antidiabetic agents.	**SGLT2 inhibitors are withheld 3–4 days; GLP-1 receptor agonists** are managed by a risk-based multisociety pathway rather than routine withholding.

Examiner's Tip — verbatim mark-earning lines

- "**Bridging is not needed with DOACs** — their short half-life makes the anticoagulant-free interval brief."
- "For a mechanical prosthetic valve, use **intravenous unfractionated heparin** for bridging, because the effect of LMWH cannot be fully reversed."
- "**Prasugrel should not be used in patients aged 75 or above** — no ischaemic benefit and more major and fatal bleeding."
- "**Elective surgery needing antiplatelet interruption within 30 days of stenting is harmful.**"
- "Memorise the stop intervals: **prasugrel 7, clopidogrel 5, aspirin 4–5, ticagrelor 3 days**."
- "In the elderly undergoing PCI, **bleeding is commoner than recurrent thrombosis** — hence radial access, short DAPT, a PPI and a drug-eluting stent."
- "Avoid **sodium phosphate bowel preparations and phosphate enemas** in older patients — they cause hyperphosphataemia and acute phosphate nephropathy."
- "Every periprocedural checklist in geriatrics must include **capacity, advance directives, delirium risk and a plan for restarting antithrombotics**."

Q3. Constipation in the Elderly — Prevention and Management

High-Yield Points

Definition and Epidemiology

1. Constipation is defined by **difficult, infrequent or incomplete defecation**. Rome IV criteria require two or more of straining, hard or lumpy stools, sensation of incomplete evacuation, sensation of anorectal blockage, manual manoeuvres, and fewer than three spontaneous bowel movements a week, for at least three months.
2. **Complaints of constipation and laxative use are very common in older people.** Twenty to thirty per cent of community-dwelling elders use laxatives at least weekly, and up to 50% of elderly women use them.
3. **When comorbidities are controlled for, constipation is no more common in elders than in younger people. Stool frequency does not change with age** — what elders complain of is **straining and hard stools**.

Physiology and Aetiology

4. **Colonic transit is generally well preserved with ageing.** The change is in the pelvic floor — older women show reduced opening of the anorectal angle and greater perineal descent, and **pudendal neuropathy** becomes commoner.
5. Most causes relate to **medication use or coexisting medical illness**. The commonest drugs are **opiates, calcium channel blockers (verapamil is the worst), NSAIDs and drugs with anticholinergic effects**.
6. Diseases that contribute include **diabetes mellitus, Parkinson's disease, stroke, hypothyroidism, hypercalcaemia, depression and dementia**. Hysterectomy in women over 50 prolongs colonic transit.
7. **Immobility and reduced fluid and fibre intake are traditionally blamed but the evidence is weak**, and increased physical activity does not reliably improve constipation.
8. Changes can be mapped anatomically — **central nervous system** (awareness of the need to defecate: stroke, dementia, Parkinson's), **peripheral nerves** (pudendal injury), **enteric nervous system** (rectal desensitisation, diabetes), and **skeletal muscle/incoordination** (birth trauma, sphincterotomy, scleroderma).

Consequences

9. Constipation **reduces quality of life and self-perceived health**, increases urinary frequency, urgency and dysuria, and worsens mood — all of which improve with treatment.
10. **Faecal impaction is common — about 7% of institutionalised elderly have impacted faeces on rectal examination.** The first sign may be **overflow diarrhoea and rectal incontinence**; if a rectal examination is not done, this is misdiagnosed and wrongly treated with antidiarrhoeals.
11. Immobile or cognitively impaired patients risk **faecal impaction, stercoral ulceration and colonic perforation**.

Evaluation

12. History, drug review and a **digital rectal examination are mandatory**. In patients whose colorectal cancer screening is up to date and who have no worrisome symptoms such as bleeding or weight loss, **an empirical therapeutic trial is appropriate**; investigate only those who fail to respond.
13. For non-responders: colonic transit study, anorectal manometry and balloon expulsion test, defecography, and a **plain abdominal radiograph to assess stool burden**.

Stepwise Management

14. **Step 1 — review and stop constipating medications** and substitute a less constipating alternative where one exists; then increase dietary fibre and fluid intake. Use the **safest, best-tolerated and least expensive laxatives first**.
15. **Step 2 — bulking agents:** bran 4–10 g, psyllium 3–6 g, methylcellulose 2–4 g, calcium polycarbophil 2–4 g, hydrolysed guar gum 3–6 g. **Avoid bulking agents in faecal impaction, in bedbound patients and where fluid intake is poor.** Side effects are bloating and flatulence.

16. **Stool softeners such as docusate have no randomised evidence of efficacy** — one trial showed no improvement over placebo and another found psyllium superior. Their therapeutic role is limited.
17. **Step 3 — osmotic laxatives: polyethylene glycol (PEG) 17–34 g is the best tolerated in elders** and improves both stool consistency and frequency. Lactulose 10–40 g and sorbitol 70% 15–60 mL are alternatives but cause bloating and cramps from bacterial fermentation. **Magnesium and saline salts should generally be avoided** — hypermagnesaemia occurs even with normal renal function; avoid in renal disease, end-stage liver disease and heart failure.
18. **Step 4 — stimulant laxatives:** senna 15 mg, bisacodyl 5–10 mg, sodium picosulfate 5–10 mg. In practice the commonest effective combination is an **osmotic plus a stimulant** laxative.
19. **Secretagogue:** lubiprostone 8–24 µg twice daily, a chloride channel activator, has good evidence in older people.

Special Situations

20. **Opioid-induced constipation: peripherally acting mu-opioid receptor antagonists (PAMORAs)** — methylnaltrexone, naloxegol and naldemedine — reverse constipation **without reducing analgesia or precipitating withdrawal**, and are superior to placebo. Methylnaltrexone is given by injection and is reserved for refractory symptoms. PEG and stimulant laxatives are also used.
21. **Faecal impaction:** give PEG or another osmotic laxative with or without enemas; a hard or large rectal bolus needs **manual removal**; water-soluble hyperosmotic contrast enemas may also relieve it. **Bulking agents must be avoided.**
22. **Enemas:** limit to acute situations and institutional care. **Phosphate enemas cause serious hyperphosphataemia, particularly in renal impairment; soap-sud enemas should never be used.** Small-volume tap water enemas are useful; large-volume ones can cause hyponatraemia; arachis oil retention enemas lubricate and soften impacted faeces (check nut allergy). Any enema carries a risk of colonic perforation.
23. **Dyssynergic defecation: biofeedback is the established mainstay**, given over four to six sessions, and improves defecatory function in about 70%. It is superior to diet, exercise, laxatives or diazepam. If both slow transit and dyssynergia coexist, **treat with biofeedback first**.
24. **Refractory slow-transit constipation:** nearly 90% respond to laxatives. Subtotal colectomy with ileorectal anastomosis is rarely needed and works best in patients without upper gut dysmotility or major psychological symptoms. Antegrade enemas through a caecostomy are an alternative.
25. **Dementia: prevention is the preferred strategy** because the patient may not recognise or communicate symptoms. Review drugs, increase fibre, fluid and activity, and use a **regular osmotic laxative** rather than rectal preparations. Non-verbal signs of the need to defecate include **fidgeting, pacing and pulling at clothes**; educate relatives and carers.
26. **Stroke:** constipation is associated with dependency, bedpan use for defecation, high NIHSS score, polypharmacy, dehydration and inactivity, and predicts a poor outcome at 12 weeks. **Abdominal massage and visceral mobilisation** improve frequency and reduce pain and incomplete evacuation.
27. **Other agents:** large randomised trials of **probiotics have failed to show benefit**. Misoprostol and colchicine are reserved for refractory cases and are limited by cramping. **Neostigmine is reserved for acute colonic pseudo-obstruction** in hospitalised patients.
28. **Prevention:** maintain mobility, use the **gastrocolic reflex** by toileting 20–30 minutes after meals, ensure privacy and an appropriate toilet position, provide a commode rather than a bedpan, keep hydration and fibre adequate, correct hypothyroidism and hypercalcaemia, and review the drug chart at every visit.

Latest Updates (2023 – Present)

- **AGA–ACG Joint Clinical Practice Guideline on Chronic Idiopathic Constipation (Gastroenterology 2023;164:1086–1106; Am J Gastroenterol 2023;118:936)** — ten GRADE-based recommendations, the first joint guideline of its kind.
- **Strong recommendations: polyethylene glycol** (the only over-the-counter agent strongly recommended for chronic use), **bisacodyl and sodium picosulfate** (short-term, under 4 weeks, or as rescue therapy), and the prescription agents **linaclotide, plecanatide and prucalopride** after over-the-counter agents fail.

- **Conditional recommendations:** fibre supplements (especially psyllium), **magnesium oxide**, lactulose, **senna** and lubiprostone. This was the first guideline to endorse magnesium oxide and senna as evidence-based options.
- **Docusate is not recommended** — the guideline found no evidence of benefit, confirming long-standing geriatric teaching.
- **Prucalopride**, a highly selective 5-HT4 agonist, has a reassuring cardiovascular safety profile (unlike cisapride and tegaserod) and is therefore acceptable in older adults; the guideline notes no signal of excess cardiovascular events.
- **Evaluate for a defecatory disorder before escalating drugs** — oral agents work poorly in dyssynergic defecation; refer for **anorectal manometry, balloon expulsion and pelvic floor biofeedback**.
- **Newer options:** the **vibrating capsule** (FDA-cleared, drug-free, uses vibration to stimulate colonic motility) is attractive in polypharmacy; **elobixibat**, an ileal bile acid transporter inhibitor, is available in some Asian markets; **tenapanor** (NHE3 inhibitor) is approved for IBS-C.
- **Opioid-induced constipation:** network meta-analyses continue to support PAMORAs over placebo; **oral naldemedine and naloxegol** have largely replaced injectable methylnaltrexone for routine use, and are useful in palliative and cancer care in the elderly.
- **Constipation as a prodrome:** accumulating cohort data confirm constipation may precede motor Parkinson's disease by 10–20 years and is associated with cognitive decline — it should prompt a neurological review rather than being dismissed.
- **Indian context:** the Indian Society of Gastroenterology consensus and routine Indian practice place **lactulose, PEG and ispaghula (isabgol) husk** as the mainstays, chosen for cost and availability; linaclotide, prucalopride and lubiprostone are marketed in India but cost is a limiting factor. Rising urban prevalence is linked to low dietary fibre in refined-cereal diets, reduced physical activity and polypharmacy.

Current Practice Update

Textbook Position	Current Evidence / Guideline Position
A stepwise approach — bulking agent first, then osmotic, then stimulant.	**PEG is now first-line** and the only OTC agent with a strong recommendation for chronic use; **fibre has only a conditional recommendation** and is often poorly tolerated in frail elders.
Stool softeners have limited evidence; docusate is of doubtful value.	**Docusate is explicitly not recommended** and should be deprescribed (2023 AGA/ACG).
Magnesium salts are generally avoided in elders.	**Magnesium oxide is conditionally recommended** as an effective, inexpensive option — but the geriatric caution stands: **avoid in eGFR <30 mL/min**, and monitor for hypermagnesaemia.
Lubiprostone is listed among the agents with the best evidence in elders.	Lubiprostone has been **downgraded to a conditional recommendation** (low certainty); **linaclotide, plecanatide and prucalopride carry strong recommendations**.
Methylnaltrexone (injection) is the important advance for opioid-induced constipation.	**Oral PAMORAs — naldemedine and naloxegol — are now preferred** for routine use; injectable methylnaltrexone is reserved for refractory or terminal care settings.
Stimulant laxatives are the third step in chronic use.	Bisacodyl and sodium picosulfate carry a **strong recommendation but for short-term (<4 weeks) or rescue use**; long-term daily use is not endorsed.

Examiner's Tip — verbatim mark-earning lines

- "**Stool frequency does not change with normal ageing** — elders complain of **straining and hard stools**, not of infrequency."
- "When comorbidity is controlled for, **healthy elders are no more likely to develop constipation than younger people**."
- "**Always do a rectal examination** — overflow diarrhoea from faecal impaction is otherwise misdiagnosed and treated with antidiarrhoeals, which is dangerous."
- "**Avoid bulking agents in faecal impaction, in bedbound patients and where fluid intake is poor.**"
- "**Docusate is ineffective and should not be prescribed.**"
- "**PEG is the best-tolerated osmotic laxative in the elderly**; magnesium salts should be avoided in renal disease and saline salts in heart failure and end-stage liver disease."
- "**PAMORAs reverse opioid-induced constipation without affecting analgesia or precipitating withdrawal.**"
- "**Biofeedback is the mainstay for dyssynergic defecation** and works in about 70% — treat dyssynergia before considering colectomy for slow transit."
- "**Never use soap-sud enemas; avoid phosphate enemas in renal impairment.**"
- "In dementia, **prevention is the strategy** — non-verbal cues that a patient needs to defecate are **fidgeting, pacing and pulling at clothes**."

Q4. Recent Trends in the Treatment of Hepatitis C Infection

*The uploaded textbook covers hepatitis C only briefly — it states that chronic HCV is prevalent in the elderly, that up to 85% of patients are asymptomatic with at least half having normal liver function tests, that screening should be offered, and that **newer therapies can halt disease progression and cure most patients**. It does not detail individual regimens. The regimen-level content below is therefore drawn from AASLD/IDSA, EASL, WHO and Indian national guidance, as permitted.*

High-Yield Points

Background and Goal of Therapy

1. HCV is a blood-borne RNA virus with **six major genotypes**. In India **genotype 3 predominates**, followed by genotype 1; genotype 4 is seen in some regions.
2. **Acute hepatitis C is uncommon but chronic infection is prevalent in the elderly.** Up to 85% are asymptomatic and at least half have normal transaminases, so it is missed unless actively sought.
3. The end point of treatment is **sustained virological response at 12 weeks (SVR12)**, which equals cure. Cure reduces all-cause mortality, hepatic decompensation, hepatocellular carcinoma and the need for transplantation.
4. Fibrosis is now staged **non-invasively** — **FIB-4, APRI and transient elastography (FibroScan)** have largely replaced liver biopsy.

The Older Modalities (of historical and viva importance)

5. **Interferon-alfa, then pegylated interferon plus ribavirin** gave SVR of only about 40–55% in genotype 1 and needed 24–48 weeks. It was **particularly poorly tolerated in the elderly** — flu-like symptoms, cytopenias, depression, thyroid and autoimmune disease, and ribavirin-induced haemolytic anaemia.
6. The **first-generation protease inhibitors boceprevir and telaprevir** (2011) added efficacy but also anaemia, rash and a heavy pill burden. Both interferon-based therapy and first-generation protease inhibitors are now **obsolete**.

Direct-Acting Antivirals — the Current Standard

7. Three DAA classes are combined: **NS3/4A protease inhibitors ('-previr')**, **NS5A inhibitors ('-asvir')**, and **NS5B polymerase inhibitors ('-buvir')**, of which sofosbuvir is the nucleotide backbone.
8. **Pan-genotypic regimens are now standard, so routine genotyping is no longer required:**
9. **Sofosbuvir 400 mg + velpatasvir 100 mg**, one tablet daily for **12 weeks** — the most widely used pan-genotypic regimen.
10. **Glecaprevir 300 mg + pibrentasvir 120 mg** daily for **8 weeks** in treatment-naive patients without cirrhosis or with compensated cirrhosis — the shortest course, and safe in **severe renal impairment and dialysis**.
11. **Sofosbuvir 400 mg + daclatasvir 60 mg** for 12 weeks — WHO-recommended, generic, inexpensive, and the workhorse regimen of India's national programme.
12. **Simplified treatment algorithms** allow treatment of all patients aged 12 and above with minimal baseline testing and minimal on-treatment monitoring; the SVR12 rate exceeds **95%** across genotypes.

Special Groups

13. **Compensated cirrhosis (Child–Pugh A):** the same pan-genotypic regimens are used, usually for 12 weeks.
14. **Decompensated cirrhosis (Child–Pugh B or C): protease inhibitors are contraindicated** (glecaprevir, voxilaprevir) because of hepatotoxicity. Use **sofosbuvir/velpatasvir with weight-based ribavirin for 12 weeks**, or 24 weeks without ribavirin if ribavirin is not tolerated.
15. **Retreatment after DAA failure: sofosbuvir/velpatasvir/voxilaprevir for 12 weeks** is the salvage regimen, with SVR of 96–98%.
16. **Renal disease:** glecaprevir/pibrentasvir requires no dose adjustment at any level of renal function including dialysis; sofosbuvir-based regimens are now also considered safe at eGFR below 30.

Issues Specific to Older Patients

17. **DAAs are as effective and as well tolerated in older patients as in the young** — age is not a reason to withhold treatment. The dominant issue is **drug–drug interaction**, not toxicity.
18. **Key interactions: amiodarone with sofosbuvir causes severe symptomatic bradycardia and is contraindicated**; proton pump inhibitors and antacids reduce absorption of velpatasvir and ledipasvir; statins (especially rosuvastatin and atorvastatin) need dose reduction; **rifampicin, carbamazepine, phenytoin, phenobarbitone and St John's wort are contraindicated** as enzyme inducers.
19. **Screen for HBsAg before starting a DAA** — **HBV reactivation is a black-box warning** and can be fulminant. Give prophylactic antiviral cover or monitor closely.
20. **After cure, hepatocellular carcinoma surveillance must continue lifelong** with six-monthly ultrasound in patients with cirrhosis or advanced (F3) fibrosis, because SVR reduces but does not abolish HCC risk. Varices screening also continues in cirrhotics.

Screening, Prevention and Programmes

21. The textbook cites the **CDC birth-cohort recommendation (one-time anti-HCV testing for all adults born 1945–1965)**. This has since been widened to **one-time testing of all adults at least once in a lifetime**, plus periodic testing for those at ongoing risk.
22. **There is no vaccine for hepatitis C**, and cure does not confer immunity — **reinfection is possible**. Prevention rests on injection and blood safety, single-use needles, sterile dental and salon practice, and harm-reduction services.
23. **India's National Viral Hepatitis Control Programme (NVHCP)**, launched in 2018 under the National Health Mission, provides **free diagnosis and free treatment** with generic pan-genotypic DAAs, targeting elimination as a public health threat by 2030.

Latest Updates (2023 – Present)

- **AASLD–IDSA HCV Guidance (2023 update, and subsequent revisions):** a **simplified treatment algorithm** for treatment-naive adults without cirrhosis or with compensated cirrhosis — no genotyping, minimal pre-treatment work-up and reduced monitoring.
- **Shortened salvage therapy:** based on POLARIS-3, **8 weeks of sofosbuvir/velpatasvir/voxilaprevir** gave the same SVR12 (96%) as 12 weeks of sofosbuvir/velpatasvir in treatment-naive genotype 3 patients with compensated cirrhosis — useful where resistance-associated substitution testing is unavailable.
- **Lancet 2025 factorial randomised non-inferiority trial: sofosbuvir–daclatasvir was non-inferior to sofosbuvir–velpatasvir**. This is a major finding for low- and middle-income countries, as it validates the cheapest generic first-line combination.
- **Shortening therapy:** the Indian **RESOLVE** multicentre randomised trial (ILBS-led) compared **8 versus 12 weeks of sofosbuvir/velpatasvir in treatment-naive non-cirrhotic adults**; shorter courses reduce cost and improve completion rates in programme settings.
- **WHO Global Hepatitis Report 2024:** viral hepatitis deaths are rising, and although curative therapy exists, **diagnosis and treatment coverage remain low** — only a minority of infected people worldwide have been diagnosed or treated. Hepatitis is now the second leading infectious cause of death globally.
- **Point-of-care HCV RNA testing** (GeneXpert, molecular finger-stick platforms) enables **same-visit 'test-and-treat'**, removing the loss to follow-up that has been the main programme bottleneck.
- **Reflex RNA testing** from the same anti-HCV-positive sample is now recommended, replacing the two-visit model.
- **HCV-viraemic donor organs** (kidney, liver, heart) are now transplanted into HCV-negative recipients with short-course DAA prophylaxis, expanding the donor pool — directly relevant to older transplant candidates.
- **Elderly-specific data:** real-world cohorts and meta-analyses confirm **SVR rates above 95% in patients over 65 and over 75**, with no increase in serious adverse events; the barrier is drug–drug interaction and under-referral, not efficacy.
- **Cure improves extrahepatic outcomes** — cohort data show reduced cardiovascular events, improved glycaemic control in type 2 diabetes, improved cognition and quality of life, and resolution of cryoglobulinaemic vasculitis and HCV-related lymphoproliferative disease.

- **Indian data:** HCV prevalence is estimated at roughly 0.3–0.5% nationally with regional clustering (Punjab, north-east, Andhra Pradesh). Punjab's **Mukh Mantri Punjab Hepatitis-C Relief Fund** demonstrated large-scale public-sector cure and became a model for NVHCP scale-up.

Current Practice Update

Textbook Position	Current Evidence / Guideline Position
Newer therapies can halt progression and cure most patients with hepatitis C.	Cure is now the routine expectation — **SVR12 exceeds 95%** with 8–12 week all-oral, once-daily, interferon-free pan-genotypic regimens.
CDC recommends one-time anti-HCV testing for adults born between 1945 and 1965.	The birth-cohort strategy has been replaced by **universal one-time adult screening**, with reflex RNA testing on the same sample and repeat testing for ongoing-risk groups.
Genotype-specific approaches and treatment guided by specialist referral.	**Genotyping is no longer required.** Simplified, decentralised, pan-genotypic treatment can be delivered by non-specialists, including at primary care level.
The textbook does not specify regimens.	First line: **sofosbuvir/velpatasvir 12 weeks, glecaprevir/pibrentasvir 8 weeks, or sofosbuvir/daclatasvir 12 weeks**; salvage: **sofosbuvir/velpatasvir/voxilaprevir 12 weeks**.
No specific caution is given regarding pre-treatment testing.	**HBsAg must be checked before starting a DAA** — HBV reactivation carries a black-box warning; and **amiodarone with sofosbuvir is contraindicated** (severe bradycardia).
Screening for hepatitis C should be done in most patients; treatment cures the disease.	After cure, **six-monthly HCC surveillance continues lifelong** in those with cirrhosis or F3 fibrosis, and reinfection remains possible as there is no vaccine.

Examiner's Tip — verbatim mark-earning lines

- "**SVR12 equals cure** — and cure reduces all-cause mortality, decompensation and hepatocellular carcinoma, but does **not** abolish HCC risk in the cirrhotic."
- "Name the three pan-genotypic first-line regimens: **sofosbuvir/velpatasvir 12 weeks, glecaprevir/pibrentasvir 8 weeks, sofosbuvir/daclatasvir 12 weeks**."
- "**Protease inhibitors are contraindicated in decompensated cirrhosis** — use sofosbuvir/velpatasvir with ribavirin."
- "**Sofosbuvir/velpatasvir/voxilaprevir is the salvage regimen** after DAA failure — SVR 96–98%."
- "**Glecaprevir/pibrentasvir needs no dose adjustment even on dialysis** — the regimen of choice in advanced CKD."
- "Two examination-favourite interactions: **amiodarone plus sofosbuvir causes severe bradycardia**, and **PPIs reduce velpatasvir absorption**."
- "**Always check HBsAg before a DAA** — HBV reactivation is a black-box warning."
- "India's **National Viral Hepatitis Control Programme (2018)** provides free diagnosis and generic pan-genotypic DAA treatment, targeting elimination by 2030."
- "**There is no hepatitis C vaccine** — cure does not prevent reinfection."

Q5. Treatment of Chronic Hepatitis B Infection — Older and Newer Modalities

The uploaded textbook covers hepatitis B briefly — it notes that around 257 million people worldwide are HBsAg positive, lists the high-risk groups to be screened, states that ***the screening tests are surface antigen and antibody****, that* ***HBsAg-positive patients need six-monthly ultrasound for hepatocellular carcinoma (which can occur even without cirrhosis)****, and that* ***patients with cirrhosis or persistently raised transaminases with detectable HBV DNA should be treated with oral once-daily antivirals such as tenofovir, which prevent progression of and reverse fibrosis****. Detail on individual regimens and newer agents is supplemented from WHO, AASLD, EASL and APASL guidance.*

High-Yield Points

Virology and Natural History

1. HBV is a partially double-stranded DNA hepadnavirus. It persists as **covalently closed circular DNA (cccDNA)** in the hepatocyte nucleus and integrates into host DNA — this is **why complete cure is not achievable** with current drugs.
2. The infection passes through phases: **HBeAg-positive chronic infection (immune tolerant), HBeAg-positive chronic hepatitis, HBeAg-negative chronic infection (inactive carrier), HBeAg-negative chronic hepatitis, and the HBsAg-negative (occult) phase**. Phase determines whether to treat or to monitor.
3. **Elderly patients are usually in the HBeAg-negative phase**, with fluctuating ALT and HBV DNA, longer duration of infection, and therefore a higher cumulative risk of cirrhosis and hepatocellular carcinoma.

Goals and End Points

4. **The realistic goal is durable suppression of HBV DNA**, which prevents progression, reverses fibrosis, reduces decompensation and lowers HCC risk. Cirrhosis regression on long-term therapy is well documented.
5. End points are **virological (undetectable HBV DNA), biochemical (normal ALT), serological (HBeAg seroconversion; and the ideal, HBsAg loss with or without anti-HBs)**, and histological.
6. **'Functional cure' means sustained HBsAg loss with undetectable HBV DNA off treatment.** It is achieved in only about 1% per year with current nucleos(t)ide analogues, and is the target of all new drug development.

Older Modalities

7. **Conventional interferon-alfa** and then **pegylated interferon alfa-2a 180 µg subcutaneously once weekly for 48 weeks** — the advantages are a **finite duration, no resistance, and the highest rate of HBsAg loss**.
8. **Interferon is largely unsuitable for older patients** — flu-like illness, cytopenias, depression, thyroid dysfunction and autoimmune flares. It is **absolutely contraindicated in decompensated cirrhosis and autoimmune disease**.
9. **Lamivudine 100 mg daily** — the first oral nucleoside analogue; low genetic barrier, with resistance in about 70% by five years (YMDD mutation). **No longer first-line.**
10. **Adefovir dipivoxil** — weak antiviral effect and dose-dependent nephrotoxicity; **telbivudine** — potent but high resistance and myopathy/neuropathy. Both are obsolete.

Current First-Line Oral Therapy

11. Three high-barrier agents are first-line: **tenofovir disoproxil fumarate (TDF) 300 mg daily, tenofovir alafenamide (TAF) 25 mg daily, and entecavir 0.5 mg daily** (1 mg if lamivudine-experienced). All are once-daily, oral, and produce virtually no resistance over a decade.
12. **Choice in the elderly is decided by renal and bone safety.** TDF causes proximal tubular dysfunction, hypophosphataemia and loss of bone mineral density — a real hazard in patients already at risk of falls and fracture. **Prefer TAF or entecavir if eGFR is below 60, if there is osteoporosis, or if other nephrotoxic drugs are being used.** Entecavir itself needs dose reduction in renal impairment.

13. **Duration is usually indefinite.** In HBeAg-positive patients, treatment may be stopped after seroconversion plus at least 12 months of consolidation; in HBeAg-negative patients and in all cirrhotics, treatment is **lifelong** unless HBsAg is lost.
14. **Decompensated cirrhosis:** treat with **entecavir or tenofovir immediately and lifelong; never use interferon**. Refer simultaneously for transplant assessment.

Indications for Treatment and Monitoring

15. Classical indications: **any cirrhosis with detectable HBV DNA; HBV DNA above 2000 IU/mL with ALT above the upper limit of normal; significant fibrosis or inflammation; extrahepatic manifestations; and a family history of cirrhosis or HCC**.
16. **Antiviral prophylaxis is mandatory before immunosuppression.** All HBsAg-positive patients receiving chemotherapy, **rituximab**, high-dose steroids, TNF inhibitors or transplantation need prophylactic tenofovir or entecavir; anti-HBc-positive, HBsAg-negative patients need monitoring or prophylaxis depending on the regimen. This is a common and lethal omission in older oncology patients.
17. **Hepatocellular carcinoma surveillance every six months with ultrasound (with or without alpha-fetoprotein) is required in HBsAg-positive patients even without cirrhosis** — HBV causes HCC more often than other liver diseases in the absence of cirrhosis. Continue surveillance even after HBV DNA becomes undetectable.
18. Monitoring on therapy: **HBV DNA and ALT every 3–6 months, creatinine and serum phosphate on TDF, and quantitative HBsAg** where available; assess adherence at every visit.

Coinfection and Prevention

19. **Hepatitis D** is the most severe form of viral hepatitis; test every HBsAg-positive patient for anti-HDV. **Bulevirtide**, an entry inhibitor blocking the NTCP receptor, is the first specific approved therapy for chronic hepatitis D.
20. **HIV coinfection:** use an antiretroviral regimen containing tenofovir plus lamivudine or emtricitabine, so that both viruses are treated. Never treat HBV alone with lamivudine in a coinfected patient.
21. **Prevention:** hepatitis B vaccine with a **timely birth dose** (part of India's Universal Immunisation Programme), plus antiviral prophylaxis in highly viraemic pregnant women, is the foundation of elimination. Older adults who are non-immune and at risk should be vaccinated; response rates are lower with age, so post-vaccination anti-HBs testing and double-dose or adjuvanted schedules may be needed.

Newer Modalities in Development

22. **Antisense oligonucleotide — bepirovirsen.** The phase 2b B-Clear study showed sustained HBsAg and HBV DNA loss in roughly 9–10% of patients; phase 3 programmes are ongoing.
23. **Small interfering RNA (siRNA) — xalnesiran, imdusiran, elebsiran.** Xalnesiran with or without an immunomodulator produced HBsAg loss in a subset of patients (NEJM 2024). These silence viral transcripts and reduce HBsAg.
24. **Capsid assembly modulators** (core protein allosteric modulators) block nucleocapsid formation and cccDNA replenishment; **entry inhibitors** (bulevirtide) block NTCP; **nucleic acid polymers** (REP 2139) block subviral particle release.
25. **Immunotherapeutic approaches** — therapeutic vaccines, TLR7/TLR8 agonists, PD-1 checkpoint inhibitors and engineered T cells — aim to restore the exhausted HBV-specific T-cell response. Most current strategies use **combination therapy** on a nucleos(t)ide analogue backbone.

Latest Updates (2023 – Present)

- **WHO Guidelines on the prevention, diagnosis, care and treatment of chronic hepatitis B (released March 2024 at APASL, Kyoto)** — the single most important recent change. They **substantially simplify and expand eligibility for treatment**.
- **Expanded eligibility (WHO 2024):** treat **all adults and adolescents with cirrhosis or significant fibrosis (APRI >0.5 or transient elastography >7 kPa)**; treat where **HBV DNA >2000 IU/mL with ALT above the upper limit of normal**; and treat those with coinfection (HIV, HCV, HDV), a family history of cirrhosis or HCC, extrahepatic manifestations, or immunosuppression.
- **Simplified criteria for resource-limited settings:** where HBV DNA testing is unavailable, treatment may be started on **APRI alone, or on persistently abnormal ALT alone** — designed to overcome the

fact that **only about 3% of people with chronic hepatitis B globally were on treatment in 2022**.

- **Comparative analyses (JAMA Network Open 2024)** confirm the WHO 2024 criteria make a markedly higher proportion of treatment-naive patients eligible than AASLD 2018, EASL 2017 or APASL 2015 — the practical consequence is that many patients previously 'monitored' now qualify for antivirals.
- **Expanded PMTCT:** antiviral prophylaxis for pregnant women is broadened, with simpler criteria to determine eligibility, alongside universal timely birth-dose vaccination.
- **Point-of-care HBV DNA (viral load) testing and reflex testing** are recommended to decentralise care; **routine HDV testing** of all HBsAg-positive people is now advised.
- **Journal of Hepatology 2024** commentary on the WHO update sets out the implications for the next AASLD/EASL/APASL revisions — the direction of travel internationally is towards **'treat more, treat earlier, treat simply'**.
- **Treat-all debate:** multiple expert panels (including the broad Chinese Society of Hepatology 2022 criteria, which use any detectable HBV DNA) argue for further expansion, on the basis that even 'immune-tolerant' patients accumulate HBV integration and HCC risk.
- **TAF is increasingly preferred over TDF in older adults** because of superior renal and bone safety, though with a small unfavourable lipid effect; entecavir remains an excellent, inexpensive alternative in nucleos(t)ide-naive patients with normal renal function.
- **Functional cure pipeline (2023–2025):** siRNA and antisense agents (bepirovirsen, xalnesiran, imdusiran) combined with immunomodulators are in phase 2b/3; none is yet approved, and **nucleos(t)ide analogues remain the standard of care**.
- **Bulevirtide** has full European approval for chronic hepatitis D, with improving long-term combined virological and biochemical response data.
- **Indian context:** HBsAg prevalence is approximately 1–3%, classing India as intermediate endemicity with an estimated 3–4 crore people infected. The **National Viral Hepatitis Control Programme** provides free tenofovir and free testing, and the hepatitis B birth dose is part of the Universal Immunisation Programme. Cost and access make **generic tenofovir the practical first-line agent** in most Indian settings.

Current Practice Update

Textbook Position	Current Evidence / Guideline Position
Treat patients who have cirrhosis or persistently elevated transaminases with detectable HBV DNA.	**WHO 2024 substantially widened eligibility** — treat all with cirrhosis or significant fibrosis (APRI >0.5), and treat on ALT or APRI alone where HBV DNA testing is unavailable.
Tenofovir is named as the oral once-daily antiviral of choice.	Three first-line options exist — **TDF, TAF and entecavir**. **TAF or entecavir is preferred in older adults** with eGFR <60, osteoporosis or concurrent nephrotoxic drugs.
Six-monthly ultrasound surveillance for HCC in HBsAg-positive patients.	Unchanged and reinforced — **surveillance continues lifelong even after HBV DNA becomes undetectable**, because HBV DNA integration means HCC risk is reduced but not eliminated.
Screening tests for hepatitis B are surface antigen and antibody.	Add **anti-HBc, HBV DNA, HBeAg/anti-HBe, and anti-HDV testing in all HBsAg-positive people** (WHO 2024); point-of-care viral load testing is now recommended for decentralised care.
Antivirals prevent progression of and reverse fibrosis.	Confirmed, and extended — long-term nucleos(t)ide therapy is now documented to produce **regression of established cirrhosis** and to reduce decompensation, HCC and transplant need.
The textbook does not discuss newer agents.	**siRNA and antisense agents (bepirovirsen, xalnesiran) and capsid assembly modulators** target functional cure, but remain investigational; **bulevirtide** is the first approved therapy for hepatitis D.

Examiner's Tip — verbatim mark-earning lines

- "**cccDNA in the hepatocyte nucleus is why hepatitis B cannot be cured** — the achievable goal is durable viral suppression, and the aspirational goal is functional cure, meaning HBsAg loss."
- "Name the three first-line agents: **tenofovir disoproxil fumarate, tenofovir alafenamide and entecavir** — all high genetic barrier, once daily."
- "In an older patient with reduced eGFR or osteoporosis, **choose TAF or entecavir over TDF**."
- "**Interferon is contraindicated in decompensated cirrhosis**; use entecavir or tenofovir and refer for transplantation."
- "**Every HBsAg-positive patient starting rituximab, chemotherapy or high-dose steroids needs antiviral prophylaxis** — reactivation can be fulminant."
- "**HBV causes hepatocellular carcinoma even without cirrhosis** — hence six-monthly ultrasound surveillance in all HBsAg-positive patients."
- "The **WHO 2024 guidelines expanded and simplified treatment eligibility**, allowing initiation on APRI or ALT alone where viral load testing is not available."
- "**Test every hepatitis B patient for hepatitis D** — bulevirtide is the first specific approved therapy."

Q6. Liver Transplantation

High-Yield Points

Definition, Types and Indications

1. **Liver transplantation is often the best treatment for advanced cirrhosis, acute hepatic failure, and some liver cancers.** It is the only definitive therapy for end-stage liver disease.
2. Types are **deceased-donor transplantation (whole or split graft)**, **living-donor transplantation (usually right lobe from a near relative)**, auxiliary transplantation, and domino transplantation. **In India the large majority of liver transplants are living-donor**, because of the shortage of deceased donors.
3. Indications are: decompensated cirrhosis of any cause; **acute liver failure**; **hepatocellular carcinoma within Milan criteria** (a single lesion ≤5 cm, or up to three lesions each ≤3 cm, with no vascular invasion or extrahepatic spread); metabolic liver disease; and intractable complications such as refractory pruritus or recurrent cholangitis.
4. The commonest indications in older recipients are now **metabolic dysfunction-associated steatohepatitis (MASH/NASH), alcohol-related liver disease and hepatocellular carcinoma**. Hepatitis C as an indication has fallen sharply since direct-acting antivirals.

Selection and Timing — the MELD Score

5. The decision on when to offer transplantation is based on the **Model for End-stage Liver Disease (MELD)**, a mathematical formula that grades life expectancy in a cirrhotic patient.
6. **MELD parameters are creatinine, sodium, bilirubin and INR.** Multiple online calculators exist.
7. **At a MELD score of 17, a patient is likely to do better with a transplant than without one.** Practice is to **refer to a transplant clinic once MELD exceeds 15**, provided the patient is a reasonable candidate.
8. **A 'reasonable candidate' is one who is otherwise healthy — with no cancer, no dementia and no cardiovascular disease.** Kidney disease is not a bar, as such patients can be listed for a combined liver–kidney transplant.
9. **In patients over 70, additional requirements are a good performance status, a stable and supportive home life, and a reasonable likelihood of living 10 years with the transplant.**
10. Referral should also be triggered by the **first episode of decompensation** — ascites, variceal bleeding, hepatic encephalopathy or spontaneous bacterial peritonitis — even if MELD is still low.

Outcomes and the Age Question

11. **Carefully selected patients over 65 have a good prognosis, with about 80% five-year survival.**
12. In the Mayo Clinic study comparing **42 patients over 70 with 42 matched patients under 60, five-year survival was identical**. **Chronological age alone is therefore not a contraindication.**
13. **Donor age is likewise not an absolute bar** — Eurotransplant data show that **using livers from donors older than 70 should not be discouraged**, because survival rates are good. The liver is less affected by age than other organs.
14. **Geriatric patients who are otherwise healthy should be considered for liver transplantation** — under-referral on grounds of age alone is a recognised failure of care.

Pre-transplant Evaluation in the Older Candidate

15. The work-up must cover: **cardiac assessment** (echocardiography, stress testing, coronary angiography where indicated — coronary disease is the commonest reason for exclusion), **pulmonary assessment** for hepatopulmonary syndrome and portopulmonary hypertension, renal function, **malignancy and infection screening**, dental clearance, bone density, and psychosocial and caregiver assessment.
16. Assess **nutrition, sarcopenia and frailty** formally — these predict waitlist mortality and post-transplant recovery better than age. Assess cognition, because dementia is an exclusion, and confirm **abstinence, insight and social support** in alcohol-related disease.

Immunosuppression and Complications

17. Immunosuppression uses **induction (basiliximab or anti-thymocyte globulin)** and **maintenance with a calcineurin inhibitor (tacrolimus), an antimetabolite (mycophenolate) and tapering steroids**; mTOR inhibitors (everolimus, sirolimus) are used to spare renal function and in HCC.

18. **Older recipients need less immunosuppression** because of immunosenescence — they have **less acute rejection but more infection, more de novo malignancy and more calcineurin-inhibitor nephrotoxicity**. Drug–drug interactions with tacrolimus (azoles, macrolides, calcium channel blockers, statins) are a major practical hazard in the polypharmacy setting.

19. Complications: **primary non-function, hepatic artery thrombosis, biliary strictures and leaks, acute and chronic rejection, CMV and opportunistic infection, post-transplant lymphoproliferative disorder, recurrence of the original disease, chronic kidney disease, metabolic syndrome, osteoporosis and de novo malignancy**.

Legal Framework in India and Alternatives

20. In India, transplantation is governed by the **Transplantation of Human Organs Act 1994, amended in 2011 (Transplantation of Human Organs and Tissues Act, THOTA) with Rules 2014**. It defines brain-stem death, requires certification by a **board of four medical experts**, permits near-relative and swap donation, requires **authorisation committee approval for non-near-relative (altruistic) donation**, and makes **commercial dealing in organs a punishable offence**.

21. The organisational network is **NOTTO at national level, ROTTOs regionally and SOTTOs at state level**, under the National Organ Transplant Programme.

22. For patients who are not candidates, the alternatives are **optimal medical management of cirrhosis** (beta-blockers and band ligation for varices, diuretics and albumin for ascites, TIPS for refractory ascites or bleeding, lactulose and rifaximin for encephalopathy), **locoregional therapy for HCC (TACE, radiofrequency ablation, SBRT)**, and **early palliative care with advance care planning**. Median survival in untreated decompensated cirrhosis is about two years.

Latest Updates (2023 – Present)

- **MELD 3.0 was implemented for organ allocation in 2023.** It adds **female sex and serum albumin** to the existing variables, updates the creatinine ceiling and the interactions between components. It corrects the systematic disadvantage that MELD-Na imposed on women, who were being under-prioritised because of lower muscle mass and hence lower creatinine.
- **Machine perfusion has transformed organ preservation. Normothermic machine perfusion (NMP)** allows ex-vivo perfusion at body temperature with viability testing before implantation; **hypothermic oxygenated perfusion (HOPE)** reduces ischaemia–reperfusion injury and biliary complications.
- **UNOS/OPTN analyses (Transplantation Direct 2024; World J Transplant 2025)** show that NMP increases the use of donation-after-circulatory-death and extended-criteria grafts, and is associated with **better two-year patient and graft survival** in DCD recipients after propensity matching.
- **Machine perfusion is directly relevant to older recipients** — matched analyses show that despite receiving more marginal grafts (older donors, more DCD), elderly NMP recipients had **similar graft survival and a shorter length of stay**, so NMP may mitigate donor risk factors that would otherwise be relative contraindications.
- **Normothermic regional perfusion (NRP)** in the donor before retrieval, alone or combined with NMP, is now an established DCD strategy.
- **The Liver Frailty Index (grip strength, timed chair stands, balance testing)** is now a standard, validated waitlist assessment; **CT-measured sarcopenia at the L3 level** independently predicts waitlist and post-transplant mortality. Structured **prehabilitation** before transplantation is increasingly offered.
- **Early liver transplantation for severe alcohol-associated hepatitis** is now accepted in carefully selected patients, replacing the rigid six-month abstinence rule; selection is by psychosocial assessment rather than by a fixed time interval.
- **Allocation reform:** the United States has moved from regional boundaries to **acuity circles** and is moving towards **continuous distribution**, reducing geographic inequity.

- **Expanded donor pool: HCV-viraemic donors** are now used for HCV-negative recipients with short-course DAA prophylaxis, and **anti-HBc-positive donors** are used with antiviral prophylaxis — both are especially valuable for older candidates who cannot afford a long wait.
- **MASH/MASLD is now among the leading indications for liver transplantation** in high-income countries and is rising rapidly in India; these recipients are older and carry more cardiovascular and metabolic risk, so cardiac work-up standards have tightened.
- **Indian context:** India performs roughly 3,000–4,000 liver transplants a year, **the great majority living-donor**, and is second only to the USA in absolute LDLT numbers. Deceased-donation rates remain below one per million population. Recent national policy changes have **removed the upper age limit of 65 years for registration for deceased-donor organs**, removed the domicile requirement so a patient may register in any state, and removed the registration fee — all of which improve access for older Indian patients.

Current Practice Update

Textbook Position	Current Evidence / Guideline Position
MELD is based on creatinine, sodium, bilirubin and INR.	**MELD 3.0 (2023)** additionally incorporates **female sex and serum albumin**, correcting the disadvantage previously experienced by women on the waitlist.
Refer to a transplant clinic when MELD exceeds 15; transplant benefit begins around MELD 17.	Unchanged, but now supplemented by **referral at the first decompensating event** regardless of MELD, and by exception points for HCC and other conditions.
A reasonable candidate has no cancer, no dementia and no cardiovascular disease.	HCC **within Milan criteria is an indication, not an exclusion**; criteria have been extended (UCSF, up-to-seven) and downstaging protocols are accepted. Cardiovascular disease is assessed and treated rather than being an automatic bar.
Patients over 70 need good performance status and a likely 10-year survival.	Age-based thresholds have been replaced by **objective frailty and sarcopenia measurement (Liver Frailty Index, L3 skeletal muscle index)**, which predict outcome far better than chronological age.
Livers from donors older than 70 give good survival and should not be discouraged.	Reinforced, and extended — **machine perfusion allows objective viability testing** of marginal and older grafts before implantation, further safely expanding the donor pool.
Alcohol-related liver disease requires a period of abstinence before listing.	**Early transplantation for severe alcohol-associated hepatitis** is now accepted with psychosocial selection, without a mandatory six-month rule.

Examiner's Tip — verbatim mark-earning lines

- "**MELD uses creatinine, sodium, bilirubin and INR** — refer above 15, and transplant benefit exceeds risk at 17."
- "**Carefully selected patients over 65 achieve about 80% five-year survival**, and the Mayo series showed identical five-year survival in patients over 70 compared with matched patients under 60."
- "**Age is not a contraindication to liver transplantation — frailty is.**"
- "**Livers from donors older than 70 should not be discouraged** — the liver ages better than most other organs."
- "**Milan criteria: one lesion ≤5 cm, or up to three lesions each ≤3 cm, with no vascular invasion or extrahepatic spread.**"
- "Older recipients have **less rejection but more infection and malignancy** because of immunosenescence — reduce immunosuppression accordingly."
- "In India, transplantation is governed by **THOTA 1994/2011 with Rules 2014**; brain death is certified by a **board of four experts**, and commercial dealing is punishable."
- "Quote **MELD 3.0 and machine perfusion** as the two practice-changing advances since 2023."

Q7. Eye Donation

Eye donation is not covered as a topic in the uploaded textbook. *The textbook refers to corneal transplantation only in passing — noting that the corneal endothelium undergoes degenerative change with ageing, that these cells seldom proliferate in adult life so the cell population falls, and that in some patients* ***endothelial dystrophy progresses spontaneously to corneal oedema, and such cases require corneal transplants****. The content below is therefore drawn from the Transplantation of Human Organs and Tissues Act, the Standards of Eye Banking in India (DGHS/NPCB&VI;), Eye Bank Association of India documents and current peer-reviewed literature.*

High-Yield Points

Definition and Need

1. **Eye donation is the voluntary donation of the eyes after death**, for corneal transplantation, for research, or for training. **Only the cornea is transplanted — the whole eye cannot be transplanted**, because the optic nerve cannot be reconnected.
2. **Corneal blindness is a leading cause of avoidable blindness in India**, with a large accumulated backlog and a substantial annual incidence. Demand for donor tissue far exceeds supply, and **the utilisation rate of collected corneas is only about 50%**.
3. Corneal blindness is important in geriatrics because **pseudophakic and aphakic bullous keratopathy, Fuchs endothelial dystrophy, corneal degeneration and infective keratitis** are the typical indications in older Indian patients — and because **most donors are older adults**.

Legal and Organisational Framework in India

4. Eye banking is governed by the **Transplantation of Human Organs Act 1994, amended in 2011 (THOTA), with Rules 2014**. Eye banks and eye donation centres must be registered under the Act, and **commercial dealing in tissue is a punishable offence**.
5. **The National Programme for Control of Blindness and Visual Impairment (NPCB&VI;)** provides grants-in-aid for eye collection and infrastructure; the **Eye Bank Association of India (EBAI)** sets professional standards; and the **National Eye Bank at AIIMS, New Delhi** is the apex institution.
6. The service structure is a three-tier one — **eye donation centres** (motivate and collect), **eye retrieval centres** (retrieve tissue) and **eye banks** (retrieve, evaluate, preserve and distribute). Only a registered eye bank may distribute tissue.
7. **Service fees may be charged only to recover the cost** of retrieval, processing, storage and distribution; **there must be no profiteering**, and the charging policy must be approved by the competent authority.

Who Can Donate and Who Cannot

8. **Anyone can donate — any age, any sex, any blood group.** Spectacle wearers, people who have had cataract surgery, and those with diabetes or hypertension can all donate. Pledging during life is encouraged but is not legally binding.
9. **Absolute systemic contraindications:** death of unknown cause; **HIV; hepatitis B; hepatitis C; active syphilis; rabies; septicaemia; leukaemia and lymphoma; disseminated malignancy; Creutzfeldt–Jakob disease and other prion disease; active viral encephalitis; subacute sclerosing panencephalitis; congenital rubella; Reye syndrome; and tetanus**.
10. **Ocular contraindications:** retinoblastoma and other ocular malignancy, active ocular or intraocular inflammation, and previous anterior segment surgery of unknown nature. Prior refractive surgery is a relative contraindication for optical grafts.
11. **Even where an absolute contraindication exists, the family must be told clearly, and the eyes are not retrieved unless the family is fully informed and still wishes to donate.** Tissue unsuitable for transplantation may be used for research and training.

Procedure — What to Do at the Time of Death

12. **Inform the nearest eye bank immediately.** Eyes should be retrieved **within 6 hours of death** (this may be extended when the body is refrigerated).
13. Simple measures while awaiting the team: **close the eyelids; switch off the fan; raise the head of the body by about six inches; place moist cotton or a cold pack over the closed lids; instil**

antibiotic drops if available; and keep a copy of the death certificate ready.

14. **Written consent must be obtained on the prescribed form from the legal custodian or next of kin**, even if the deceased had pledged. Death must be certified by a registered medical practitioner, or the retrieval team must satisfy itself that life is extinct.
15. **Retrieval is by either enucleation (removal of the whole globe) or in-situ corneoscleral rim excision.** Corneoscleral excision is preferred in hospital programmes — it is quicker, uses less tissue and gives better cosmesis. It is performed by trained personnel using sterile technique, in a laminar flow hood or operating room where done in a laboratory.
16. **A donor blood sample is mandatory** and is tested for **HIV 1 and 2, HBsAg, anti-HCV and syphilis (VDRL/TPHA)**. The tissue is graded on **slit-lamp and specular microscopy**; **endothelial cell density (generally above 2000–2500 cells/mm²) determines whether tissue is optical grade or usable only for tectonic or therapeutic purposes**.

Preservation and Use

17. **Preservation options:** moist chamber (up to 24–48 hours), **McCarey–Kaufman medium (2–4 days)**, **Optisol-GS at 2–8°C (up to 14 days)** and **organ culture at 31–37°C (up to 4 weeks)**.
18. **Uses:** optical keratoplasty (penetrating keratoplasty, DALK, DSAEK, DMEK), therapeutic and tectonic grafts, patch grafts for glaucoma drainage devices, limbal stem cell transplantation, and research and surgical training.
19. **One donor traditionally gives sight to two people.** With modern component (lamellar) surgery, a single cornea can be split so that **one donor may benefit more than two recipients**.

Sources, Barriers and Promotion

20. **Voluntary (home-death) donation** gives lower yield and poorer tissue quality because of delay. **The Hospital Cornea Retrieval Programme (HCRP), launched by EBAI in 1991, is the main source of good-quality tissue** — trained grief counsellors approach the families of all patients dying in hospital.
21. **HCRP advantages:** no potential donor is missed; a detailed medical history is available to assess eligibility; younger donors are accessed; the death-to-retrieval interval is short; and it is cost-effective. **Structured grief counselling is the single most effective intervention** for raising consent conversion.
22. **Barriers:** lack of public awareness; family refusal at a time of grief; myths about disfigurement, religious objection and beliefs about rebirth; failure of hospital staff to intimate the eye bank; shortage of trained counsellors; non-uniform standards between eye banks; and inadequate accreditation.
23. **Promotion: National Eye Donation Fortnight is observed from 25 August to 8 September** every year. Other measures are pledge registration, mass media and community campaigns, involvement of religious and community leaders, and **sensitisation of ICU, casualty and ward staff** to identify and refer every eligible death.

Geriatric and Ethical Aspects

24. **Donor age is not a bar.** The Cornea Donor Study showed that graft survival at five and ten years is comparable across donor ages from 12 to 75 years — so older donors are entirely acceptable and are in fact the main donor pool.
25. The geriatrician's role is practical: **raise eye donation during advance care planning**, include it in advance directive discussions alongside organ and tissue donation, counsel families sensitively, and ensure timely intimation to the eye bank after an expected death.
26. **Ethical principles:** voluntary and informed consent; no reward, inducement or coercion; dignity and respect for the body; confidentiality between donor and recipient families; equitable allocation on clinical need; and complete **documentation, traceability and recall** systems.

Latest Updates (2023 – Present)

- **Standards of Eye Banking in India (2020, NPCB&VI; with EBAI)** — an updated national standard replacing the 2009 DGHS document. It sets accreditation criteria (an eye bank must collect at least **25 surgical-grade tissues a year** to be accredited, otherwise it is re-designated as an eye retrieval centre), mandates periodic re-inspection, restates that **service fees may only recover cost with no profiteering**, and requires documented tissue tracking and recall.

- **AIOS–EBAI consensus statement on cornea and eye banking during the COVID-19 era (Indian Journal of Ophthalmology 2020)** — added respiratory-infection screening of donors, deferral criteria, PPE requirements and safe restart protocols. It remains the template for pandemic preparedness in eye banking.
- **Shift from penetrating keratoplasty to endothelial and lamellar keratoplasty: DSAEK and DMEK for endothelial disease and DALK for stromal disease** now dominate practice. Because these use only part of the cornea, **a single donor cornea can be split between recipients**, effectively multiplying the impact of each donation.
- **Pre-cut and pre-loaded tissue** prepared by the eye bank (eye-bank-prepared DMEK/DSAEK grafts) reduces intra-operative tissue loss and surgeon preparation time, and is being adopted by larger Indian eye banks.
- **Cell-based therapy: cultivated human corneal endothelial cell injection** (pioneered in Japan) has shown durable restoration of corneal clarity in bullous keratopathy, and further trials are ongoing. If successful at scale, a single donor cornea could treat dozens of recipients.
- **Bioengineered corneal implants** from medical-grade porcine collagen (reported in Nature Biotechnology, 2022, with two-year outcomes in India and Iran) restored vision in advanced keratoconus with a minimally invasive, suture-free technique — a potential low-cost alternative to donor tissue in resource-limited settings.
- **National Organ Transplant Programme policy changes (2023–2024)** — the Union Health Ministry directed states to **remove the upper age limit of 65 years for registration for deceased-donor organs**, to **remove the domicile requirement** so that a patient may register in any state, and to **abolish registration fees**. These reduce structural barriers for older patients across transplantation.
- **2025 prospective Indian studies (including from Gandhi Medical College, Bhopal)** confirm that **dedicated grief counselling within the HCRP significantly increases both willingness to donate and actual conversion to donation** — supporting investment in counsellor posts rather than in mass publicity alone.
- **Persisting gap:** India procures a substantial number of corneas each year but the **utilisation rate remains around 50%**. Current policy emphasis has therefore shifted from raising collection numbers to **improving tissue quality, shortening death-to-preservation time, standardising medical criteria and strengthening HCRP**.
- **Regenerative and alternative approaches** — simple limbal epithelial transplantation (SLET), cultivated limbal epithelial transplantation (CLET) and keratoprosthesis (Boston KPro, LVP-KPro) — reduce dependence on donor tissue in bilateral limbal stem cell deficiency and repeated graft failure.

Current Practice Update

Textbook Position	Current Evidence / Guideline Position
The textbook mentions only that corneal endothelial dystrophy progressing to corneal oedema requires a corneal transplant.	Endothelial disease is now treated by **DSAEK or DMEK rather than full-thickness penetrating keratoplasty**, with faster visual recovery, lower rejection rates and the ability to split a single donor cornea between recipients.
No guidance is given on the source of donor tissue.	The **Hospital Cornea Retrieval Programme (HCRP)**, not voluntary home-death donation, is now the principal source of surgical-grade tissue in India.
No preservation guidance is given.	**Optisol-GS at 2–8°C allows storage up to 14 days** and organ culture up to 4 weeks, replacing moist-chamber storage for planned surgery.
No donor age guidance is given.	The **Cornea Donor Study** established that graft survival is comparable for donors aged 12 to 75 — **older age is not a bar to corneal donation**.
Corneal transplantation depends entirely on donor tissue.	**Cultivated corneal endothelial cell therapy, bioengineered collagen implants, SLET/CLET and keratoprosthesis** are emerging alternatives that will reduce dependence on donor corneas.

Examiner's Tip — verbatim mark-earning lines

- "**Only the cornea is transplanted — the whole eye cannot be transplanted**, because the optic nerve cannot be reconnected."
- "**Eyes must be retrieved within 6 hours of death.** Close the lids, switch off the fan, raise the head six inches, apply moist cotton or ice packs, and inform the eye bank at once."
- "**Consent must come from the legal custodian even if the deceased had pledged** — a pledge is not legally binding in India."
- "Name the absolute contraindications: **death of unknown cause, HIV, hepatitis B and C, rabies, septicaemia, syphilis, leukaemia and lymphoma, disseminated malignancy, CJD, viral encephalitis, SSPE, congenital rubella and tetanus**."
- "**Age is not a bar to eye donation** — spectacle wearers, cataract-operated patients, diabetics and hypertensives can all donate."
- "**The Hospital Cornea Retrieval Programme (EBAI, 1991) is the main source of good-quality tissue** — it avoids missing donors, gives a full medical history and shortens the death-to-retrieval interval."
- "**National Eye Donation Fortnight runs from 25 August to 8 September** each year."
- "Eye banking is regulated under **THOTA 1994/2011 with Rules 2014**, supported by NPCB&VI;, with EBAI setting standards and the National Eye Bank at AIIMS as the apex centre."
- "**One donor gives sight to two people** — and with lamellar surgery, to more than two."

Q8. Sleep Protocol

*In geriatric practice a 'sleep protocol' means a **standardised, non-pharmacological package of measures used to promote sleep in older people — particularly hospitalised older people — so that sedative-hypnotic drugs can be avoided**. It has two components: the ward-level non-pharmacological sleep protocol (a core element of delirium prevention), and the sleep hygiene and behavioural programme used for chronic insomnia. Both are covered below.*

High-Yield Points

Definition and Rationale

1. A sleep protocol is a **written, nurse-delivered, non-drug bundle of measures given at bedtime** to any older patient who has difficulty initiating sleep or who requests a sedative, with the aim of avoiding hypnotic drugs.
2. It is needed because **hospitalised older people sleep badly** — noise, light, nocturnal observations and blood sampling, pain, nocturia, unfamiliar environment and daytime inactivity all fragment sleep — and because **sedative-hypnotics in older people cause delirium, falls, fractures, daytime sedation, cognitive impairment and increased mortality**.
3. **Normal ageing changes in sleep** must be distinguished from disease: **increased sleep latency, reduced deep (slow-wave) sleep, reduced REM sleep, more frequent awakenings, reduced sleep efficiency, phase advance (early to bed and early to rise), and more daytime napping**. Total sleep need does not change much.

The Classical Non-pharmacological Sleep Protocol

4. The original protocol (McDowell and Inouye, JAGS 1998) has **three components — a warm drink (milk or herbal tea), relaxation music or tapes, and a back rub or massage**.
5. **It is given to any patient complaining of difficulty initiating sleep or requesting a sedative; the sedative is given only if the patient still requests it after one hour.** Adherence in the original study was 74%, and it **substantially reduced the use of sedative-hypnotic drugs** with no toxicity.
6. The sleep protocol is one of six standardised protocols in the **Hospital Elder Life Program (HELP)**, alongside those for cognitive impairment, immobility, visual impairment, hearing impairment and dehydration. **HELP prevents delirium in 30–40% of cases** and reduces falls, length of stay and cost.

Environmental and Unit-Level Measures

7. **Reduce noise:** silent pagers and mobile phones, lowered alarm and television volume, soft-close bins and doors, staff conversation moved away from the bedside, and designated **'quiet hours' (typically 10 pm to 6 am)**.
8. **Control light:** dim corridor and bay lights at night, use torches rather than overhead lights, and provide **eye masks and earplugs** to willing patients.
9. **Cluster nursing activities** so that patients are not woken repeatedly. **Avoid non-essential night-time observations, blood sampling, weighing and medication rounds**; give diuretics early in the day and avoid overnight intravenous fluids where possible.
10. **Treat what wakes the patient:** pain (multimodal analgesia), dyspnoea, cough, nocturia, gastro-oesophageal reflux, pruritus, cramps and anxiety.

Daytime Measures — Chronotherapy

11. **Increase daytime activity, especially outdoors and in sunlight;** get the patient out of bed, dressed in day clothes, and mobilised. **Avoid prolonged daytime sleep and going to bed too early.**
12. **Scheduled bright-light therapy** resynchronises the circadian rhythm by entraining the suprachiasmatic nucleus to the light/dark cycle, and is particularly useful in institutionalised elders and in dementia. Timing must be individualised.
13. **Exercise timing matters:** afternoon exercise advances the biological clock, while exercise at night delays the circadian phase. Light exercise should be finished **at least three hours before bedtime**.

Sleep Hygiene Measures

14. **Keep a regular schedule** — go to bed and wake at the same time daily; constant changes increase the probability of severe, chronic sleep difficulty.
15. **Optimise the environment** — temperature about 18–21°C, no noise or light, a proper mattress. **Avoid heavy meals, but do not go to bed hungry or thirsty**; dairy products contain tryptophan, which helps induce sleep.
16. **Limit stimulants** — coffee, tea, cola, alcohol and nicotine, especially after 5 pm. Alcohol is often self-prescribed for insomnia but **shortens latency and then fragments the second half of the night**.
17. **Remain in bed no more than eight hours** — spending too long in bed produces fragmented, light sleep. **Allow a nap after the midday meal of no more than 30 minutes.** Follow a fixed **bedtime ritual**.

Assessment Before and During the Protocol

18. **Take a full sleep history** from the patient and, where possible, the bed partner or caregiver. Ask specifically about mood, since insomnia and depression have a two-way relationship.
19. **Rule out treatable specific sleep disorders** — **obstructive sleep apnoea** (loud snoring, witnessed apnoea, daytime sleepiness; treat with CPAP), **restless legs syndrome** (check iron; dopaminergics), **periodic limb movement disorder**, **REM sleep behaviour disorder** (clonazepam; and it predicts Parkinson's disease and dementia), and **advanced sleep phase**.
20. **Review the drug chart** — beta-blockers, corticosteroids, NSAIDs, bronchodilators, diuretics, SSRIs, cholinesterase inhibitors, decongestants and caffeine-containing preparations all disturb sleep.
21. **Keep a sleep diary for a minimum of two consecutive weeks**, because of day-to-day variability. Actigraphy or wrist accelerometers may help. **Polysomnography, multiple sleep latency testing and maintenance of wakefulness testing are not routinely required** for insomnia unless another disorder is suspected.

When Drugs Are Considered

22. **Cognitive behavioural therapy for insomnia (CBT-I) is the first-line treatment** — sleep restriction, stimulus control, relaxation, cognitive restructuring and sleep hygiene. **It is effective even in patients with cognitive decline**, and its benefit outlasts drug therapy.
23. **Melatonin is the hypnotic of first choice when a drug is indicated in a patient over 55.** It improves sleep latency and quality without altering sleep architecture, shows efficacy for up to six months with **no tolerance, rebound or withdrawal**, does not impair next-morning psychomotor performance or driving, and **does not increase postural instability (unlike zolpidem)**. In mild-to-moderate Alzheimer's disease it was associated with significantly less cognitive impairment.
24. **Avoid benzodiazepines and Z-drugs.** They shorten sleep latency and increase total sleep time but **do not improve, and may worsen, daytime function**. Z-drugs such as zolpidem cause awakenings, complex behaviours (sleepwalking, hallucinations) and rebound; the class causes memory and cognitive impairment, disinhibition, delirium, falls, accidents and increased mortality.
25. **Avoid antihistamines** — their anticholinergic effect causes urinary retention, constipation and delirium. **Avoid quetiapine even at low doses**, and use other antipsychotics only with great caution because of the risk of sudden death in the elderly.
26. If a hypnotic is unavoidable, use the **lowest effective dose for the shortest possible time, with a documented review date and exit plan**, and never continue a hospital-initiated hypnotic on the discharge prescription.

Special Settings and Outcomes

27. **Institutionalised elders** are at particular risk because of comorbidity, polypharmacy and the institutional environment (loud noise, low light exposure, few circadian cues). They need an **integrated pharmacological and environmental approach**; circadian disorders respond well to chronotherapy, melatonin and properly scheduled light therapy, **thereby avoiding the need for sedative-hypnotics**.
28. **Outcomes of a sleep protocol:** reduced hypnotic use, less delirium, fewer falls, shorter length of stay, better patient satisfaction, and no adverse events — at negligible cost. Monitor with a sleep diary, the **Pittsburgh Sleep Quality Index, Insomnia Severity Index and Epworth Sleepiness Scale**, and hand over the sleep plan at every nursing shift change.

Latest Updates (2023 – Present)

- **2023 AGS Beers Criteria** — benzodiazepines, non-benzodiazepine 'Z-drug' hypnotics (zolpidem, zopiclone, eszopiclone) and first-generation antihistamines remain **potentially inappropriate medications in all older adults** and should be avoided for insomnia. This reinforces the non-pharmacological protocol as the default.
- **Digital CBT-I (dCBT-I)** delivered by app or web is now shown to be effective in older adults and addresses the chronic shortage of trained therapists; it is increasingly recommended as a first step where face-to-face CBT-I is unavailable.
- **Dual orexin receptor antagonists (DORAs) — suvorexant, lemborexant and daridorexant — are the most important new drug class.** A systematic review and meta-analysis in older adults showed that compared with placebo they **reduce wake after sleep onset and latency to persistent sleep and increase total sleep time at both 1 and 3 months**, including subjective total sleep time.
- **Safety profile of DORAs in the elderly: daridorexant showed a favourable safety profile**, whereas lemborexant and suvorexant had higher rates of treatment-emergent adverse events than placebo, without an increase in serious adverse events. Class effects to counsel about are **nightmares, abnormal dreams, sleep paralysis, hallucinations and next-day somnolence**.
- **Agent selection by sleep parameter:** network meta-analyses suggest **lemborexant is best for sleep onset** and **daridorexant for sleep maintenance**; daridorexant also has the lowest rebound risk, and is therefore the preferred agent when switching during deprescribing.
- **Deprescribing guidance:** expert consensus opposes a blanket prohibition on long-term use of DORAs and some non-benzodiazepine agents, but requires **periodic re-evaluation of the risk-benefit balance, particularly in older adults**; when stopping, **taper gradually** rather than stopping abruptly.
- **DORAs and delirium:** small studies of suvorexant and lemborexant in critically ill and post-operative patients suggest a possible reduction in delirium incidence, in contrast with benzodiazepines; the evidence is not yet strong enough for routine recommendation.
- **Melatonin and ramelteon for delirium prevention** have shown inconsistent results across trials and meta-analyses, and melatonin is not universally endorsed for this indication — but it remains the preferred hypnotic in older adults with insomnia.
- **Multicomponent non-pharmacological bundles** — earplugs and eye masks, relaxation and guided imagery, massage, music therapy, aromatherapy and warm milk-based drinks — were confirmed in 2024–2025 systematic reviews to improve sleep quality and duration in hospitalised adults, **with no adverse events reported**. Back massage in one study increased total sleep time by more than an hour.
- **Sleep and dementia risk:** longitudinal data continue to link insomnia and short sleep with subsequent cognitive impairment, and short sleep (under six hours) with higher cardiovascular mortality — strengthening the case for treating sleep as a modifiable risk factor rather than a nuisance symptom.
- **Indian context:** hypnotic and benzodiazepine prescribing to older adults remains high and is often over-the-counter; alprazolam and diazepam are widely used despite their long half-lives. Practical priorities are **deprescribing benzodiazepines, screening for OSA (which is underdiagnosed in India), and building simple ward sleep protocols into NPHCE geriatric wards and district hospital care**.

Current Practice Update

Textbook Position	Current Evidence / Guideline Position
Sleep hygiene and cognitive behavioural therapy are the first level of action.	Unchanged and strengthened — **CBT-I is first-line for chronic insomnia at every age**, and **digital CBT-I** now extends access. Sleep hygiene alone is insufficient as monotherapy for chronic insomnia.
Melatonin is the treatment of first choice when a hypnotic is indicated in patients over 55.	Still the preferred first hypnotic, but **dual orexin receptor antagonists are now a validated second option** with meta-analytic support in older adults and a better safety profile than Z-drugs.

Textbook Position	Current Evidence / Guideline Position
Benzodiazepines and Z-drugs are listed with dosing in the pharmacological table but with cautions.	The **2023 AGS Beers Criteria classify all benzodiazepines and Z-drugs as potentially inappropriate in older adults** — they should be avoided for insomnia, and existing prescriptions should be actively deprescribed with a taper.
Antihistamines should be avoided because of anticholinergic effects; quetiapine is not recommended.	Unchanged and reinforced by anticholinergic burden and antipsychotic mortality data.
Polysomnography and MSLT are not routinely recommended for insomnia.	Unchanged, but **consumer wearables and validated actigraphy** are increasingly used for longitudinal monitoring, complementing the two-week sleep diary.
The pharmacological table does not include the orexin antagonist class.	**Suvorexant, lemborexant and daridorexant** are now available; select **lemborexant for sleep-onset insomnia and daridorexant for sleep-maintenance insomnia and where deprescribing is planned**.

Examiner's Tip — verbatim mark-earning lines

- "The classical non-pharmacological sleep protocol has **three components — a warm drink, relaxation music, and a back rub** — and the sedative is given only if the patient still asks for it after one hour."
- "The sleep protocol is one of the **six HELP protocols**; the other five target cognitive impairment, immobility, visual impairment, hearing impairment and dehydration. **HELP prevents delirium in 30–40% of cases.**"
- "Name the ageing changes in sleep: **increased sleep latency, less slow-wave and REM sleep, more awakenings, reduced sleep efficiency, and phase advance**."
- "**CBT-I is first-line, and is effective even in patients with cognitive decline.**"
- "**Melatonin is the hypnotic of first choice in patients over 55** — no tolerance, no rebound, no withdrawal, and unlike zolpidem it does not increase postural instability."
- "**Benzodiazepines and Z-drugs do not improve daytime function and may worsen it**, and are potentially inappropriate in all older adults under the 2023 Beers Criteria."
- "**Avoid antihistamines and avoid quetiapine even at low doses** for sleep in older people."
- "Keep a **sleep diary for a minimum of two consecutive weeks**; polysomnography is not routinely needed for insomnia."
- "In institutionalised elders, use an **integrated environmental and pharmacological approach with chronotherapy, melatonin and timed light therapy — thereby avoiding sedative-hypnotics**."

Rehabilitation in Geriatric Medicine

MD Geriatric Medicine — Examination Compilation

High-Yield Answers with Latest Updates (2023–2026)

Ten Questions • Textbook Core + Current Evidence + Indian Context

Primary references: Pathy’s Principles and Practice of Geriatric Medicine (6th edition)
Kane’s Essentials of Clinical Geriatrics

Prepared for Dr. Sudarsan | Compiled 19 August 2026

Contents

1.	Definition, evaluation and components of rehabilitation in the elderly	15 marks
2.	Heading an interdisciplinary rehabilitation ward — team selection, job responsibilities, conflict management and care planning	15 marks
3.	Role of the occupational therapist on the team caring for the older person, on the ward and at discharge	15 marks
4.	Stroke rehabilitation — factors to consider before initiating long-term rehabilitation	15 marks
5.	Rehabilitation of an older adult with low vision	15 marks
6.	Rehabilitation of a patient with osteoarthritis of the knee joints	15 marks
7.	Community-based rehabilitation	15 marks
8.	Mobility aids	15 marks
9.	Assistive devices for the elderly	15 marks
10.	Artificial limb — rehabilitation of the elderly amputee	15 marks

How to use this compilation

Each answer follows the same architecture: **High-Yield Points** (textbook core) → **Latest Updates (2023–present)** with named trials and guidelines → **Current Practice Update** comparing textbook position with current evidence → **Exam One-Liners** for rapid recall. All laboratory and clinical values are in Indian conventional units. Indian epidemiology, programmes and legal frameworks are integrated throughout.

Five threads that run through every answer in this module

1. **WHA76.6 (May 2023)** — the first-ever World Health Assembly resolution on rehabilitation; rehabilitation is now an essential service within Universal Health Coverage.
2. **2.41 billion people** worldwide would benefit from rehabilitation (up 63% since 1990); **over half in LMICs receive none**.
3. **“No rehabilitation potential” is no longer an acceptable reason to deny access** (BGS 2024).
4. **Geriatric rehabilitation now reduces mortality, care-home admission and functional impairment** (BMC Medicine meta-analysis 2024).
5. **Indian delivery web:** NPHCE (19 Regional Geriatric Centres, 2 National Centres for Ageing) + DDRCs in all districts + ADIP/ALIMCO + Rashtriya Vayoshri Yojana + AB-PMJAY extended to all aged ≥70 years (2024).

Q1. Definition, evaluation and components of rehabilitation in the elderly

High-Yield Points

A. Terminology and Definition

1. **Rehabilitation** is a process that seeks to **minimise activity and participation restrictions resulting from impairment**.
2. **UN definition:** a **goal-oriented and time-limited process** enabling an impaired person to reach an **optimum mental, physical and/or social functional level**, giving them the tools to change their own life. Includes measures to **compensate for lost function** (technical aids) and to **facilitate social adjustment or readjustment**.
3. The current framework is the **WHO ICF (2001)**, replacing ICIDH (1980). Part 1 — Functioning and Disability: (a) Body Functions and Structures, (b) Activities and Participation. Part 2 — Contextual Factors: (a) Environmental, (b) Personal.
4. **Key definitions: Impairment** = problem in body function/structure; **Activity** = execution of a task; **Activity limitation** = difficulty executing activities; **Participation** = involvement in a life situation; **Participation restriction** = problem in that involvement. **Functioning** is the umbrella positive term; **disability** the collective negative term.
5. Functioning results from a **dynamic interaction between the health condition and contextual factors**. **Vicious cycles** develop easily — e.g. Parkinson's disease → fall → hip fracture → loss of confidence → isolation → depression → further decline.
6. **Why the elderly differ:** the biggest single reason is **lack of physiological reserve** to combat a disabling insult. Recovery is **prolonged** and often **does not return to the premorbid state**; ongoing limitations from osteoarthritis, Parkinson's disease and other comorbidities coexist.

B. Determinants of Disability

7. **Determinants of activity restriction:** type of impairment (nature and severity), **presence of associated impairments**, **degree of physiological reserve**, and **level of physical fitness**. Weight training improves muscle strength and functional capacity even in old age.
8. **Determinants of participation restriction:** *Intrinsic* — attitude, personality, **ability to adjust**, cultural issues. *Extrinsic* — financial resources, housing, other resources, **social supports** (spouse, family, neighbours, friends, pets). **Elderly women are particularly disadvantaged**, often widowed and living alone.
9. **Psychological aspects:** loss of function produces a **grief-like reaction** — denial → anger → depression → acceptance. Over **40% become depressed after myocardial infarction**; depression is high after stroke and worsens prognosis. **Kemp's model:** motivation is driven by **wants, beliefs and rewards**, counteracted by the **cost** in pain and effort.

C. Principles of Rehabilitation

10. **Early intervention is crucial** — avoidable restriction occurs soon after impairment and once established may be irremediable (e.g. correct positioning of a flaccid hemiplegic arm prevents **shoulder subluxation**).

11. **The team approach is essential** — medical and nursing staff, **physiotherapist, occupational therapist, speech and language therapist**, clinical psychologist, dietician, social worker. **Doctors treat impairment; remedial therapists treat activity restriction; social workers treat participation restriction; nurses have a holistic role.** Regular **formal team meetings** review progress, revise goals, plan discharge and arrange follow-up.

D. The Rehabilitation Process — Eight Steps

12. **Assessment → Goal setting → Therapy → Aids and adaptations → Education → Psychological support → Evaluation → Follow-up.** These overlap in practice and must be revisited **at least weekly**.

E. Evaluation / Assessment

13. **Purpose:** to confirm the problem is **remediable**, to select suitable patients, and to record a **baseline functional status** so progress can be measured. It must address both *"the problem in the individual"* and *"the individual with the problem"*. Denying rehabilitation to those who may benefit even slightly is unjustified.
14. **Assessment of activity restriction** begins with the clinical history, comparing **premorbid** and **current** function using **ADLs** (washing, grooming, dressing, toileting, feeding) and **IADLs** (cooking, telephone, laundry, shopping, housework, transport). Ask **how the person manages** — is the task neglected, or is help given?

Standardised scales endorsed by the Royal College of Physicians and British Geriatrics Society:

Domain	Scale	Comment
Basic ADLs	**Barthel Index**	Observation of what the patient *does*; **ceiling effect** in ambulatory patients
Vision, hearing, communication	**Lambeth Disability Screening Questionnaire**	Postal questionnaire
Memory and cognition	**Abbreviated Mental Test (AMT)**	10 items from the longer Roth–Hopkins test
Depression	**Geriatric Depression Scale**	30 items; 15-item short form
Subjective morale	**Philadelphia Geriatric Center Morale Scale**	Distinct from depression

15. The **Functional Independence Measure (FIM)** scores **18 functions** across **self-care, sphincter control, mobility, locomotion, communication and social cognition**. **Assessment of participation restriction is subjective, unstable over time and largely neglected** in clinical and research settings.

F. Components / Remaining Steps

16. **Goal setting:** goals must be **realistic**, agreed by **the patient and all team members**, split into **short-term (intermediate)** and **long-term (final)** goals with **flexible time frames**, reviewed regularly. Patients with ambitious goals progress more — so set goals that are **ambitious but achievable** (e.g. wheelchair independence rather than walking for some amputees).
17. **Therapy:** the doctor manages disease, comorbidity, risk factors and complications; **OT** enhances ADL competence; **physiotherapist** targets cardiorespiratory, neuromuscular and

locomotor function; **speech and language therapist** manages communication and swallowing.

18. **Aids and adaptations:** a **prosthesis** replaces a body part; an **orthosis** is applied externally to support, improve function, or restrict/promote movement. Aids may **foster dependence if wrongly prescribed**, yet many in real need never obtain even basic aids. **Home modification** (handrails, ramps, shower/toilet access) after an early **OT home visit** facilitates timely discharge.
19. **Education and secondary prevention:** teach disease, aetiology, risk factors, management and prognosis; individual or group formats; **discussion groups allow mutual support**. **Psychosocial support:** good listening, regular reassurance, **honest positive feedback without false expectations**, the ward **"therapeutic community"**, and **self-help groups** after discharge.
20. **Discharge planning and follow-up:** discharge is a **transition, not the conclusion**, of rehabilitation. **Evaluation:** rehabilitation is **resource-intensive**, so at minimum **entry versus discharge data** must be collected using standardised measures.
21. **Settings:** rehabilitation hospitals, designated units in general hospitals, general wards, nursing homes, **day hospitals**, community day centres, outpatient centres and the patient's own home. **Stroke units improve survival and functional outcome**; evidence for ortho-geriatric units is described as less convincing. **Intermediate care** = intensive, short-term, community- or home-based rehabilitation that prevents admission, enables early discharge and maximises independent living.
22. **Emerging technologies:** wearable **sensors** giving objective quantitative data, **virtual reality platforms**, and **video-capture technology** — overcoming the subjectivity and inter-observer variability of existing scales.

Latest Updates (2023–Present)

1. WHO / policy — the biggest change

The **first-ever resolution dedicated to rehabilitation, WHA76.6 "Strengthening rehabilitation in health systems", was adopted by all 194 Member States at the 76th World Health Assembly in May 2023**. As much as **half of those needing rehabilitation do not receive it**, especially in LMICs. It calls for **expanding and integrating rehabilitation into health systems as part of Universal Health Coverage**, stressing **primary care** and **emergency preparedness and response**, and names the **critical shortage of rehabilitation workforce**. WHO will report progress in **2026, 2028 and 2030**. The **3rd Global Rehabilitation 2030 meeting (July 2023)** launched the **World Rehabilitation Alliance**.

2. Efficacy now proven for hard outcomes

A **2024 systematic review and meta-analysis (BMC Medicine)** of RCTs in adults ≥65 years found that **geriatric rehabilitation in inpatient and day-hospital settings reduced mortality, long-term care home admission and functional impairment**, with **no difference in length of stay, mood or quality of life**.

3. End of "no rehabilitation potential" as a rationing tool

The **British Geriatrics Society 2024 report "Reablement, Rehabilitation and Recovery — everyone's business"** argues that rehabilitation **reduces hospital admissions, length of stay and carer burden while improving function**, should be **a component of every service caring for older people**, and that **rationing access on the dubious basis of "no rehabilitation potential" must end** (Age and Ageing, 2025).

4. Prehabilitation — rehabilitation moved *before* the insult

The **PREHAB randomised clinical trial (JAMA Surgery, 2023)** showed multimodal prehabilitation reduced postoperative complications and enhanced functional capacity after colorectal cancer surgery. A **2025 JAGS meta-analysis** confirmed that multimodal prehabilitation before colorectal cancer surgery **shortens hospital stay, lowers complication rates and speeds bowel recovery, particularly in older populations**. A **2025 meta-analysis in frail older adults** used the **6-minute walk test and length of stay** as endpoints, noting that frailty reduces resilience to surgical stress and the **preoperative window is a better opportunity than the postoperative period**. **Psychological prehabilitation** (mainly CBT) reduced length of stay, pain, anxiety and depression (Annals of Surgery, 2025).

5. Technology and telerehabilitation — from "emerging" to mainstream

A **network meta-analysis of 52 RCTs (npj Digital Medicine, 2023)** compared **VR exergaming, serious gaming, wearables and telerehabilitation** for balance and functional mobility in adults ≥60. A growing body of evidence supports **eHealth improving outcomes in geriatric rehabilitation** (Age and Ageing, 2025). However, a **2025 JMIR Aging rapid review** flags **ethics and equity concerns — internet access, technology adoption and digital literacy**.

6. Related guideline updates

A **clinical practice guideline on Comprehensive Geriatric Assessment in hospital** (issued May 2024, published Dtsch Arztebl Int 2025) provides 12 evidence-based and 8 consensus-based recommendations, including a **screening-CGA algorithm to determine operability and indication for surgery**. **Guidelines for Rehabilitation Interventions in Older Adults with Sarcopenia (2025)** address sarcopenia as a driver of falls, disability and mortality — **resistance training plus protein supplementation** remains the core intervention.

7. Indian context

NPHCE delivers promotional, preventive, curative and **rehabilitative** services through **19 Regional Geriatric Centres** and **2 National Centres for Ageing (AIIMS New Delhi; Madras Medical College, Chennai)**, with coverage extended to **713 districts**. **AB-PMJAY was extended in 2024 to all senior citizens aged ≥70 years**, giving **₹5 lakh per family per year free treatment irrespective of socioeconomic status**. **LASI wave-1 (2017–18, n = 31,477 aged ≥60): 7.4% had ADL dependency**; injurious falls 5.7%, impaired vision 13.7%, impaired hearing 9.6%, impaired cognition 9.3%, incontinence 4.0%; a higher number of geriatric and chronic conditions predicted ADL dependency.

Current Practice Update

Issue	Textbook Position	Current Evidence (2023–present)
Framework	ICF (2001) with impairment–activity–participation	ICF retained, now nested within the WHO Healthy Ageing / intrinsic capacity model and **WHA76.6**, making rehabilitation an **essential health service within UHC**
Evidence base	"Research is coming of age"; efficacy still being proven	**Proven** — 2024 meta-analysis shows reduced mortality, reduced institutionalisation, better function
Patient selection	Assess first to ensure the problem is "remediable"	Selection retained, but **"no rehabilitation potential" is no longer an acceptable reason to deny access** (BGS 2024)
Timing	Early intervention *after* the impairment	Extended **backwards to prehabilitation** — pre-operative exercise, nutrition and psychological support in frail surgical patients
Technology	"Emerging" — sensors, VR, video capture	**Mainstream** — telerehabilitation, exergaming and wearables have RCT/NMA evidence; **digital equity** is now the limiting issue
Ortho-geriatric units	Evidence "less convincing" than stroke units	**Superseded** — orthogeriatric co-management is standard of care in hip fracture pathways
Setting	Range of options; intermediate care emerging	Strong push toward **primary-care-integrated and home-based rehabilitation**, explicitly named in WHA76.6
Funding (India)	Not addressed	**AB-PMJAY ≥70 years (2024)** + NPHCE Regional Geriatric Centres provide delivery and financing

Exam One-Liners

- **Rehabilitation** = goal-oriented, time-limited process to restore optimum physical, mental and social function (UN definition).
- **ICF 2001** = Functioning/Disability + Contextual Factors; replaced ICIDH 1980.
- **Impairment → activity limitation → participation restriction** is the core chain.
- Greatest difference in elderly rehabilitation = **reduced physiological reserve**.
- **8 steps:** Assessment, Goals, Therapy, Aids, Education, Psychological support, Evaluation, Follow-up.
- **Barthel Index** = basic ADLs, has a **ceiling effect**; **FIM** = 18 items, 6 domains.
- **AMT** = 10 items; **GDS** = 30 items (15 short form); **PGC Morale Scale** = subjective morale.
- **Prosthesis** replaces a body part; **orthosis** is applied externally.
- **Intermediate care** = short-term intensive community rehab to prevent admission and enable early discharge.
- **Stroke units improve survival and function** — the strongest single evidence in geriatric rehabilitation.
- **WHA76.6 (May 2023)** = first-ever WHO resolution on rehabilitation; ~50% of global need unmet.

- **Prehabilitation** is the 2023–2025 paradigm shift — intervene before the surgical insult.
- Discharge is a **transition, not the end**, of the rehabilitation process.

Q2. Heading an interdisciplinary rehabilitation ward — team selection, job responsibilities, conflict management and care planning

High-Yield Points

A. Concept and Rationale

1. **Geriatric care implies team care.** No single discipline can address the impairment–activity–participation chain in a frail older person. Team care means **trusting other disciplines** with the tasks for which they are trained, while ensuring they **never work in isolation**.
2. **Terminology:** *Multidisciplinary* = each discipline assesses and treats in parallel. **Interdisciplinary** = disciplines assess separately but **plan and set goals jointly at a common team conference**, with shared responsibility for a single care plan.
3. **The ward runs on the 8-step process:** Assessment → **Goal setting** → Therapy → Aids and adaptations → Education → Psychological support → Evaluation → Follow-up. The head of the ward must ensure every step has a **named owner and a target date**.

B. Team Selection and Composition

4. **Minimum core team:** physician (geriatrician), nurses, physiotherapist, occupational therapist and social worker. **Optimal team** adds speech and language therapist, clinical psychologist, dietician, pharmacist, prosthetist/orthotist, podiatrist and home-care coordinator. **Dedicated administrative support is essential** and often forgotten.
5. **Special geriatric expertise among team members is crucial** — a therapist trained in general adult practice is not automatically competent in frailty, delirium and multimorbidity. Select on **interest, aptitude and training in ageing**.
6. **Optimum working team size is 4–7 members**, united by a **common sense of direction and purpose**. Every member must be able to state **their specific and their general contribution**. A **flexible working style crossing conventional professional boundaries** gives the greatest job satisfaction and the most effective patient care.
7. **Staffing must be structurally supported:** dedicated units require **additional therapist establishment** and **environmental adaptations** (handrails, non-slip flooring, orientation clocks and calendars, safe walking circuits).

C. Job Responsibilities

The core rule: doctors manage impairment; remedial therapists manage activity restriction; social workers manage participation restriction; nurses have a holistic brief influencing both.

Member	Core responsibility on a rehabilitation ward
Geriatrician (team head)	Diagnose and treat impairment and comorbidity; **deprescribe**; anticipate and prevent complications; medical fitness for therapy; chair the team conference; governance and audit
Rehabilitation nurse	**24-hour rehabilitation nursing** — reinforce therapy goals between sessions; skin, continence, nutrition, delirium prevention; **encourage the patient to *do* rather than be *done for***
Physiotherapist	Relieve pain; maintain and improve **joint range of motion, strength, endurance, coordination**; **gait and balance training**; assess need for and teach use of walking aids and wheelchairs
Occupational therapist	Assess and enhance **basic ADLs and IADLs**; **environmental assessment and home visit**; prescribe **assistive devices** and home modifications
Speech and language therapist	**Communication disorders and dysphagia**; swallow assessment; diet-texture recommendations
Clinical psychologist	Mood, motivation, cognition; adjustment and grief reaction; behavioural interventions
Dietician	Nutritional assessment, oral nutritional supplementation, protein adequacy for muscle rebuilding
Social worker	**Participation restriction**; family and carer assessment; finances, benefits, placement, community linkage
Pharmacist	Medication reconciliation, **anticholinergic burden**, falls-risk drugs, adherence aids
Prosthetist / orthotist	Design, construction and fitting of **prostheses** (replace a body part) and **orthoses** (support, improve function, restrict or promote movement)
Patient and family carer	**Full team members** — they set the goals that matter and continue therapy after discharge

D. Team Process and Leadership

9. **Effective teams do not arise spontaneously.** They evolve through **forming → storming → norming → performing**, and reaching the performing stage needs **commitment, skill and time**. The **storming phase is normal and expected**, not a sign of failure.
10. **The interdisciplinary team conference is the single most critical structure.** Held **after each member has completed their individual assessment**, it brings all perspectives together, **generates new ideas, sets priorities, disseminates the full assessment, and avoids duplication or incongruity.** Most successful trials of geriatric assessment included such a conference.
11. **The team leader must strike a balance** between **over-structuring** ward activity and allowing individuals to **function totally independently in a non-hierarchical vacuum**. Both extremes damage care.
12. **Efficient team care does not mean endless meetings. Co-location** allows frequent informal exchange, supplemented by **regular formal meetings**. What the team needs is a **common language and a common playbook**.

E. Care Planning

13. **Five elements for every problem in the care plan:** (i) the **problem**, (ii) the **goal**, (iii) the **approach/intervention**, (iv) the **target date**, (v) the **discipline responsible**.
14. **Timing:** initial assessment within days of admission; **formal care-planning meeting within 1–2 weeks**; therapy review **daily to weekly**; full care plan review **at least every 3 months** or on significant change. Reassessment at least **weekly** during active rehabilitation.
15. **Goal-setting rules: realistic**, based on **premorbid function**, **agreed by the patient and every team member**, split into **short-term** and **long-term** goals with **flexible, regularly reviewed time frames**. Set goals that are **ambitious but achievable**.
16. **Not every patient can improve.** For those who cannot, the plan must shift explicitly to **maintenance of function and prevention of further decline**. **Small functional gains still improve the chance of discharge home and quality of life.**
17. **WHO ICOPE 5-step pathway:** screen → detailed person-centred assessment → set goals and build a personalised care plan with the older person, caregiver and team → implement and monitor with specialist referral → enlist community and support the caregiver.
18. **Documentation: SOAP format** for progress notes; a **face sheet** carrying the essential database (diagnoses, drugs, contacts, treatment-intensity preferences) that travels with the patient; and **flow sheets** with serial functional scores (**Barthel Index, FIM**).

F. Conflict Management

19. **The commonest source of conflict is goal divergence** — *"a programme can be seriously compromised if key people differ on what each is trying to achieve."* The primary preventive strategy is **explicit, documented, jointly agreed goals** with the patient present. Other recurring sources: **role blurring**, **discharge-timing pressure**, **family-versus-patient wishes**, and **resource competition** for therapy slots.

> **Textbook-derived conflict prevention on the ward**
>
> - Ensure every member knows **what the other professions can do and when to call on their skills** — ignorance of others' scope drives most territorial disputes.
> - Use the **team conference to eliminate duplication and incongruity** before it becomes friction.
> - Accept **role flexibility across boundaries** as a strength, but keep **core accountability clearly assigned**.
> - Involve the **patient and carer** at some stage of every plan, preserving the **principle of choice** — this pre-empts family conflict.
> - In ethical disagreements, **the older person's own wishes are paramount**. **Avoid paternalism and the temptation to infantilise the frail patient.**
> - Build **trust deliberately** — like a sports team, healthcare teams must practise together.

G. Evaluation and Governance

20. **Rehabilitation is resource-intensive and must prove its effectiveness.** At minimum, collect **standardised entry and discharge data** on every patient.
21. **Run a team-based QAPI cycle:** each discipline reports its data → team reviews **quality indicators** → sets targets → develops a plan and a measurement method → **assigns an individual or subcommittee** → progress report at the next meeting. Use **run charts,**

fishbone diagrams and root-cause analysis. Ward indicators: **falls with injury, pressure ulcers, restraint use, Barthel/FIM gain, discharge-to-home rate, length of stay, 30-day readmission.**

22. **Geriatricians are ideally placed to lead** because **interdisciplinary management and teamwork are at the core of geriatric training**, and geriatricians are comfortable **entrusting responsibility to team members and sharing the problem-solving process**.

Latest Updates (2023–Present)

1. Chairing the team meeting is an evidence-based skill

A **2025 observational study of interprofessional collaboration during multidisciplinary team meetings in geriatric rehabilitation (BMC Geriatrics)** found collaboration **improves when the meeting is led by a designated person with leadership skills** — the ability to organise and to build strong team relationships. The **chair is accountable for coordination and adherence**, and **involving complementary participants — professionals, patients and informal caregivers — shifts the discussion from monodisciplinary or multidisciplinary objectives to common interprofessional goals.**

2. Psychological safety is the current framework for team climate

A **2025 Frontiers in Medicine** paper applies **Clark's four stages — inclusion safety, learner safety, contributor safety and challenger safety** — and identifies **well-defined roles and responsibilities as a direct driver of psychological safety**. **AHRQ's TeamSTEPPS** was developed to overcome the difficulty of speaking up in a hierarchical system, **empowering every team member to voice observations and concerns**.

3. Named conflict-management tools for clinical teams

AHRQ TeamSTEPPS (reviewed 2023) provides: **formative feedback**, **CUS** (when conflict places the patient at risk), the **Two-Challenge Rule** (differing information), and the **DESC script**.
DESC = **D**escribe the specific situation → **E**xpress your concern → **S**uggest alternatives → state **C**onsequences in terms of patient/team goals.
CUS = "I am **C**oncerned… I am **U**ncomfortable… this is a **S**afety issue."

4. Conflict-management styles in practice

A **2024 systematic review in Nursing Reports** (22 studies, 2014–2024) found **accommodation and collaboration/integration are the most common styles among nurses, with compromising also frequent**, and recognises **five styles: dominating, integrating, compromising, avoiding and obliging**. **Avoidance was most common among experienced and managerial nurses**, while **collaboration was favoured by more highly educated nurses**; younger nurses tended toward compromising and accommodating, older nurses toward collaboration. **Competing was the least prevalent**. **Proactive strategies are more effective at preventing conflict than reactive ones**, and tailored conflict-management training is recommended.

5. Interdisciplinary care written into guidelines, and the case for the ward

The **2024 VA/DoD Clinical Practice Guideline for Stroke Rehabilitation** is written with **explicit emphasis on an interdisciplinary care approach**. The **2024 BMC Medicine meta-analysis** showed geriatric rehabilitation **reduced mortality, care-home admission and functional impairment**; the **BGS 2024 report** argues rehabilitation should be a component of every service for older people and that denying access on the basis of "no rehabilitation potential" should end. A **2023 BMC Geriatrics focus-group study** identified **involvement of the person with dementia in goal setting** and the **mediating role of team members** as the key challenges, but found the refined model achieved **well-functioning, competence-enhancing and time-saving teamwork**.

6. Indian context

NPHCE delivers rehabilitative geriatric services through **19 Regional Geriatric Centres** and **2 National Centres for Ageing**. **AB-PMJAY extended to all citizens aged ≥70 years** (₹5 lakh per family per year) creates a funding pathway for post-acute rehabilitation. Practical constraints in Indian units: **absent or shared therapist posts, no dedicated clinical psychologist or prosthetist, and heavy dependence on family attendants** — so **task-sharing and structured family training** must be built into the care plan from day one.

Current Practice Update

Issue	Textbook Position	Current Evidence (2023–present)
Team meeting leadership	Leader is "usually the senior physician"; balance structure vs autonomy	**A designated, trained chair with organisational and relational skills** is the key determinant of collaboration — can be any discipline
Team development	Forming–storming–norming–performing	Retained, supplemented by **psychological safety (Clark's 4 stages)** as the measurable team climate outcome
Conflict management	Addressed indirectly — align goals, avoid duplication, build trust	**Named, teachable tools**: TeamSTEPPS **DESC, CUS, Two-Challenge Rule**; **collaboration** preferred, **avoidance** commonest but least effective
Patient in the team	Involve patient "at some stage"	Patient and informal caregiver are **participants in the team meeting itself**, converting disciplinary objectives into **common interprofessional goals**
Patient selection	Assess first to confirm the problem is remediable	**"No rehabilitation potential" is no longer an acceptable exclusion criterion** (BGS 2024)
Evidence for the ward	Stroke units proven; ortho-geriatric units less convincing	**Geriatric rehabilitation proven to reduce mortality and institutionalisation**; orthogeriatric co-management is standard care
Team size	4–7 members	Unchanged, but **pharmacist and dietician are now near-core**, given deprescribing and sarcopenia evidence

Exam One-Liners

- **Multidisciplinary** = parallel working; **interdisciplinary** = shared goals and one common care plan.
- **Doctors treat impairment, therapists treat activity restriction, social workers treat participation restriction, nurses do all three.**
- **Optimum team size = 4–7**, with a shared sense of direction.
- **Team stages:** forming → storming → norming → performing; storming is normal.
- The **interdisciplinary team conference** sets priorities, shares assessments and **prevents duplication and incongruity**.
- **Every care-plan entry needs:** problem, goal, approach, **target date**, responsible discipline.
- **Care plan timing:** meeting within **1–2 weeks**, review **3-monthly** or on change; therapy review **weekly**.
- **Commonest cause of team conflict = divergent goals.**
- **DESC** = Describe, Express, Suggest, Consequences; **CUS** = Concerned, Uncomfortable, Safety issue.
- **Five conflict styles:** dominating, integrating, compromising, avoiding, obliging. **Collaboration is best; avoidance is commonest.**
- **QAPI cycle:** report data → set targets → plan → assign owner → re-measure at next meeting.
- Effective team care needs **a common language and a common playbook** — it never happens spontaneously.

Q3. Role of the occupational therapist on the team caring for the older person, on the ward and at discharge

High-Yield Points

A. Conceptual Basis

1. **Occupational therapy (OT) views humans as "occupational beings" — who we are is defined by what we do. Occupations** are the ordinary and extraordinary things people do daily that occupy time, ensure survival, maintain well-being, nurture others, contribute to society and carry cultural meaning.
2. **The core aim of OT in old age is to enable the elder to keep doing the activities that are personally meaningful to them**, thereby preserving **quality of life** — not merely to restore muscle power.
3. **In ICF terms, the doctor works on impairment while the OT works on the Activity and Participation boxes** — OT converts a treated disease into a life that can actually be lived.
4. **The guiding model is PEOP (Person–Environment–Occupation–Performance/ Participation)**, complementary to the WHO ICF:
   Elder quality of life = f (Personal attributes × Environmental factors × Occupational performance/participation)
   - **Person (intrinsic):** physiological, motor, **cognitive**, neurobehavioural, psychological and spiritual capacity; plus **motivation, roles, habits and routines**.
   - **Environment (extrinsic):** social support, economic systems, **culture and values**, technology, the **built environment** and the natural environment.
5. **OT services span the whole continuum: primary** (health promotion, home-safety and fall prevention, driver screening), **secondary** (arthritis, stroke, dementia), **tertiary** (rehabilitation, long-term care, palliative and hospice care). Delivered at **three levels**: to individuals and families, as **consultation to community organisations**, and as **consultation to government agencies**.

B. The OT Process

6. **The process:** patient identification → **occupational profile** → **occupational performance assessment** → identification of needs, problems and priorities → **collaborative goal setting** → occupation-based intervention → ongoing assessment and refinement → outcomes met → **final/discharge assessment**. The **Occupational Therapy Practice Framework (OTPF)** is the guiding protocol.
7. **The occupational profile is the first and most distinctive step.** It records the person's **occupational history, current patterns of daily living, interests, values and needs**, then their **own priorities and concerns**. The approach is **top-down and client-centred** — the elder names the activities that matter, and the plan is built backwards from them.
8. **Occupational performance assessment** uses **valid, standardised instruments**, but crucially **the therapist observes the actual performance of the selected activity** — skilled observation covering **affect, cognitive/executive function and motor function**

together. **Assessment is ideally done in the person's own environment, since performance in an unfamiliar setting can be quite different.**

9. **The third focus is identifying barriers — biological, psychological and social —** between the older person and their desired activities.

C. Role on the Ward

10. **Baseline and premorbid functional assessment.** Establish competence in **basic ADLs** (feeding, dressing, bathing, grooming, toileting, transfers) and **IADLs** (cooking, cleaning, shopping, telephone, laundry, medication and money management, transport), comparing **premorbid with current** status.
11. **Core ward objectives (Kane):** restore, maintain and improve **independent function**; evaluate and improve **sensory and perceptual-motor function**; evaluate and improve **ADL performance**; **fabricate and fit upper-limb splints**; improve **coping and problem-solving skills**; improve **use of leisure time**.
12. **Ward modalities:** assessment of **bed mobility, transfers and wheelchair propulsion**; **ADL practice in actual or simulated environments** (a mock kitchen or bathroom on the ward); **task-oriented activities** such as crafts and projects; and **teaching the use of assistive and safety devices**.
13. **Prescribing and training in aids is the OT's specific expertise.** Common items: **long-handled reacher, sock aid, built-up cutlery and cooking utensils, dressing stick, raised toilet seat with rails, shower chair/tall stool, handheld shower, grab bars and rails. Devices are recommended only if they improve participation in an activity the elder or family has identified as important. A wrongly chosen aid fosters dependence rather than independence** — yet many with real need never obtain even basic aids.
14. **Disease-specific ward work:** reinforcing **hip precautions** after arthroplasty; **upper-limb positioning and splinting** in hemiplegia to prevent contracture and subluxation; **task simplification, sequencing cues and environmental orientation** in dementia; **energy conservation and joint protection** in arthritis, COPD and heart failure; **transfer devices** (trapeze, transfer pole, raised quarter-length siderail) and **bed/toilet height matched to lower-leg length** as part of **restraint reduction**.
15. **Education and secondary prevention: body mechanics, joint protection, energy conservation, activity and exercise guidance, sleep hygiene**; psychosocially — **role adjustment, balanced purposeful activity, time management, mastery and self-esteem, and socialisation skills**.
16. **Team role:** the OT feeds directly into the **interdisciplinary team conference**, translating medical impairment into functional terms. **The OT's functional verdict frequently decides the discharge destination.**

D. Role at Discharge

17. **The pre-discharge home visit is the OT's signature intervention. Early OT input, usually including a home visit, ensures that modifications are appropriate and timed to facilitate hospital discharge** — done late, it becomes a cause of delayed discharge rather than a solution.
18. **Home environmental assessment and modification:** improved **lighting in halls and stairways**, **grab bars in the bathroom**, **removal of loose rugs and clutter**, **ramps and railings**, non-slip surfaces, covers on electrical sockets and locks on cupboards where cognition is impaired. **Interventions that assess and modify home safety features**

reduce falls, and are more effective when carried out specifically by an occupational therapist.

19. **Practical discharge preparation:** review **all ADL and IADL implications immediately before discharge**; arrange equipment to be in place **before the patient goes home**; advise on **furniture height for sitting, rising, eating and leisure** (including chair-leg extensions); train the **family carer**; and arrange a **separate community OT visit after discharge** for tasks assessable only at home, such as kitchen work.
20. **Post-discharge and community role:** graded return to **IADLs and valued roles**, **carer education and coping support**, adaptation of **technology** (smartphone, appliances, voice-activation software), **driving assessment** (OT and neuropsychology form the second tier, after clinical assessment and before on-road testing), and helping the person **come to terms with driving cessation by focusing on preserved areas of achievement** while the social worker arranges alternative transport.
21. **Discharge from OT itself is a defined step.** Services are discontinued when **targeted outcomes are met**, *or* when the elder **cannot achieve them despite refinement of the plan**. A **final assessment** is performed, and outcome data are used to plan future therapy, discontinue services and **evaluate the service programme**.
22. **The commonest cause of failed OT discharge planning is well-meaning over-help.** If caregivers bathe and dress a patient who has independence goals, **those goals will not be achieved** — "helping" produces **deconditioning, dependency, contractures and the sequelae of immobility**. A **restorative philosophy of care must continue at home**.
23. **Fundamental principle:** OT supports the older person's **autonomy in setting priorities and making decisions**, and preserves their **mastery and control over their own environment and lifestyle**.

Latest Updates (2023–Present)

1. Home hazard reduction — high-certainty evidence and OT's strongest card

The **2023 Cochrane review (Clemson et al., CD013258)** included **22 studies from 10 countries with 8463 community-dwelling older people** (mean age 78, 65% women). **Home fall-hazard interventions reduce the overall rate of falls by 26% (RaR 0.74, 95% CI 0.61–0.91; 12 studies, 5293 participants; moderate-certainty evidence)**, and are **substantially more effective in people selected for higher fall risk, with a 38% reduction (RaR 0.62, 95% CI 0.56–0.70)**. In contrast, **assistive devices alone (eyeglasses, specialised footwear, bed alarms) and education alone do not reduce falls.**

2. NICE NG249 (April 2025) — the OT is named in guideline text

NICE recommends **offering a home hazard assessment and intervention using a validated tool**, and states: **"consider having the home hazard assessment and intervention… carried out by an occupational therapist."** If not an OT, it may be done by an **appropriately trained healthcare professional, or a trained therapy assistant or technician under supervision**. The rationale is explicit: **greater clinical benefit was seen when the intervention was delivered by an occupational therapist**, and **health economic modelling found OT delivery both less costly and more effective** than delivery by an assistant or technician. Benefit was greatest in people who had **fallen at**

least once in the previous year. NG249 also **advises against standalone falls risk-prediction tools**.

3. Real-world implementation, ED extension and early supported discharge

A **pilot implementation study of OT-led environmental assessment and modification** in a regional Australian health service (2019–2022; 279 chart audits of people ≥65 at high fall risk) found **71% had sustained ≥1 fall and 52% ≥2 falls** in the preceding year; the intervention was **delivered per protocol, embedded and sustained**, with **OT precision in selecting recipients improving over time**; a **national implementation study began in 2025**. A **Geriatric ED Guidelines 2.0 systematic review (Academic Emergency Medicine, 2025)** appraised **PT/OT evaluations introduced into ED care for fall-risk evaluation and prevention** — promising but not yet definitive. A **2024 BMC Geriatrics systematic review and meta-analysis** examined **early supported discharge** after orthopaedic surgery, where OT is the pivotal discipline.

4. Policy and Indian context

WHA76.6 (May 2023) calls for expanding and integrating rehabilitation into health systems within **Universal Health Coverage**, with emphasis on **primary care**; the **World Federation of Occupational Therapists (WFOT)**, with ReLAB-HS, has issued guidance translating the resolution into advocacy for **inclusion of occupational therapy services within UHC**. In India, **Rashtriya Vayoshri Yojana** provides **free assisted-living devices** to BPL senior citizens (monthly income ≤ ₹15,000) with **doorstep delivery for those aged ≥80**, **ALIMCO** as sole implementing agency; at least **30% of beneficiaries in each district must be women**. The OT must **prescribe within what RVY, ALIMCO and the family can actually obtain**, and must **train the family attendant**, since one-to-one community OT follow-up is rarely available.

Current Practice Update

Issue	Textbook Position	Current Evidence (2023–present)
Home hazard assessment	Home modification promotes activity and participation; more effective when done by an OT	**Quantified and guideline-mandated — 26% fall reduction overall, 38% in high-risk people** (Cochrane 2023); NICE NG249 names the OT
Who should do it	Advice on aids "best left to OTs or others with expertise"	**Health-economic modelling shows OT delivery is both cheaper and more effective** than assistant/technician delivery
Assistive devices alone	Aids reduce activity and participation restriction	**Devices or education alone do not reduce falls** — they work only inside a targeted, individualised package
Targeting	Assess before entry to ensure the problem is remediable	**Target by fall history** — benefit is concentrated in those who fell in the last year
Risk prediction tools	Not addressed	**NICE 2025 advises against standalone falls risk-prediction tools** — assess and act, don't score
Setting of assessment	Ideally in the person's own environment	Reinforced, plus **remote/video and 3D home assessment** emerging where OT numbers limit home visits
Timing	Early OT input including a home visit facilitates discharge	Extended into **ED-based OT evaluation** and **early supported discharge** models
Scope	Individual, community and agency levels	**Named in global policy** — WHA76.6 and WFOT advocacy place OT inside UHC packages

Exam One-Liners

- **OT enables occupation; physiotherapy restores movement.** OT owns the **Activity and Participation** domains of the ICF.
- **PEOP model:** Person–Environment–Occupation–Performance/Participation; complementary to the WHO ICF.
- **Occupational profile** = the client-centred, **top-down** first step.
- **Assess by observation, and assess at home** — performance in an unfamiliar environment differs.
- **OT ward objectives:** independent function, sensory-perceptual motor function, ADLs, **upper-limb splinting**, coping/problem-solving, leisure.
- **Classic OT aids:** long-handled reacher, **sock aid**, built-up cutlery, dressing stick, raised toilet seat with rails, shower chair, grab bars.
- **A wrongly prescribed aid fosters dependence, not independence.**
- **The pre-discharge home visit must be early**, or it becomes a cause of delayed discharge.
- **Home fall-hazard reduction: 26% fewer falls overall, 38% fewer in high-risk older people** (Cochrane 2023).
- **NICE NG249 (2025):** offer home hazard assessment with a validated tool; **consider having an OT do it** — cheaper and more effective.

- **Devices alone and education alone do not prevent falls.**
- **OT is the second tier of driving assessment**, between clinical evaluation and the on-road test.
- **Over-helping at home undoes rehabilitation** — deconditioning, dependency, contractures.
- **OT discharge criteria:** goals met, **or** goals unachievable despite refinement — then final assessment and discontinuation.
- **India: Rashtriya Vayoshri Yojana** supplies free assistive devices to BPL elders through **ALIMCO**.

Q4. Stroke rehabilitation — factors to consider before initiating long-term rehabilitation

High-Yield Points

A. Principle of Selection

1. **Every stroke patient deserves an assessment and consideration for intensive rehabilitation.** Selection is about *matching the programme to the person*, never about denying entry.
2. **Formal assessment before entry is essential** — to confirm the problems are **remediable** and to determine optimal management. **It is unfair to subject a person who cannot benefit to a demanding programme, raise false expectations and waste resources; equally, those who can benefit even to a limited extent must not be denied access.**

B. Factors Determining Prognosis Before Starting Long-Term Rehabilitation

The five classical prognostic factors (Kane):

Factor	What to assess
1. Availability and implementation of a sound programme	Is a properly resourced multidisciplinary team, unit and follow-up service actually available? A good candidate with no programme gains nothing
2. Mentation	Cognition, comprehension, **memory and learning capacity** — rehabilitation is **motor relearning**, so a patient who cannot retain instruction cannot progress
3. Motivation	The patient's drive to engage; the strongest modifiable predictor of outcome
4. Prognosis for neurological return	Site, size and type of lesion; severity of deficit; time since onset
5. Vigour	Physiological reserve, stamina, cardiorespiratory fitness, nutrition — the capacity to *tolerate* daily therapy

4. **Mentation in detail:** assess **conscious level, orientation, attention, memory, comprehension and executive function**, using tools such as the **Abbreviated Mental Test**. Look for **pre-existing dementia, post-stroke delirium and post-stroke dementia** (roughly **a quarter of stroke survivors are demented after stroke, of whom about 10% were already demented before the first stroke**). **Aphasia must be distinguished from cognitive failure** — a patient with pure expressive dysphasia may have intact learning capacity.
5. **Motivation in detail:** use **Kemp's model** — motivation is driven by the patient's **wants, beliefs and rewards**, opposed by the **cost in pain and effort**. Modify each element: set attainable goals, correct false beliefs, create rewards, reduce the physical and emotional cost. At one extreme are patients who set ambitious goals; at the other, those who **surrender autonomy and adopt the "sick role"**.
6. **Screen for depression before and during rehabilitation. Depression is common after stroke, persists despite participation in a rehabilitation programme, and worsens**

prognosis — an untreated depression will be misread as “poor motivation” and wrongly used to exclude the patient. Use the **Geriatric Depression Scale**; treat before concluding the patient is unsuitable.

7. **Prognosis for neurological return depends heavily on timing. Most neurological recovery occurs in the first month after stroke; by the end of the third month, little if any further return can be expected.** Rehabilitation must therefore be **started early**.
8. **Not all deficits are equally disabling. Motor loss is often the least disabling. Sensory and perceptual loss, aphasia, loss of balance, hemispatial (hemicorporeal) neglect, hemianopia and cognitive impairment cause more severe and often untreatable disability.**
9. **Vigour and reserve:** assess **premorbid functional status, cardiorespiratory fitness, nutrition, anaemia, sarcopenia, pain and continence**. The elderly patient’s core problem is **lack of physiological reserve**. **Coexisting impairments** (osteoarthritis, Parkinson’s disease, visual and hearing loss, prior stroke) each **multiply** disability.
10. **Medical stability and complication risk must be settled first.** Confirm **swallowing safety, control of blood pressure and glycaemia, absence of active sepsis, adequate cardiac status, control of seizures**. **Complications to prevent actively: pressure sores, contractures, phlebitis, pulmonary embolism, aspiration pneumonia and faecal impaction**.
11. **Social and environmental factors decide participation.** *Intrinsic:* attitude, personality, **ability to adjust**, cultural factors. *Extrinsic:* **financial resources, housing, availability of a carer, social supports**. **Elderly women living alone are especially disadvantaged.**
12. **Set the goals before starting.** Goals must be **realistic, agreed by the patient and every team member, ambitious but achievable**, split into **short-term** and **long-term** goals with **flexible, regularly reviewed time frames**. Goal divergence between team members is the commonest cause of a failed programme.
13. **Decide the discharge destination and the endpoint at the start** — home with family, home with supports, day hospital, or long-term care. Assessment must address **both “the problem in the individual” and “the individual with the problem”**.

C. Content of Stroke Rehabilitation

14. **Acute phase measures:** change position **at least 2-hourly**; **position joints to prevent contractures**; **position to prevent aspiration pneumonia**; perform **range-of-motion exercises**. **A flaccid hemiplegic limb is at risk of shoulder subluxation** — proper handling and limb positioning in the immediate post-stroke period minimises this risk.
15. **Later phase measures: ADL training, ambulation training, functional activities for the affected side, muscle re-education exercises, perceptual training, and training in transfer technique.** Treatment aims both to **re-educate affected muscles** and to **enhance remaining capacity in unaffected areas**.
16. **Specific evidence-based techniques: constraint-induced movement therapy** produces significant and clinically relevant improvement in arm motor function **in patients 3–9 months after a first stroke**; **treadmill training** repeatedly improves **gait and balance**; **robot-assisted motor training** and **brain–computer interfaces** show early positive results.
17. **Long-term: ongoing exercise and physical activity after the formal programme are critical** — they maintain function and quality of life **and help prevent further strokes**.

D. Setting and Endpoint

18. **Organised stroke unit care improves survival and functional outcomes** (Stroke Unit Trialists' Collaboration). **Patients cared for in stroke centres are more likely to be alive, independent and living at home one year after the stroke.**
19. **Stroke patients may do better in dedicated acute rehabilitation facilities than in skilled nursing facilities.** Whatever the setting, **the basic principles and the multidisciplinary team remain the same**.
20. **When the patient stops making progress despite intensive therapy, the goal shifts from restoration to coping.** Assess the need for **braces, orthoses and assistive devices** for both ambulation and ADLs, and switch to **maintenance of function and prevention of decline**.
21. **Even the most severely affected can gain something.** Complete functional recovery occurs in **less than half** of stroke patients, but **immobility and its complications can almost always be prevented or minimised**.
22. **Discharge is a transition, not the conclusion.** Follow-up is essential so that any new or worsening activity or participation restriction is detected and remedied.

Latest Updates (2023–Present)

1. NICE NG236 (18 October 2023) — the biggest change is *dose*

NICE's stroke rehabilitation guideline, which **replaces CG162**, recommends: **offer needs-based rehabilitation for at least 3 hours a day, on at least 5 days a week**, covering a range of multidisciplinary therapy including **physiotherapy, occupational therapy and speech and language therapy**. Once the person has left hospital, **care and rehabilitation should continue for as long as it continues to help them achieve their treatment goals**. **Early supported discharge should be offered to people who can move from bed to chair independently or with assistance**, provided a safe environment can be arranged, with **therapy continued at the same intensity and level of skilled support as in hospital**. The previous daily target of **40–45 minutes has been raised to 3 hours**.

2. NG236 rationale and the ESD evidence

The committee found that **early supported discharge reduced physical dependency and length of hospital stay**, and either improved or did not worsen health-related quality of life, with **no difference in mortality, carer quality of life, Caregiver Strain Index or readmission rates**, though **a clinically important increase in falls** was seen in a limited number of studies. Rehabilitation **should not be withdrawn after a fixed period**. NG236 also recommends **NMES as an option for shoulder pain**, and a **trial of NMES, FES or TENS for focal spasticity**.

3. VA/DoD 2024 and drugs for motor recovery

The **2024 VA/DoD Clinical Practice Guideline**, updating the 2019 version, provides recommendations for rehabilitation management after stroke with **explicit emphasis on an interdisciplinary care approach**. AHA/ASA guidance holds that the effectiveness of **fluoxetine or other SSRIs to enhance motor recovery is not well established**, the effectiveness of **levodopa is not well established**, and **dextroamphetamine or**

methylphenidate to facilitate motor recovery is not recommended. **NMES may be considered for shoulder pain**; a **formal fall-prevention programme during hospitalisation** is recommended, with **exercise plus balance training after discharge**.

4. Neuromodulation has entered clinical practice

The **2021 FDA approval of the Vivistim paired vagus nerve stimulation system** was the first regulator-approved VNS therapy for **upper-extremity motor recovery** after stroke. **Long-term follow-up shows sustained benefit at one year, with 66% of patients achieving clinically meaningful improvement.** Transcutaneous approaches remain investigational. Current evidence supports **implantable VNS paired with rehabilitation in carefully selected patients with chronic upper limb impairment**.

5. India — the ATTEND trial is the key cautionary result

India has only about 35 stroke units, mainly in urban centres, so most people have no access to specialised stroke care or conventional rehabilitation. The **ATTEND trial randomised 1250 patients within one month of stroke** to family-led, trained caregiver-delivered, home-based rehabilitation across **14 Indian hospitals**. It **more than doubled inpatient rehabilitation time (5 hours vs 2 hours)** and added **30 minutes daily for one month after discharge — yet produced no improvement in modified Rankin Scale outcome.**

Exam interpretation: *quantity of therapy alone is not enough* — the **skill and specificity of the therapy delivered** matters, which is why NG236 specifies “multidisciplinary therapy”, not simply “3 hours of activity”.

6. Delivering the dose is the current bottleneck

The **first six months after stroke is the optimum window for neuroplasticity**, and exercise must be **repetitive, task-specific and high-dose**. However, delivering three hours a day in community settings is difficult, and **around 40% of stroke survivors are discharged home without any further rehabilitation**. **Telerehabilitation is recommended in the new guidance** as one way to bridge this gap.

Current Practice Update

Issue	Textbook Position	Current Evidence (2023–present)
Therapy dose	Not quantified; "early and multidisciplinary"	**NICE NG236 (2023): at least 3 hours/day on at least 5 days/week** — up from 45 minutes
Duration	Most neurological return by 1 month; little after 3 months	Neurological timeline holds, but **functional gains continue** — continue as long as goals are being met, not to a fixed cut-off
Patient selection	Assess to ensure the problem is remediable	Reinforced, but **"no rehabilitation potential" is no longer an acceptable exclusion** (BGS 2024)
Early supported discharge	Not specifically addressed	**Standard of care** — reduces dependency and length of stay; **watch for increased falls**
Drugs for motor recovery	Not emphasised	**Fluoxetine, levodopa: not established. Amphetamines/methylphenidate: not recommended**
Neuromodulation	Neuromuscular stimulation mentioned	**Paired VNS (Vivistim) is FDA-approved** for chronic upper limb deficit, with 1-year sustained benefit
CIMT	Effective 3–9 months after first stroke	Unchanged; remains a **selected-patient** therapy requiring some active wrist and finger extension
More therapy = better?	Implied	**ATTEND (India): doubling therapy hours via untrained family caregivers did not improve outcome** — dose must be *skilled* dose
Setting	Stroke units improve survival and function	Confirmed; extended to **stroke centres with better 1-year independence**, plus **telerehabilitation**

Exam One-Liners

- **Five prognostic factors: availability of a sound programme, Mentation, Motivation, Prognosis for neurological return, Vigour.**
- **Most neurological recovery occurs in the first month; little further return after 3 months.**
- **Motor loss is the least disabling deficit**; neglect, hemianopia, aphasia, sensory loss, balance loss and cognitive damage are far more disabling.
- **Depression is not poor motivation** — screen with GDS and treat before excluding a patient.
- **Kemp's model:** motivation = wants + beliefs + rewards − cost.
- **Acute phase:** 2-hourly turning, joint positioning, aspiration-preventing posture, range-of-motion exercises.
- **Later phase:** ADL training, ambulation, affected-side functional activity, muscle re-education, perceptual training, transfer technique.
- **Flaccid hemiplegia → shoulder subluxation**; prevented by correct handling and positioning from day one.
- **CIMT works 3–9 months after a first stroke** for arm motor function.

- **Stroke units improve survival and function** — the strongest evidence in rehabilitation.
- **NICE NG236 (2023): ≥3 hours of multidisciplinary therapy per day, ≥5 days per week.**
- **ATTEND (India): family-led rehabilitation doubled therapy time but did not improve mRS.**
- **When progress stops, shift the goal from restoration to coping** — braces, orthoses and assistive devices.
- **Complete functional recovery occurs in fewer than half**, but complications of immobility are almost always preventable.

Q5. Rehabilitation of an older adult with low vision

High-Yield Points

A. Definition and Concept

1. **Low vision** is visual impairment that **cannot be corrected by spectacles, contact lenses, medical treatment or surgery**, but where **useful residual vision remains** — irreversible vision loss with retained function, distinct from total blindness.
2. **Vision rehabilitation** enables a person with irreversible low vision to **maximise use of residual vision, learn compensatory skills, adapt the environment, and continue participating in daily and social life.** The goal is **function and quality of life, not visual acuity.**
3. **First rule: exclude and treat every reversible cause.** In India, **around 93% of blindness and 97% of visual impairment in people aged ≥50 years is avoidable** — chiefly **untreated cataract and uncorrected refractive error**. *Low vision rehabilitation is never a substitute for cataract surgery or a pair of spectacles.*

B. Epidemiology and Burden

4. **As many as 75% of older adults have significant visual or auditory dysfunction that they never report to their physician** — hence **active screening is essential**. These impairments **limit functional activity and lead to social isolation and depression**, and **correcting remediable conditions improves the ability to perform daily activities.**
5. **Indian data:** In the **National Blindness and Visual Impairment Survey (RAAB, 2015–19)**, among those aged ≥50 years, **blindness prevalence was 1.99%** and **more than one-quarter were visually impaired**. Causes of blindness: **cataract 66.2%, corneal opacity 8.2%, cataract surgical complications 7.2%, posterior segment disease 5.9%, glaucoma 5.5%. LASI wave-1** found **impaired vision in 13.7%** of Indians aged ≥60.

C. Age-Related Changes and Common Causes

6. **Normal ageing changes:** reduced **visual acuity** (choroid, pigment epithelium, retina, rod/cone function); **difficulty gazing upward and sustaining convergence**; **rising intraocular pressure**; **presbyopia** from nuclear sclerosis and ciliary muscle atrophy; **increased hyperopia or myopia**; **reduced tear secretion** (dry eye); **corneal endothelial degeneration**.

Condition	Functional deficit	Rehabilitation implication
Cataract	**Glare intolerance**, gradual acuity loss	Mostly reversible — **surgery when it interferes with activities**; interim: optimum eyeglasses, UV lenses, glare control
Open-angle glaucoma	**Peripheral field loss first**, central acuity preserved long	**Mobility and falls training** are the priority; magnification helps little in tunnel vision
Age-related macular degeneration	**Central acuity loss** — reading, driving and **recognising faces**	**Magnification and eccentric viewing training**; **total blindness does not occur** — peripheral vision is retained
Diabetic retinopathy	Central loss from macular oedema/ischaemia; patchy field loss	**Tight glycaemic and BP control** plus laser; allow for **neuropathy and reduced tactile substitution**

D. Consequences That Rehabilitation Must Target

8. **Visual impairment is an established risk factor for falls and fractures**, and **patients who have had cataract surgery have a lower risk of hip fracture.**
9. **Depression is a predictable consequence, not an incidental one.** Clinical depression occurs as a result of the fall in quality of life in AMD **and should be actively treated** — a core part of the rehabilitation plan.
10. **Other consequences:** loss of ADLs and IADLs (reading, cooking, medication identification, finances), **loss of driving**, social withdrawal, malnutrition, and **increased dependency and institutionalisation risk.**

E. Assessment

11. **Ophthalmological screening: visual acuity; lens and fundus; intraocular pressure; ophthalmoscopy; tonometry; visual fields;** and the simple functional test — **ability to read newspaper-sized print.**
12. **Functional assessment goes beyond acuity: near and distance acuity, contrast sensitivity, glare sensitivity, visual fields, reading ability, light and dark adaptation**, and what the person can actually do at home. Tools: **Lambeth Disability Screening Questionnaire** and the **Vision Function Questionnaire**.
13. **Assess vision as part of intrinsic capacity.** Under **WHO ICOPE**, **vision is a critical component of person-centred assessment**; **Step 2** assesses visual impairment and eye disease, and **Step 3 interventions include reading glasses, referral for comprehensive eye and vision care, vision rehabilitation for irreversible low vision, and home adaptation to prevent falls. Treating visual impairment enables maintaining family and social relationships, accessing information, moving safely and performing manual tasks.**
14. **Always screen for coexisting hearing loss — dual sensory impairment** multiplies disability, isolation and cognitive risk, and changes every rehabilitation strategy.

F. Components of Low Vision Rehabilitation

Category	Examples
Magnification	**Hand-held, stand and spectacle-mounted magnifiers; telescopes for distance; electronic/CCTV magnifiers**
Lighting	**Lighting intensifiers without glare**; task lighting behind and to the side; **night light to assist dark adaptation**
Glare control	**Tinted glasses**, filters, matt surfaces, avoiding polished floors
Contrast and size	**Large-print newspapers, books and magazines**; bold-tip pens; dark plate on light tablecloth
Non-optical/ tactile	Talking clocks and watches, talking BP and glucose meters, **tactile markers on appliances and medication boxes**, needle threaders, signature guides
Digital	Screen readers, smartphone magnification and text-to-speech, voice assistants

16. **Environmental modification and home safety.** Interventions that **assess and modify home safety features reduce falls, especially among older adults with severe visual impairment**, and are **more effective when carried out specifically by occupational therapists.** Measures: **improved lighting in halls and stairways, grab bars, removal of rugs and clutter, contrast marking of stair edges, covers on electrical sockets, locks on cupboards.**
17. **Optical correction pitfalls in falls prevention: routine vision screening alone did not reduce falls**; assessment and treatment of vision impairment **reduces falls only when coupled with an exercise programme**. **Replacing multifocal lenses with single-vision lenses for outdoor activity is effective**, and **cataract surgery reduces falls in older women — though second-eye surgery does not show the same effect.**
18. **Training and skills teaching: visual skills training** (**eccentric viewing**, steady fixation, scanning for field loss); **orientation and mobility training** (**long cane**, sighted-guide technique, safe road and stair negotiation); **ADL retraining** (labelling, pouring, cooking, grooming, **safe medication identification**); and **reading rehabilitation** — **prescribing a magnifier without training usually fails.**
19. **Psychological and social support: counselling, adjustment support, treatment of depression, self-help and peer groups**, family education, and linkage to **welfare benefits and disability certification**. Address the **grief reaction** to lost independence and lost driving.
20. **Review the drug chart — ophthalmic drops are systemically absorbed:**

Drop	Systemic effects relevant to the older person
β-blockers (timolol)	**Bradycardia, hypotension, syncope, heart failure**, bronchospasm; **confusion, depression, fatigue, hallucinations, memory impairment**; hyperkalaemia
Adrenergics (phenylephrine, epinephrine)	Extrasystoles, palpitation, **hypertension, myocardial infarction**, tremor, sweating
Cholinergics (pilocarpine)	Bronchospasm, salivation, nausea, vomiting, diarrhoea, abdominal pain
Anticholinergics (atropine)	**Ataxia, restlessness, confusion, hallucinations, aggression**, insomnia, photophobia, **urinary retention**

→ Assess other illnesses and medicines, **use the minimum effective dose, and monitor for systemic toxicity.**

21. **The team: ophthalmologist and optometrist**, **low vision therapist**, **occupational therapist**, **orientation and mobility instructor**, **nurse**, **clinical psychologist/social worker**, **dietician**, and the **family carer**. **Devices should be prescribed only if they improve participation in activities the person and family have identified as important.**
22. **Outcome measures:** vision-specific quality of life (**Impact of Vision Impairment, VFQ-25**), **reading speed**, **ADL/IADL independence**, **falls rate**, and **depression scores** — not visual acuity, which by definition will not improve.

Latest Updates (2023–Present)

1. WHA78.7 (May 2025) — the landmark policy change

At the **78th World Health Assembly (19–27 May 2025)**, Member States endorsed a landmark resolution on **“Primary prevention and integrated care for sensory impairments, including vision impairment and hearing loss, across the life course.”** It recognises **cost-effective interventions such as cataract surgery, assistive technologies (spectacles, hearing aids, implants) and rehabilitative services**, and promotes **integrated systematic vision and hearing screening and early intervention within health benefit packages and health information systems**. Over **2.2 billion people are affected by vision impairment and 1.5 billion by hearing loss**.

2. WHO’s framing of vision rehabilitation

WHO describes **vision rehabilitation as an essential component of eye care services** enabling people with **irreversible low vision** to **maximise functional abilities, enhance quality of life and participate fully in society**, and notes it is **very effective** for irreversible loss from **diabetic retinopathy, glaucoma, trauma and AMD**. Frameworks: the **World Report on Vision (2019)** and **WHA73.4 on Integrated People-Centred Eye Care (2020)**. WHO has issued a **Planning guide for integrated vision rehabilitation** for integrating services within national health systems.

3. SPECS 2030 — the refractive error target

Only about **one-third of people with vision impairment due to refractive error have spectacles that effectively correct it**. The **WHA74 (2021) global target** is to increase

effective coverage of refractive error by 40 percentage points by 2030, supported by **WHO SPECS 2030**. WHO and the **Universal Postal Union partnered in 2025** to improve access to eyeglasses.

4. What low vision rehabilitation achieves — the Cochrane position

The Cochrane review of **44 studies** (mostly older adults with AMD) covered **psychological therapies and group programmes, methods of enhancing vision, multidisciplinary rehabilitation programmes, and balance/home-safety programmes**. Key message: **low vision rehabilitation does not appear to have an important impact on health-related quality of life (very low-certainty evidence)**, but there is **low-certainty evidence that some interventions — particularly psychological therapies and methods of enhancing vision — may improve vision-related quality of life**.
*Exam interpretation: measure the **right** outcome — vision-specific QOL, not generic health QOL.*

5. Occupational therapy has the strongest single evidence base

Practice guidelines conclude that **strong evidence supports the role of occupational therapy for older adults with low vision**, recommending: **routine low vision rehabilitation for ADL and IADL impairments; multicomponent interventions for ADL/IADL performance and leisure and social participation; stand-based electronic magnification to enhance reading; visual skills training for central field impairment; and client-centred problem-solving training**, with mainstream technology considered case by case.

6. Telerehabilitation and mood

A **multicentre randomised controlled trial (Bittner et al., 2024)** randomised **61 patients with visual impairment** to **telerehabilitation or in-office magnifier training** 1–4 months after receiving new magnifiers: **reading ability improved significantly, and outcomes did not differ between telerehabilitation and in-office care.** A **2025 systematic review and meta-analysis** of low-vision rehabilitation techniques (**magnifiers, telescopes, microperimeter feedback**) in AMD found that **low-vision rehabilitation improves depression**, while improvement in reading speed was non-significant because of heterogeneity.

7. Falls guidance and Indian context

The **2023 Cochrane review** showed **home fall-hazard interventions reduce falls by 26% overall and 38% in higher-risk people**, while **assistive devices alone and education alone do not**. **NICE NG249 (April 2025)** recommends **offering a home hazard assessment with a validated tool**, **considering an occupational therapist to carry it out**, and referring to an ophthalmologist if visual impairment is **caused by cataract**. In India, the **RAAB 2015–19 survey** found blindness prevalence **1.99%** in those ≥50, with **cataract (66.2%)** leading, and **untreated cataract (48.0%) and uncorrected refractive error (41.5%)** the major contributors to visual impairment; **about 93% of blindness and 96% of visual impairment was avoidable, and more than half of avoidable causes were treatable. Rashtriya Vayoshri Yojana** supplies **free spectacles** and other devices through **ALIMCO**; services run through **NPCBVI** alongside **NPHCE**.

Current Practice Update

Issue	Textbook Position	Current Evidence (2023–present)
Framing	Vision problems managed disease-by-disease within "sensory impairment"	**Vision rehabilitation is a named health system service** integrated into UHC — WHA78.7 (2025) plus WHO's planning guide
Aids	Magnifier, lighting intensifiers, tinted glasses, night light, large print	Retained, but **device prescription alone is insufficient — training produces the gain**; **stand-based electronic magnification** specifically recommended
Central field loss	Reading glasses or magnification for AMD	**Visual skills training (eccentric viewing) is now specifically recommended**
Psychological care	"Depression may occur and should be treated"	**Psychological therapy is one of the two intervention types with evidence of improving vision-related QOL**; rehabilitation itself **improves depression in AMD**
Outcome measured	Not specified	**Vision-related QOL, reading, ADL/IADL and mood** — generic health QOL shows no important change
Service delivery	Clinic-based	**Telerehabilitation is non-inferior to in-office care** for magnifier training and reading
Falls	Visual impairment is a risk factor for falls and fractures	**Vision correction alone does not reduce falls** — works with **exercise** and **OT-led home hazard modification**; **single-vision lenses outdoors**; first-eye cataract surgery helps, second-eye does not
Refractive error	Optimum eyeglasses mentioned	**SPECS 2030: a 40-percentage-point increase in effective refractive error coverage by 2030**

Exam One-Liners

- **Low vision = irreversible visual impairment with useful residual vision** — rehabilitation targets **function, not acuity**.
- **Treat the treatable first** — in India **~93% of blindness in those ≥50 is avoidable**, mostly cataract and refractive error.
- **75% of older adults have unreported visual or hearing dysfunction** — screen actively.
- **AMD → central loss** (reading, faces, driving); **glaucoma → peripheral loss** (mobility, falls).
- **Total blindness does not occur in AMD** — peripheral vision is retained.
- **Aids:** magnifier, **lighting intensifiers without glare**, tinted glasses, **night light**, large print.
- **Eccentric viewing training** is the specific technique for central scotoma.
- **A magnifier prescribed without training usually fails.**
- **Visual impairment predicts falls and fractures; cataract surgery lowers hip-fracture risk.**
- **Vision correction reduces falls only when combined with exercise and home hazard modification.**

- **Timolol drops can cause bradycardia, heart failure, bronchospasm, confusion and depression.**
- **Depression in AMD is expected and treatable** — and rehabilitation itself improves it.
- **Telerehabilitation is as effective as in-office low vision training.**
- **WHA78.7 (2025)** = first WHA resolution combining **vision and hearing** care across the life course.

Q6. Rehabilitation of a patient with osteoarthritis of the knee joints

High-Yield Points

A. Definition and Pathology

1. **Osteoarthritis (OA)** is characterised by **cartilage destruction with subsequent joint space loss, osteophyte formation and subchondral sclerosis.** It is the **most common joint disease of older adults** and the **major cause of knee, hip and back pain.**
2. **OA is not, by definition, an inflammatory disease**, although **synovial hypertrophy and joint effusions are typical.** Progression involves **chondrocyte regulation of the extracellular matrix, genetic influences, local mechanical factors and inflammation.**

Feature	Osteoarthritis	Inflammatory arthritis
Duration of stiffness	**Minutes**	Hours
Pain	**Usually with activity**	Even at rest and at night
Fatigue	Unusual	Common
Swelling	Common, **little synovial reaction**	Very common, with synovial proliferation
Erythema and warmth	Unusual	Common

B. Burden

4. **Musculoskeletal disorders are the commonest cause of disability in old people** — almost **60% of people aged ≥65 report arthritis or chronic joint symptoms. High BMI is associated with an increased risk of knee OA in older persons.**
5. **Indian context:** the **pooled prevalence of knee OA among elderly Indians is about 47%.** Contributing factors specific to India: **squatting, cross-legged floor sitting, Indian-style toilets, heavy manual labour, obesity and postmenopausal status.**

> **C. The Vicious Cycle — the central concept in OA rehabilitation**
>
> **People with arthritis avoid activities that provoke joint pain → muscles weaken → joints become unstable and easily injured → pain increases → exercise is avoided further → cardiorespiratory fitness falls critically**, particularly in the very old, **in whom even simple ADLs demand oxygen uptakes close to the age-associated maximum. The end result is an unfit, inactive, arthritic person whose independence is compromised.**
>
> *Rehabilitation exists to break this cycle, and every component below is aimed at one of its links.*

7. **The good news that drives the whole programme:** pathological OA changes occur against a background of **age-related loss of flexibility that is itself reversible**. **Exercise increases joint flexibility and improves the strength, size and resilience of cartilage, ligaments and muscles.** Arthritic patients should be **encouraged to develop physical fitness and set realistic goals.**

D. Assessment

8. **History:** nature and duration of disability, **pain (site, pattern, aggravating and relieving factors)**, comorbidities, **drugs that affect mobility**, motivation and psychological factors, and the home environment.
   Examination: skin, cardiopulmonary status, **musculoskeletal assessment — muscle tone and strength (MRC 0–5, especially knee extension), joint range of motion, foot deformities and lesions** — neurological deficits, and **levels of mobility: bed mobility, transfers, standing balance, gait, stair climbing, and pain with movement.**
9. **Investigations: plain radiography** remains the main modality for assessing severity and progression; **MRI and ultrasound are more accurate and comprehensive** but not needed routinely.
10. **Outcome tools: WOMAC** (pain, stiffness, physical function), **Timed Up and Go, 30-second chair stand, 6-minute walk test**, and **Barthel Index/FIM**.

E. Goals and Therapeutic Exercise

11. **The goal is to restore function and prevent further disability**, using a **restorative philosophy of care**: **careful assessment of function and underlying capability, realistic goals, prevention of secondary disability and the complications of immobility, evaluation and adaptation of the environment, and ongoing motivation of both patient and caregivers.**
12. **Non-pharmacological management should be the focus of intervention** — weight loss, physical therapy to strengthen the related musculature, local ice and heat, and exercise programmes to maintain strength and function.
13. **Physiotherapy objectives: relieve pain; evaluate, maintain and improve joint range of motion; improve strength, endurance, motor skills and coordination; improve gait and stability; assess the need for and teach the use of assistive devices. Modalities: active exercise (isometric and isotonic) and passive exercise**, sitting exercise programmes, heat (hot packs, paraffin, diathermy, ultrasound), hydrotherapy and TENS.
14. **A practical knee OA exercise prescription:**
    - **Quadriceps strengthening** — static quadriceps sets, straight-leg raises, **inner-range quadriceps**, progressing to sit-to-stand and step-ups
    - **Hip abductor and extensor strengthening** — reduces the adduction moment across the knee
    - **Daily range-of-motion exercises** — **particularly important in restoring function in arthritic joints**
    - **Balance and proprioceptive training** — the knee is unstable and falls risk is high
    - **Low-impact aerobic exercise** — walking, cycling, aquatic exercise
    - **Flexibility** — hamstring and calf stretching; avoid fixed flexion contracture
15. **Adherence is the biggest problem, not the prescription. Compliance with range-of-motion exercise is low at all ages. Joining others in group activities adds a social component and increases compliance** — hence supervised group programmes rather than a printed home sheet.
16. **Weight management: weight-loss therapy improves physical function, quality of life and the medical complications of obesity in older persons**, but **must minimise muscle and bone loss.** Primary approach is **lifestyle change** with a **modest energy reduction of**

500–700 kcal/day, and **regular physical activity is particularly important in obese older people to preserve muscle and bone mass.**

F. Pain Management

17. **Time the analgesia to the exercise.** Because **OA pain is typically exacerbated by exercise, it is best to medicate beforehand** — this single measure converts a failed physiotherapy session into a successful one.
18. **Pharmacological adjuncts:** simple analgesics, **topical and oral NSAIDs, intra-articular corticosteroid injection, viscosupplementation. Glucosamine and chondroitin show no evidence of significant pain improvement** beyond a possible placebo effect, and **vitamin D does not reduce pain or repair structural damage. Refractory pain is best managed by a multidisciplinary pain team.**

G. Orthoses, Footwear and Walking Aids

19. **Footwear: patients with painful knees benefit from soft-soled shoes with cushioned heels (e.g. jogging shoes).** A **rocker-bottom shoe** reduces forefoot pain by assisting weight transfer from posterior to anterior. **Metatarsalgia** is reduced by an **internal pad placed proximal to the MTP joints**, and **medial arch collapse** by a **medial arch-supporting orthosis.**
20. **Walking stick rules:**
    - A cane **held in the contralateral hand reduces weight on an arthritic hip**
    - **For knee pain, the patient should experiment with holding the cane in either hand** — the contralateral rule is not automatic for the knee
    - **Cane length must equal the distance from the wrist crease to the floor** — a wrong length creates secondary problems in other joints
    - **Ferrules (stick rubbers) must be checked regularly and replaced when worn, to reduce the risk of falls**
21. **Occupational therapy and the home: a home-based assessment by an occupational therapist is often invaluable. Simple ergonomic measures and aids — tap turners, zipper pulls, sock pulls, stretch laces, long-handled shoe-horns and Velcro fasteners — notably reduce joint strain and consequent pain.** Add **raised toilet seat, grab bars, shower chair, raised chair and bed heights, and first-floor living.** *In the Indian setting, converting an Indian-style squatting toilet to a Western-style commode is one of the highest-yield single modifications.*
22. **Physical modalities — know the evidence limits. Heat, including baths and spas, has traditionally been used for arthritic joints. There is no evidence that hydrotherapy produces measurable functional improvement in arthritic joints, although it does promote self-confidence.** Use these as **adjuncts that enable exercise**, not as treatments in their own right.
23. **Surgery: total knee arthroplasty** is reserved for severe symptoms and functional failure despite optimal conservative care, requiring **pre-operative optimisation (prehabilitation, quadriceps strengthening, weight and comorbidity control)** and structured post-operative rehabilitation. **Arthroscopic surgery for knee OA has been shown in a controlled trial to be of no benefit.**
24. **Honest evidence position: there is little evidence for the efficacy of structured multidisciplinary rehabilitation programmes in musculoskeletal disorders generally. Time-limited, goal-directed multidisciplinary rehabilitation is of proven benefit only**

in chronic back pain, other chronic pain, and after hip fracture in frail older people — which is why **exercise, weight loss and self-management, not the programme label, carry the evidence in knee OA.**

Latest Updates (2023–Present)

1. Guideline consensus — the "core set"

OARSI emphasises a **core set of non-pharmacological management for all patients**: **formal exercise (aerobic, strengthening, neuromuscular, aquatic or mind-body), weight reduction in overweight or obese patients, and continuing education**. **Assistive devices such as bracing and walkers are recommended only in select cases. Pharmacological therapy is adjunctive — oral and topical NSAIDs**, with **short-course intra-articular corticosteroid** if these fail. Critically for geriatrics: **for individuals with cardiovascular comorbidities or frailty, no oral NSAID is recommended**; for gastrointestinal comorbidity, **COX-2 inhibitors or an NSAID plus PPI**.

2. ACR/Arthritis Foundation — the specific do's and don'ts

Strongly recommended for knee and hip OA: exercise, self-efficacy and self-management programmes, weight loss, and use of a cane. Tai chi is strongly recommended; **yoga is conditionally recommended for knee OA.** The guideline **strongly recommends against TENS**, and **conditionally recommends against manual therapy, massage, modified shoes and weighted insoles**, and (for knee OA) **against pulsed vibration therapy.**

3. The biggest single change — GLP-1 receptor agonists (STEP 9, NEJM 2024)

The **STEP 9 trial** randomised **407 adults with obesity (mean age 56, 81.6% women, mean BMI 40.3) and moderate knee OA with moderately severe pain** 2:1 to **once-weekly subcutaneous semaglutide 2.4 mg or placebo**, alongside physical activity and lower-calorie diet counselling, for **68 weeks**. **Body weight fell by 13.7% versus 3.2%** (treatment difference −10.5; P<0.001), and the **WOMAC pain score improved by −41.7 versus −27.5** (treatment difference −14.2, 95% CI −20.0 to −8.3; P<0.001). **SF-36 physical function also improved more with semaglutide (+12.0 vs +6.5 points; P<0.001).** This **may reduce the need for NSAIDs and opioids**; an earlier trial of **liraglutide 3.0 mg showed no significant pain difference.**
*Exam framing: in the obese older patient with knee OA, **weight loss is now a pharmacologically achievable rehabilitation intervention** — but its role in frail, sarcopenic elders is unproven and it must be paired with **resistance exercise and protein adequacy** to protect muscle.*

4. GLA:D® — the reference model for structured OA rehabilitation

The Danish **GLA:D programme** delivers **guideline-aligned group education and supervised exercise therapy** for hip or knee OA through **2–3 education sessions of 90 minutes plus 12 supervised neuromuscular exercise sessions of 60 minutes, twice weekly for six weeks.** Around **6000 participants enrol annually across nearly 300 Danish physiotherapy clinics**, with a national registry at baseline, 3 and 12 months.

5. Digital delivery and injectables

A **2025 single-blind RCT** randomised **42 older adults (mean age 68.4) with mild-to-moderate knee OA** to a **sensor-augmented telerehabilitation system (electromyographic and inertial sensing)** or **conventional face-to-face physiotherapy for eight weeks**. Systematic reviews in **2024–2025** support **digitally delivered exercise** and **telerehabilitation for pain, strength and balance**; the limiting factor is **digital readiness**. Recent reporting indicates **platelet-rich plasma shows no long-term advantage over standard care in early knee OA**.

6. Indian epidemiology and a practical intervention target

A **2025 systematic review and meta-analysis (14 studies, 5029 participants)** found the **pooled prevalence of knee OA among elderly persons in India to be 47% (95% CI 38.4–55.8%)**, attributing the burden partly to **strenuous manual labour and cultural practices such as sitting cross-legged and squatting.** A **2024 community study in rural South India** found knee OA in **more than one-third of adults aged ≥40**, with predictors including **obesity, diabetes, hypertension, sedentary lifestyle, menopause and use of Indian-style toilets**; the authors recommend **affordable alternatives to squatting toilets, knee-strengthening exercise tailored to postmenopausal women, and better rural access to physical therapy.**

Current Practice Update

Issue	Textbook Position	Current Evidence (2023–present)
Core treatment	Non-pharmacological management should be the focus	Reinforced — **exercise + weight management + education is the universal "core set"**
Exercise type	Daily range-of-motion exercise; general fitness	Broadened to **aerobic, strengthening, neuromuscular, aquatic or mind-body**; **tai chi strongly recommended**
Exercise dose	Not specified	**GLA:D template: 12 supervised neuromuscular sessions over 6 weeks plus structured education**
Weight loss	Lifestyle change, 500–700 kcal/day deficit	**Semaglutide 2.4 mg produced 13.7% weight loss and clinically meaningful WOMAC pain reduction (STEP 9, NEJM 2024)**
NSAIDs in the elderly	Analgesics/NSAIDs listed as options	**Oral NSAIDs not recommended in cardiovascular comorbidity or frailty**; **topical NSAIDs first-line**
TENS and heat	Listed among physiotherapy modalities	**TENS is strongly recommended against**; heat and hydrotherapy remain **enablers of exercise**
Glucosamine/ chondroitin, vitamin D	No evidence of benefit	Unchanged — **do not prescribe**
Injections	IA steroid and viscosupplementation listed	**IA steroid = short-term adjunct only**; **PRP shows no long-term advantage**
Delivery	Clinic and group programmes	**Digital and sensor-augmented telerehabilitation** now have RCT support
Programme evidence	Little evidence for structured MDT rehabilitation in musculoskeletal disease	Still true at programme level, but **individual components have strong, replicated evidence**

Exam One-Liners

- **The OA vicious cycle:** pain → activity avoidance → muscle weakness → joint instability → more pain → deconditioning → lost independence.
- **Age-related joint stiffness is reversible** — exercise improves strength, size and resilience of cartilage, ligaments and muscle.
- **Give the analgesic before exercise**, because OA pain is exacerbated by activity.
- **OA stiffness lasts minutes; inflammatory stiffness lasts hours.**
- **Quadriceps + hip abductor strengthening + aerobic exercise + weight loss** is the core prescription.
- **Compliance is the limiting factor — group exercise improves it.**
- **Cane length = wrist crease to floor**; check and replace worn ferrules.
- **For hip pain hold the cane contralaterally; for knee pain, try either hand.**

- **Soft-soled shoes with cushioned heels** for painful knees; **rocker-bottom sole** for rigid forefoot.
- **OT aids:** tap turners, zipper pulls, sock pulls, stretch laces, long-handled shoe-horn, Velcro fasteners.
- **Hydrotherapy improves confidence, not measurable joint function.**
- **Glucosamine, chondroitin and vitamin D do not reduce OA pain.**
- **Avoid oral NSAIDs in the frail or cardiovascularly comorbid elder** — topical NSAIDs first.
- **Arthroscopic surgery for knee OA is of no benefit.**
- **STEP 9 (2024): semaglutide 2.4 mg → 13.7% weight loss and WOMAC pain −41.7 vs −27.5.**
- **GLA:D dose: 12 supervised neuromuscular exercise sessions over 6 weeks + education.**
- **India: knee OA affects about 47% of elderly** — target **squatting, cross-legged sitting and Indian-style toilets**.

Q7. Community-based rehabilitation (CBR)

High-Yield Points

A. Definition and Concept

1. **Community-Based Rehabilitation (CBR)** is defined (ILO/UNESCO/WHO Joint Position Paper, 2004) as **a strategy within general community development for the rehabilitation, poverty reduction, equalisation of opportunities and social inclusion of all people with disabilities.**
2. **CBR is implemented through the combined efforts of people with disabilities themselves, their families, organisations and communities, and the relevant governmental and non-governmental health, education, vocational, social and other services.** It is **not** simply "physiotherapy delivered at home" — it is a **multisectoral development strategy**.
3. **Evolution:** CBR began after **Alma-Ata (1978)** to extend basic rehabilitation to people who could never reach an institution. It has since **evolved into a comprehensive, multisectoral approach now recognised as the core strategy for Community-Based Inclusive Development (CBID)** — a shift from *delivering services to the disabled* to *making the community itself inclusive*.
4. **Policy anchors:** the **Helsinki International Consultation (2003)**, the **ILO/UNESCO/WHO Joint Position Paper (2004)**, **World Health Assembly resolution WHA58.23 (2005)** urging Member States to promote and strengthen CBR, and the **WHO CBR Guidelines (2010)** developed with over 180 individuals from nearly 300 organisations, mostly in LMICs. **CBR is now implemented in over 90 countries**, and the guidelines position CBR as a means of implementing the **UNCRPD (Article 26 — Habilitation and Rehabilitation)**.

B. The CBR Matrix — the core framework

Five components, each with five elements (5 × 5 = 25 elements). It is a **flexible "pick-and-mix" framework, not a sequential programme** — no single agency delivers all 25 elements, so **partnership across sectors is essential**.

HEALTH	EDUCATION	LIVELIHOOD	SOCIAL	EMPOWERMENT
Promotion	Early childhood	Skills development	Personal assistance	Advocacy & communication
Prevention	Primary	Self-employment	Relationships, marriage & family	Community mobilisation
Medical care	Secondary & higher	Wage employment	Culture & arts	Political participation
Rehabilitation	Non-formal	Financial services	Recreation, leisure & sports	Self-help groups
Assistive devices	Lifelong learning	Social protection	Justice	Disabled people's organisations

6. **The WHO CBR Guidelines comprise 7 booklets** — an Introduction with a **Management chapter**, **one booklet per matrix component**, and a **Supplementary booklet** covering

HIV/AIDS, mental health, leprosy and disasters — areas historically neglected by CBR programmes.

7. **Core principles: inclusion, participation, sustainability, self-advocacy, empowerment, non-discrimination**, use of **local resources and appropriate technology**, **community ownership**, **intersectoral collaboration**, and **referral linkage to secondary and tertiary services**.
8. **Three-tier structure:** the **person with disability and family** at the centre → the **community and CBR worker → linkage upward to district and referral services**. The programme fails if the referral link is missing.

C. Community-Based Rehabilitation in the Older Person

9. **The rehabilitation setting should be a menu, not a single option** — stand-alone rehabilitation hospitals, designated rehabilitation units, general wards, nursing and residential homes, **day hospitals, community day centres, outpatient rehabilitation centres, and the patient's own home. Ideally a range of options should be available to meet the needs of individual patients at a given time.**

> **Critical caveat for the exam**
>
> **Community-based rehabilitation has come of age as a *complement to*, not a replacement for, hospital-based rehabilitation. In practice it only suits patients at the least disabled end of the spectrum who are otherwise well supported in the community.** A severely disabled elder living alone is not a CBR candidate for the acute phase.

11. **Intermediate care** is **intensive, short-term, community- or home-based rehabilitation that aims to prevent hospitalisation, facilitate early discharge from hospital, and/or maximise independent living.** It is the geriatric bridge between hospital and home.
12. **Community long-term care programmes that support CBR (Kane): home care (home nursing and homemaking), adult day care, adult foster care, assisted living, geriatric assessment, hospice, telephone reassurance, caregiver support, congregate housing, home repairs, congregate and home-delivered meals, respite care, and emergency alarms.**

Service	Who it is for and what it delivers
Senior centre	For the **well** elderly; recreational, usually recreation-department run
Adult day care	For those with **limited functional ability** (some specifically for cognitive impairment); supervised activities that **improve basic ADL and social skills**, and at minimum provide **respite for the primary caregiver — which may make the critical difference in allowing an impaired older person to remain at home.** Most attend **2–3 days a week**
Adult day health centre	Day care **plus nursing, physiotherapy and social work**
Day hospital	Virtually all hospital services on an ambulatory basis, with **emphasis on rehabilitation, especially physiotherapy and occupational therapy**

14. **Home-based assessment and the home visit.** A **hospital-to-home assessment programme** shortens hospital stay, allows **evaluation of the home itself and how the**

patient actually functions within it, and can be **targeted at those who will derive maximal benefit**. **Preventive home visitation** aims at primary prevention (immunisation, exercise), secondary prevention (detecting untreated problems) and tertiary prevention (improving medication use) — **but its benefits have not been definitively confirmed, so it cannot be recommended as routine practice.**

15. **Case management is the glue.** In most cases a **strategy for long-term follow-up and often formal case management should be developed to ensure that needs and services are appropriately matched and followed through. Discharge from hospital is a transition in rehabilitation, not its conclusion**, and **some follow-up is essential.**
16. **Integrated models: PACE (Program of All-Inclusive Care of the Elderly)** uses **pooled capitated Medicare and Medicaid funding to deliver integrated health and social services to older people who are nursing-home eligible but still living in the community** — the best example of creative integration, though **expensive and targeting a narrow group**. **Hospital-in-the-Home (Australia)** delivers hospital-level care at home with **quality at least equivalent to traditional hospital care** in cost, satisfaction and medical outcomes.
17. **Technology extends CBR's reach. Remote sensors, wireless data transfer, GPS, robotics and mobile computer systems facilitate early transfer from hospital-based to home-based rehabilitation**, with **cost savings, fewer hospital-acquired infections, and return to a familiar environment**. The same technology then **maintains independence, reduces caregiver burden and prevents institutionalisation.**
18. **CBR team:** the **CBR worker / community rehabilitation worker** (in India an **ASHA, ANM or Multipurpose Worker**), **family caregivers**, **physiotherapist, occupational therapist, speech therapist**, **community nurse**, **social worker**, **primary care physician with geriatric linkage**, **Panchayati Raj and self-help groups**, and **Disabled People's Organisations**. **Even in low-resourced settings, health workers can be trained to detect declines in physical and mental capacity and deliver effective interventions** — the premise of the **WHO ICOPE package**, deliberately **based in community and primary care to maximise accessibility**, with **Step 5 devoted to enlisting the community and supporting caregivers**.

D. Why CBR Matters Especially in LMICs

19. **Institutional care is almost absent in low- and middle-income countries** — only **0.1% of older people live in institutions in Iran, 0.6% in Botswana and about 1% in Brazil and Mexico, compared with 7.5% in Switzerland. This very lack of infrastructure opens the way to create alternative community-based long-term care systems.**
20. **Informal family care is the cornerstone.** In dementia, **informal care accounts for 58% of all costs in low-income and 65% in middle-income countries, versus 40% in high-income countries**, while **direct social care costs are only 4% in LMICs versus 42% in high-income countries. Most family care in developing countries is provided by women**, and this supply is shrinking with female labour-force participation, migration and smaller families.
21. **Advantages of CBR:** low cost, wide coverage, culturally appropriate, uses local resources, keeps the person in their own environment and roles, reduces institutionalisation, empowers families, and addresses **participation restriction**, which hospital services never reach.
    Limitations: variable quality and supervision, dependence on committed community workers, weak referral pathways, **poor evidence base for programme-level effectiveness**, funding

insecurity, risk of **shifting the burden onto already-overloaded women in the family**, and unsuitability for severely disabled or unsupported patients.

Latest Updates (2023–Present)

1. The scale of the need has been formally quantified

A landmark study jointly developed by **WHO and the Institute for Health Metrics and Evaluation** established that **one-third of the world's population — about 2.41 billion people — live with a health condition that would benefit from rehabilitation**, and that this **need has risen 63% since 1990 (from 1.48 billion)**. The study **establishes rehabilitation as a key strategy for universal health coverage in the 21st century and challenges the view that rehabilitation is an optional service needed only by a minority.** The main drivers are **ageing, rising non-communicable disease, and the consequences of injury**, and **more than half of those needing rehabilitation in LMICs do not receive it.**

2. WHA76.6 (May 2023) — the first-ever WHA resolution on rehabilitation

It calls for **expanding and integrating rehabilitation into health systems as part of universal health coverage**, with emphasis on **rehabilitation in primary care** and in **emergency preparedness and response**, and highlights the **critical shortage of rehabilitation workforce**. It was the **first resolution to directly address rehabilitation in 75 years of the World Health Assembly**, found global need **largely unmet — in many countries fewer than half receive the care they require** — and commits Member States to **expand financing, integrate rehabilitation into national health systems, minimise the prohibitive costs of assistive technologies such as hearing aids, glasses, wheelchairs and prosthetics, and promote research.**

3. World Rehabilitation Alliance and the Package of Interventions for Rehabilitation

Following the **3rd Global Rehabilitation 2030 meeting (July 2023)**, WHO launched the **World Rehabilitation Alliance (WRA)**, conducting evidence-based advocacy through **five workstreams: (1) rehabilitation workforce, (2) rehabilitation in primary care, (3) rehabilitation in health policy and systems research, (4) rehabilitation in emergencies, (5) external relations.** WHO's **Package of Interventions for Rehabilitation (PIR)** helps ministries of health integrate rehabilitation into health systems, **with particular focus on LMICs**. PAHO has issued a **Basic package of interventions for rehabilitation: a toolkit for primary care professionals (March 2025)**.

4. CBR is now framed as CBID; the evidence base is broadening but still thin

CBR is described as a **community development strategy** promoting **inclusion, participation and empowerment of persons with disabilities by addressing both physical and sociological barriers**, now recognised as a **core strategy for community-based inclusive development (CBID)**, with components **not implemented sequentially but as a flexible "pick-and-mix" framework** linking community resources with existing government services. A **2024 systematic review of the grey literature (International Journal of Mental Health Systems)** examined **CBR for people with psychosocial disabilities in LMICs** — reflecting the continuing reliance on non-peer-reviewed programme evidence. Alongside this, the **2024 BMC Medicine meta-analysis** showed geriatric

rehabilitation in inpatient *and day-hospital* settings **reduced mortality, care-home admission and functional impairment**, and the **BGS 2024 report** argues rehabilitation should be a component of every service for older people.

5. India — the operational structures to name in an exam

District Disability Rehabilitation Centres (DDRCs) were initially planned for 325 districts, but **with effect from 9 December 2020 all districts were approved** for DDRCs, funded under the **Deendayal Disabled Rehabilitation Scheme (DDRS) from 2020–21**. Services include **survey and identification of persons with disabilities through a camp approach, awareness generation, assessment/provision/fitment/repair of assistive devices, support to ADIP/ALIMCO camps, vocational identification and training, and referral services**. Each DDRC provides **therapeutic services — physiotherapy, occupational therapy and speech therapy** — and facilitates **disability certificates, bus passes and concessions**. Each is run by a **District Management Team headed by the District Magistrate/Collector**.

Other Indian structures: **NPHCE** (19 Regional Geriatric Centres, 2 National Centres for Ageing), **Ayushman Arogya Mandir / Health and Wellness Centres** as the primary-care delivery point, **ASHA/ANM** as the CBR workforce, **Rashtriya Vayoshri Yojana** and **ADIP/ALIMCO** for assistive devices, **Composite Regional Centres (CRCs)**, and **AB-PMJAY extended to all aged ≥70 years (2024)**.

Key legal gap: the **Rights of Persons with Disabilities Act, 2016** governs disability services, but **age-related functional decline does not automatically confer "disability" status**, so many frail elders fall outside the DDRC pathway — an argument for **explicitly linking NPHCE and DDRC services**.

Current Practice Update

Issue	Textbook Position	Current Evidence (2023–present)
Scale of need	Not quantified	**2.41 billion people worldwide (one-third of the population); need up 63% since 1990; >50% unmet in LMICs**
Status of rehabilitation	A service within geriatric medicine	**An essential health service within UHC** — WHA76.6 (2023)
CBR concept	A complement to hospital care	Reframed as **Community-Based Inclusive Development (CBID)** — a multisectoral development strategy
Who CBR suits	"Only the least disabled end of the spectrum, well supported in the community"	Still broadly true for *acute* rehabilitation, but **hospital-at-home, early supported discharge and telerehabilitation have widened the eligible group**
Delivery point	Day hospitals, day centres, home, outpatient	**Primary care is now the named priority setting** — WRA workstream 2, PAHO toolkit, WHO PIR
Preventive home visits	Benefits not confirmed; not routine	Broadly unchanged; evidence has shifted to **targeted home-hazard assessment by an OT (26–38% fall reduction)**
Assistive technology	Aids and adaptations as a rehabilitation step	**Cost of assistive technology is a named policy target**; spectacles, hearing aids, wheelchairs and prosthetics framed as core UHC entitlements
Workforce	Multidisciplinary team	**Global workforce shortage explicitly named**; task-sharing endorsed — **but ATTEND (India) showed untrained family-delivered therapy alone does not improve outcomes**
India	Not covered	**DDRCs now approved in all districts**; NPHCE, RVY/ADIP and AB-PMJAY ≥70 years form the practical CBR web

Exam One-Liners

- **CBR = a strategy within general community development** for rehabilitation, poverty reduction, equalisation of opportunities and social inclusion (**ILO/UNESCO/WHO, 2004**).
- **CBR Matrix = 5 components × 5 elements = 25:** Health, Education, Livelihood, Social, Empowerment.
- **Health component elements:** promotion, prevention, medical care, **rehabilitation, assistive devices**.
- **Empowerment elements:** advocacy and communication, community mobilisation, political participation, **self-help groups, Disabled People's Organisations**.
- **WHO CBR Guidelines (2010) = 7 booklets**; supplementary booklet covers **HIV/AIDS, mental health, leprosy and disasters**.
- **WHA58.23 (2005)** urged Member States to strengthen CBR; **CBR runs in over 90 countries**.
- **CBR is the operational vehicle for UNCRPD Article 26.**

- **CBR complements, never replaces, hospital rehabilitation.**
- **Intermediate care** = short-term intensive community/home rehabilitation to **prevent admission, enable early discharge and maximise independence**.
- **Day care may make the critical difference in keeping an impaired elder at home** — often through **carer respite** alone.
- **Senior centre = well elderly; day care = functionally limited; day hospital = rehabilitation-focused ambulatory hospital care.**
- **Institutional care is ~0.1–1% in LMICs versus 7.5% in Switzerland.**
- **Informal care = 58–65% of dementia costs in LMICs versus 40% in high-income countries.**
- **2.4 billion people need rehabilitation; over half in LMICs do not receive it.**
- **India: DDRCs are now approved in every district.**

Q8. Mobility aids

High-Yield Points

A. Definition and Purpose

1. **Mobility aids are assistive products that support or substitute for lost locomotor function**, enabling the older person to transfer, stand, walk and move about safely. They are one arm of the wider category of **“aids and adaptations”**.
2. **Purposes: widen the base of support and improve balance**; **unload a painful or weak limb**; **compensate for reduced proprioception and reaction time**; **increase confidence and reduce fear of falling**; **extend walking distance and endurance**; and **enable participation**.
3. **Aids reduce activity and participation restriction**, and range **from the simple and inexpensive to the technologically advanced**.

> **Two cardinal cautions — both examinable**
>
> • **“The provision of an aid is not always the best option, as it can foster dependence rather than independence.”**
> • **“Those in real need often do not avail themselves of even basic aids and appliances.”**
> → Under-provision and over-provision are both errors; the answer is **assessment, not reflex prescription**.

5. **Lack of mobility aids is itself listed as a cause of immobility**, alongside medical orders, restrictive devices and chemical restraints. Immobility produces **muscle atrophy, loss of strength and endurance, bone loss, joint contractures, impaired balance and coordination, more falls, and — through reduced bone mass — more serious injury when falls occur.**
6. **About one-quarter of community-dwelling older adults use at least one mobility device. The incidence of falls and recurrent falls is not associated with any particular device or with the use of multiple devices.**
7. **Older adults who own devices fall more often when they are *not* using them**, and **those who fall in the absence of their device suffer more serious injuries.** This is likely confounded, because **consistent and effective use of an assistive device itself requires robust cognitive and functional capability.**

B. Classification

8. **Ambulatory (walking) aids** — sticks/canes, crutches, walking frames and rollators. **Wheeled mobility** — manual wheelchairs, powered wheelchairs, scooters. **Transfer aids** — trapeze/monkey pole, transfer pole or bar, transfer board, hoist, raised bed and toilet seats. **Environmental aids** — handrails, ramps, grab bars, stair lifts. **Orthoses and prostheses** — an **orthosis** is applied to the external surface to **support, improve function, or restrict or promote movement**; a **prosthesis replaces a body part**.

C. Choosing the Device — Match the Aid to the Deficit

Principle: prescribe the least restrictive device that gives adequate safety. A device more supportive than needed accelerates deconditioning; one less supportive causes falls.

Device	Best indication	Support given	Main drawbacks
Single-point cane	Mild unilateral weakness or pain; slight balance loss	Widens base a little; **unloads the opposite limb by roughly 20–25%**	Minimal stability; useless in bilateral weakness
Quadripod (quad) cane / tripod	Hemiplegia; moderate unilateral weakness	Greater lateral stability, **free-standing**	Heavier, slower gait; all four tips must contact evenly; poor on stairs
Hemi-walker (walk cane)	Dense hemiplegia with one functional arm	Maximum one-handed support	Bulky, encourages asymmetric gait
Axillary crutches	Short-term non/partial weight-bearing after fracture or surgery	Large unloading	High energy cost; **axillary nerve/vessel compression** if leant on; rarely tolerated by frail elders
Elbow (Lofstrand) crutches	Longer-term partial weight-bearing with good upper-limb strength	Good unloading, frees hands	Needs coordination and grip strength
Standard (pick-up) frame	Bilateral weakness, poor balance, generalised frailty	**Maximum stability of the walking aids**	**Abolishes reciprocal arm swing, promotes flexed posture, slow, unusable on stairs**
2-wheeled front-wheeled walker	Frail elder who cannot lift a frame	Good stability with smoother gait	Slight loss of stability versus standard frame
4-wheeled rollator	Cardiorespiratory limitation, Parkinson's disease, long distances	Enables **rest stops**, better speed, carries belongings	**Can run away downhill; needs intact grip, cognition and braking ability**
Manual wheelchair	Cannot walk functional distances	Total substitution	Deconditioning if over-used; pressure and seating problems
Powered wheelchair / scooter	Adequate cognition and vision, poor upper-limb power or endurance	Independence over long distances	Cost, transport, safety and cognitive screening required

10. **The Berg Balance Scale gives a practical decision rule: 0–20 = wheelchair-bound; 21–40 = walking with assistance (needs an aid); 41–56 = independent.**

D. Fitting and Measurement

11. **Cane length should equal the distance from the wrist crease to the floor**, standing upright in usual footwear with arms hanging. **Cane length matters because a wrong length creates secondary problems in other joints.**

Parameter	Standard
Handle height (cane, crutch, walker)	Level of the **distal wrist crease / ulnar styloid**; approximately the level of the **greater trochanter**
Elbow flexion when gripping	**20–30°**
Cane tip position	About **2 inches lateral and 6 inches anterior** to the toes
Axillary crutch pad	**About 2 finger-breadths (≈5 cm) below the axilla** — never weight-bear on the axilla
Forearm crutch cuff	**1–1.5 inches distal to the olecranon**
Walker width	Roughly **2 inches wider** than the patient; front legs level with the midpoint of the shoes

12. **Which hand? A cane held in the contralateral hand limits pain by reducing weight on an arthritic hip. For knee pain, the patient should experiment with holding the cane in either hand.** In hemiplegia, the cane goes in the **unaffected hand**.
13. **Gait sequences: cane and affected leg advance together, then the sound leg** ("cane–bad–good"); **three-point gait** for non-weight-bearing; **partial weight-bearing gait** for protected loading; **four-point gait** for maximum stability. **Stairs: "up with the good, down with the bad"** — the aid goes with the affected limb.
14. **Maintenance is a safety issue. Ferrules must be checked regularly and replaced when worn, to reduce the risk of falls.** Also check **wheel locks, brakes, screws, handgrips and folding mechanisms**, and ensure the device is **stored where the patient will actually reach for it**.

E. Wheelchairs and Seating

15. **Wheelchairs were originally designed for transport, not for prolonged sitting** — their **sling-back seats do not provide appropriate support.** In nursing homes, **over 50% of residents use a wheelchair and most spend the majority of the day in it**, frequently in chairs that do not fit.
16. **Seating problems cause pain and agitation — and therefore falls and restraint use:** poor back support; a chair **too tall, too heavy or too wide**; **footrests set too high**; and the **hammock effect of the sling seat.** The uncomfortable patient attempts to transfer unsafely.
17. **Seating solutions:** a **wedge cushion under the buttocks and thighs tilts the resident backwards**; a **wedge seat prevents sliding forward**; **lateral supports or cushions correct leaning to one side**; and in **hemiplegia at risk of shoulder subluxation**, an **arm trough, elevated armrest, lateral arm support or half tray** protects the flaccid limb. **Anyone spending considerable time in a wheelchair must be referred to a physiotherapist or occupational therapist for a formal seating evaluation.**
18. **Related bed and transfer aids:** overlay mattress cushions, air or sheepskin mattresses, **pillows and leg separator cushions**, **heel pads and bed cradles** in peripheral vascular disease or pressure ulcers; **trapeze, transfer pole or bar, raised quarter- or half-length siderail**; and **bed and toilet seat height matched to the patient's lower-leg length**.

F. Who Prescribes, and Complications

19. **Advice on the suitability of aids is best left to occupational therapists or others with particular expertise. Physiotherapists advise on mobility aids**, speech therapists on communication aids, audiologists on hearing aids and continence advisors on continence aids. **These professionals also provide follow-up and ensure that aids are properly used and maintained.**
20. **Prescription is a process, not an event:** assess → select → **measure and fit → train the patient and carer → check the home environment** (thresholds, rugs, narrow doorways, stairs, Indian-style toilet) → **review use, condition and continued suitability**. **Devices should be recommended only if they improve participation in activities the person and family have identified as important.**
21. **Environmental adaptation must accompany the aid — handrails, ramps, and improved access to shower and toilet areas. Early occupational therapy input, usually including a home visit, ensures modifications are appropriate and timed to facilitate hospital discharge.**
22. **Device-related hazards:** falls from incorrect height or worn ferrules; **tripping over the frame**; **shoulder, wrist and hand overuse pain**; **carpal tunnel syndrome and ulnar neuropathy**; **axillary crutch palsy**; increased energy cost of walking; loss of reciprocal arm swing with frames; **deconditioning and learned dependence**; stigma and non-use; and, for wheelchairs, **pressure ulcers, contractures, shoulder subluxation and functional incontinence.**

Latest Updates (2023–Present)

1. The global access gap has been quantified

The **WHO–UNICEF Global Report on Assistive Technology (2022)** revealed that **more than 2.5 billion people need one or more assistive products**, and that **nearly one billion of them are denied access**, particularly in LMICs, **where access can be as low as 3% of need** versus **90% in some high-income countries**. By **2050 an estimated 3.5 billion people will need assistive technology**. **Most users need more than one product, making integrated services important. Of the 80 million people who need a wheelchair, only 5–35% have access to one depending on the country.**

2. WHO Wheelchair Provision Guidelines (2023) — the new reference standard

WHO estimates that **80 million people, about 1% of the world's population, are likely to require a wheelchair**, a number **set to grow as the global population ages**. The guidelines help Member States **increase access to wheelchairs as a priority product**, supporting the **UNCRPD** and **SDGs 3, 4 and 10**, and **complement the 2008 WHO guidelines on manual wheelchairs in less-resourced settings**, supported by **WHO Training in Assistive Products (TAP)**. Data from the **WHO rapid Assistive Technology Assessment (rATA) across 27 countries** found **1.6% of all participants reported needing a wheelchair, rising to 6.5% among those aged 65 and older**, and that **12.0% had some degree of mobility difficulty**. An appropriate wheelchair is framed **not merely as equipment but as a means of exercising human rights**.

3. The WHO "4P" framework and cost as a policy target

WHO's strategy **places people at the centre and gives equal attention to Policy, Products, Provision and Personnel as interconnected elements that must be aligned to ensure equitable access**. WHO is also Secretariat for the **Global Cooperation on Assistive Technology (GATE)**. **WHA76.6 (May 2023)** specifically commits Member States to **minimise the prohibitive costs of assistive technologies such as hearing aids, glasses, wheelchairs and prosthetics**.
*Exam use: if asked "how would you improve mobility aid provision in your district?", structure the answer as **Policy, Products, Provision, Personnel**.*

4. What mobility aids do *not* do — the falls evidence

The **2023 Cochrane review** found that **assistive devices alone — eyeglasses, specialised footwear, bed alarm systems — and patient education alone do not decrease the rate of falls**, whereas **home fall-hazard assessment and modification reduces falls by 26% overall and 38% in those at higher risk**. **NICE NG249 (April 2025)** found **insufficient evidence on assistive technologies such as footwear, foot devices and self-care and assistive devices** in community settings and issued a **research recommendation**.
*Exam framing: a mobility aid **prevents falls only as part of a package** — exercise, medication review, vision correction and home hazard modification.*

5. Guideline gaps and technology on the horizon

A **2025 scoping review (Frontiers in Rehabilitation Sciences)** searched guidelines published between **January 2008 and March 2024** across five databases and found **only 24 usable guidelines from 291 records**, concluding that **guideline development must accelerate** if assistive technology is to be delivered within UHC. Textbook-flagged directions are maturing: **robotic walking-assist devices** (with potential both to augment recovery in neuromuscular deficit and to assist ADLs including stair walking), **sensor-based gait analysis to select the most appropriate orthosis**, and **robotic gloves combined with virtual reality and arm orthoses that counterbalance limb weight** after hemiparetic stroke. **Smart canes and rollators with fall detection and GPS** are entering practice but lack older-adult validation.

6. India — the operational provision system

The **ADIP Scheme (Assistance to Disabled Persons for Purchase/Fitting of Aids and Appliances)** has operated **since 1981**, providing **durable, scientifically manufactured standard aids and appliances** to promote physical, social and psychological rehabilitation, **arrest the extent of disability and prevent secondary disability**. Grants go to **ALIMCO, National Institutes, Composite Regional Centres, District Disability Rehabilitation Centres, State corporations and NGOs**; the scheme **also funds corrective surgery where required before a device is provided**, and was **last revised with effect from 1 April 2024 and continued to 31 March 2026**.

ALIMCO is the largest manufacturer of mobility and rehabilitation aids in South Asia. Over the seven years to 2020, **ADIP spent about ₹10.8 billion and benefited 1.8 million people, largely through 9460 camps**. ALIMCO is the **implementing agency for ADIP and nodal agency for Rashtriya Vayoshri Yojana**, and has begun opening **Pradhan Mantri Divyasha–Vayoshri Kendras (PMD-VK)**. Under **RVY**, BPL senior citizens with income ≤ ₹15,000/month receive **walking sticks, elbow crutches, walkers, tripods/**

quadpods, wheelchairs, hearing aids, spectacles and dentures free, with **doorstep delivery for those aged ≥80 years**.

Current Practice Update

Issue	Textbook Position	Current Evidence (2023–present)
Framing	"Aids and adaptations" — one step in the rehabilitation process	**Assistive technology is a named entitlement within UHC**; wheelchairs are a **priority product** with dedicated WHO guidelines (2023)
Scale of need	Not quantified	**2.5 billion need assistive products (3.5 billion by 2050); ~1 billion denied access; 80 million need a wheelchair, only 5–35% have one**
Wheelchair need in older people	Not quantified	**6.5% of people aged ≥65 report needing a wheelchair** (WHO rATA)
Do aids prevent falls?	Implied benefit	**Assistive devices alone do NOT reduce falls**; they work only within a multifactorial package
Non-use	"Those in real need often do not obtain even basic aids"	Confirmed and extended — **people who own devices fall more when not using them, and those falls are more injurious**
Who prescribes	OT for aids generally, PT for mobility aids	Unchanged, but the **4P framework** makes **trained personnel an explicit system requirement**, supported by **WHO TAP**
Wheelchair seating	Referral for seating evaluation if prolonged use	Reinforced — poor seating causes pain, agitation, unsafe transfers and restraint use
Technology	Emerging robotics and sensors	Moving into practice; **guideline coverage remains sparse (only 24 AT guidelines in 16 years)**
India	Not covered	**ADIP (revised April 2024) + ALIMCO + RVY + PMD-VK**; **DDRCs** assess and fit devices at district level

Exam One-Liners

- **A mobility aid can foster dependence rather than independence** — prescribe the **least restrictive device that is safe**.
- **Lack of a mobility aid is itself a listed cause of immobility.**
- **Cane length = wrist crease to floor**; handle at the **ulnar styloid/greater trochanter**; **elbow flexed 20–30°**.
- **Cane in the hand opposite the painful hip; for the knee, try either hand; in hemiplegia, the unaffected hand.**
- **Cane–bad leg–good leg** on the flat; **up with the good, down with the bad** on stairs.
- **Axillary crutch pad sits ~2 finger-breadths below the axilla** — never bear weight on the axilla (crutch palsy).
- **A standard frame gives the most stability but abolishes arm swing and cannot be used on stairs.**

- **A rollator needs intact grip, cognition and braking ability** — it can run away downhill.
- **Berg Balance Scale: 0–20 wheelchair-bound, 21–40 walking with assistance, 41–56 independent.**
- **Worn ferrules cause falls** — check and replace at every review.
- **About a quarter of community-dwelling elders use a mobility device**; falls are **not** linked to any particular device.
- **Wheelchairs were designed for transport, not for sitting all day.**
- **Hemiplegic arm in a wheelchair needs an arm trough, elevated armrest or half tray** to prevent shoulder subluxation.
- **Assistive devices alone do not prevent falls.**
- **WHO 4P framework: Policy, Products, Provision, Personnel.**
- **India: ADIP (since 1981, revised 2024) and RVY, delivered through ALIMCO, DDRCs and CRCs.**

Q9. Assistive devices for the elderly

High-Yield Points

A. Definitions

1. **Assistive technology** is the umbrella term for **assistive products together with the systems and services that deliver them.** An **assistive product** is any product that **maintains or improves functioning and independence** — from a pair of spectacles to a powered wheelchair.
2. **Assistive devices reduce activity and participation restriction**, ranging **from the simple and inexpensive to the technologically advanced**, and help **from carrying out ADLs to maintaining mobility and promoting continence.**
3. **Two cardinal cautions: "The provision of an aid is not always the best option for an impaired person, as it can foster dependence rather than independence"**; and **"Those in real need often do not avail themselves of even basic aids and appliances."** Both under-provision and over-provision are errors.
4. **The golden rule of prescription: devices should be recommended only if they support improved participation in the activities identified as crucial to the elder and/or their family or support system.** A device that does not serve a valued activity will be abandoned.

B. Classification — the WHO Six Functional Domains

The WHO Priority Assistive Products List (APL) classifies devices into six functional domains: mobility, vision, hearing, communication, cognition and self-care.

Domain	Devices for the older person
Mobility	**Canes (single-point, quad, tripod), hemi-walker, elbow and axillary crutches, walking frames, rollators, manual and powered wheelchairs, scooters**; transfer aids (**transfer board, trapeze, transfer pole, hoist**); **prostheses and orthoses (AFO, knee brace, foot orthoses)**; **therapeutic footwear, rocker-bottom shoe**, medial arch supports
Vision	**Reading glasses and optimum spectacles**; **hand-held, stand and spectacle-mounted magnifiers; telescopes; electronic/CCTV magnifiers**; **lighting intensifiers without glare**; **tinted glasses**; **night light to aid dark adaptation**; **large-print books and newspapers**; talking watches; **white cane**
Hearing	**Behind-the-ear, in-the-ear and body-worn hearing aids**; **pocket talkers/ personal amplifiers**; **middle-ear and cochlear implants**; **amplified telephones, doorbells and TV listeners**; **closed captioning**
Communication	Communication boards and books, **speech-generating devices**, text-to-speech and voice-amplification apps, writing aids
Cognition	**Pill organisers and automated dispensers, memory aids, calendar clocks, labelled and picture-marked cupboards, medication alarms, GPS locators, simplified telephones**
Self-care	**Long-handled reachers, sock aid, dressing stick, button hook, built-up-handle cutlery, non-slip mats, plate guards**; **raised toilet seat, commode, shower chair, handheld shower, grab bars**; **absorbent continence pads**; **upper-limb splints**; **therapeutic footwear for the diabetic foot**

6. **A prosthesis replaces a body part; an orthosis is applied to the external surface of the body to provide support, improve function, or restrict or promote movement.** Their **design, construction and fitting require particular skill and technological expertise**; well-resourced centres include **prosthetists and orthotists** in the team.
7. **Environmental adaptation is part of the same prescription: handrails and ramps, improved access to shower and toilet areas, additional lighting, grab bars, and removal of loose rugs and clutter. Early occupational therapy input, usually including a home visit, ensures that modifications are appropriate and timed to facilitate hospital discharge.**

C. Domain-Specific Prescribing Points

8. **Vision — simple measures maximise residual vision:** a **magnifying device, large-print reading material, lighting intensifiers and reduction of glare.** Aids must be **combined with training** (eccentric viewing, device use) or they fail.

Hearing aid evaluation — the classic checklist

- **Exclude any contraindicating medical or correctable problem** (e.g. cerumen)
- **Greatest satisfaction if the loss is 55–80 dB; only partial help if the loss exceeds 80 dB**
- **Less satisfaction when speech discrimination is poor**
- The aid is **designed for face-to-face conversation** — set **realistic expectations**
- **May need to be combined with lip reading**
- **Loudness perception abnormalities (recruitment) may make the aid unacceptable**
- **More severe loss requires a body-worn aid** rather than behind-the-ear
- Decide **monaural versus binaural**; assess the **patient's ability to handle the aid independently**; assess **motivation**

10. **Practical hearing aid points: hearing aids are most useful in one-to-one conversation and least useful in noisy group settings. The old adage that hearing aids do not help sensorineural loss is simply not true** — modern digital aids can be **tuned to specific frequencies rather than amplifying all frequencies**. **Binaural aids improve sound localisation and discrimination. Body-worn aids give the greatest amplification and have large controls; in-the-ear aids are cosmetically preferred but harder to manipulate. Patients are often initially resistant — patient and family counselling improves use and satisfaction. Middle-ear and cochlear implants are safe and effective options** when hearing aids fail in severe loss.
11. **Continence:** absorbent pads and washable absorbent products, **bedside commode and urinal to shorten the distance to the toilet**, raised toilet seat and adequate night lighting. **Continence advisors are the correct experts.**
12. **Wheelchair seating is a device issue, not a comfort issue. Wheelchairs were designed for transport, not prolonged sitting; sling-back seats give inadequate support.** Poor back support, a chair **too tall, heavy or wide**, **footrests set too high** and the **hammock effect** cause **pain and agitation — and hence unsafe transfers, falls and restraint use.** Corrections: **wedge cushion, wedge seat, lateral supports**, and in hemiplegia an **arm trough, elevated armrest, lateral arm support or half tray**. **Prolonged users need a formal seating evaluation by a physiotherapist or occupational therapist.**
13. **Falls-related devices — know the evidence limits. Hip protectors** show **uncertain benefit in nursing home residents and no clear benefit in community dwellers**, but **for high-risk older people willing to wear them, they may reduce fear of falling**. **An anti-slip shoe device reduces outdoor falls in icy conditions**, and **compression stockings plus tools to aid slow rising** help in postural hypotension.

D. Who Prescribes, and Why Devices Fail

14. **Advice on the suitability of aids is best left to occupational therapists or others with particular expertise: physiotherapists advise on mobility aids, speech therapists on communication aids, audiologists on hearing aids, and continence advisors on continence aids. These professionals also provide follow-up and ensure that aids are properly used and maintained and continue to serve their purpose.**

15. **The four provision steps are select → fit → use (train) → follow-up. Without all four, assistive products are often of no benefit, are abandoned, or may cause physical harm.** *This one line answers most exam questions on device failure.*
16. **Common causes of device abandonment:** wrong device or wrong size; no training; **cosmetic stigma**; cost; the device not serving a valued activity; **cognitive or dexterity failure** (hearing aid batteries, walker brakes); lack of maintenance; and **carer over-help that removes the reason to use it.**
17. **Device-related complications:** falls from **worn ferrules** or wrong height; **carpal tunnel syndrome, ulnar neuropathy and shoulder pain**; **axillary crutch palsy**; **pressure ulcers, contractures and functional incontinence** from prolonged wheelchair use; **skin damage from pads**; **otitis externa and cerumen impaction** from hearing aid moulds; and **learned dependence and deconditioning.**
18. **The over-help trap:** if caregivers bathe and dress an older person who has independence goals, **those goals will not be achieved** — "helping" creates **deconditioning, dependency, contractures and the other sequelae of immobility.** A **restorative philosophy must accompany every device.**

E. Integration into Geriatric Care

19. **The WHO ICOPE pathway builds assistive devices into intrinsic-capacity management: provide an assistive device to aid mobility; reading glasses and vision rehabilitation for irreversible low vision; hearing aids and auditory aids around the house (telephone, doorbells); home adaptation to prevent falls. Treating visual impairment enables maintaining family and social relationships, accessing information, moving safely and performing manual tasks.**
20. **Emerging technology: remote sensors and accelerometers for objective progress measurement**, **virtual reality platforms** giving visual, auditory and tactile input, a **robotic glove combined with VR attached to an arm orthosis that counterbalances arm weight** after hemiparetic stroke, and **walking assist devices with potential to augment recovery and assist ADLs including stair walking.** Sensor-based gait analysis can **indicate the most appropriate orthotic device to prescribe.**

Latest Updates (2023–Present)

> **1. The global access gap**
>
> The **WHO–UNICEF Global Report on Assistive Technology (2022)** found that **more than 2.5 billion people need one or more assistive products**, yet **nearly one billion are denied access**, with access **as low as 3% of need in some low-income countries versus up to 90% in some high-income countries**. **An estimated 3.5 billion people will need assistive technology by 2050**, driven by **population ageing and rising non-communicable disease**. **Timely provision of assistive technology for older people improves independence and safety and enables them to live at home for as long as possible.**

> **2. The Priority Assistive Products List and its ongoing update**
>
> WHO introduced the **Priority Assistive Product List (APL) in 2016**, featuring **50 crucial products across mobility, vision, hearing, communication, cognition and self-care**, followed by the **Assistive Product Specifications (APS) in 2021** and the **Training in**

Assistive Products (TAP) programme in 2022. The APL is **modelled on the WHO Model List of Essential Medicines**. On **6–7 May 2025 WHO convened the first in-person meeting of the Technical Advisory Group on Assistive Technology in Geneva — 24 independent experts from all six WHO regions** — who **assessed and prioritised 300 assistive products grouped by the six functional domains** for an **updated APL**. **Since 2016, 23 countries have adopted national lists based on the WHO model.**

3. The workforce is the bottleneck — and the four provision steps

Only about 1 in 10 people worldwide have access to appropriate assistive products, and **workforce shortages are one of the biggest reasons**. WHO states that **trained personnel are essential for the proper assessment, fitting, user training and follow-up**, and that **without these four key steps assistive products are often of no benefit or abandoned, and may cause physical harm. WHO TAP** covers the provision steps — **select, fit, use, follow-up** — for **25 basic products from the APL across cognition, communication, vision, hearing, continence and mobility**, is **open-access in 10 languages**, and has been deployed in **Tanzania, Ukraine, India, Iraq, Papua New Guinea and Romania**.

4. Hearing aids and cognition — the ACHIEVE trial (Lancet 2023)

ACHIEVE randomised **977 adults aged 70–84 with untreated mild-to-moderate hearing loss and without substantial cognitive impairment** at four US sites to **hearing intervention (audiological counselling plus hearing aids)** or **health education control**, with **3-year change in global cognition** as the primary endpoint. **The result was negative in the total study population**, **but in the pre-specified subgroup of 238 participants drawn from the ARIC cardiovascular cohort — at increased risk of cognitive decline — hearing intervention slowed cognitive decline by 48% over three years. Hearing loss is present in 65% of adults over 60.**
*Exam framing: hearing aids are **not proven to prevent dementia in everyone**, but are a **reasonable intervention in the at-risk older adult** — and remain unequivocally indicated for communication, mood and social participation.*

5. Assistive devices alone do not prevent falls

The **2023 Cochrane review** found that **assistive devices alone — eyeglasses, specialised footwear, bed alarm systems — and education alone do not reduce the rate of falls**, whereas **home fall-hazard assessment and modification reduces falls by 26% overall and 38% in high-risk older people**. **NICE NG249 (2025)** found **insufficient evidence on assistive technologies in community settings** and issued a formal **research recommendation**.

6. Policy anchors and Indian provision

WHA71.8 (2018) committed Member States to improving access to assistive technology; **WHA76.6 (2023)** calls for integrating rehabilitation into health systems within UHC and specifically commits to **minimising the prohibitive costs of assistive technologies such as hearing aids, glasses, wheelchairs and prosthetics**. WHO's strategy uses the **4P framework — Policy, Products, Provision and Personnel**.

India: the **ADIP Scheme** (since **1981**, **revised with effect from 1 April 2024** and

continued to 31 March 2026) provides **durable, scientifically manufactured standard aids and appliances** and **funds corrective surgery where needed before a device is provided**, with grants to **ALIMCO, National Institutes, Composite Regional Centres, DDRCs, State corporations and NGOs**. **ALIMCO is the largest manufacturer of mobility and rehabilitation aids in South Asia**; over seven years to 2020, **ADIP spent about ₹10.8 billion and benefited 1.8 million people through 9460 camps**, and ALIMCO is opening **Pradhan Mantri Divyasha-Vayoshri Kendras (PMD-VK)**. **Rashtriya Vayoshri Yojana** supplies **free walking sticks, elbow crutches, walkers, tripods/ quadpods, hearing aids, wheelchairs, artificial dentures and spectacles** to **BPL senior citizens with income ≤ ₹15,000/month**, with **doorstep delivery for those aged ≥80 years**. **DDRCs, now approved in every district**, assess need, **fit and repair assistive devices**, and provide **physiotherapy, occupational therapy and speech therapy**.

Current Practice Update

Issue	Textbook Position	Current Evidence (2023-present)
Framing	"Aids and adaptations" — a step in the rehabilitation process	**Assistive technology is an entitlement within UHC**; the **APL is modelled on the Essential Medicines List**
Classification	Descriptive by need	**Six WHO functional domains: mobility, vision, hearing, communication, cognition, self-care**
Scale of need	Not quantified	**2.5 billion now, 3.5 billion by 2050; ~1 billion denied access; only 1 in 10 have appropriate products**
Why devices fail	"Aids may foster dependence"; "many in need do not obtain them"	Reframed as the **four provision steps — select, fit, use, follow-up**; omit any one and the device is abandoned or causes harm
Workforce	OT/PT/audiologist/continence advisor advise	**Workforce shortage named as the single biggest barrier**; **WHO TAP** trains primary care and community workers for 25 basic products
Hearing aids and cognition	Not addressed	**ACHIEVE (Lancet 2023): no effect overall, but 48% slower cognitive decline over 3 years in the at-risk subgroup**
Devices and falls	Aids implied to help	**Devices alone do not reduce falls** — benefit comes from the **package**
Wheelchairs	Seating evaluation if prolonged use	**WHO Wheelchair Provision Guidelines (2023)**; **6.5% of people ≥65 need a wheelchair**; only **5–35%** of those needing one have one
Priority product list	Not addressed	**APL under formal revision — 300 products reviewed by the WHO Technical Advisory Group in May 2025**

Exam One-Liners

- **Assistive technology = assistive products + the systems and services that deliver them.**

- **WHO APL (2016): 50 priority products across six domains** — mobility, vision, hearing, communication, cognition, self-care. Under revision; **300 products reviewed in May 2025**.
- **The four provision steps: select, fit, use, follow-up.** Miss one and the device is **abandoned or harmful**.
- **An aid can foster dependence rather than independence** — but **those in real need often never obtain even basic aids**.
- **Prescribe a device only if it serves an activity the person and family value.**
- **Prosthesis replaces a body part; orthosis supports, improves function, or restricts/promotes movement.**
- **Hearing aid: best satisfaction at 55–80 dB loss; only partial help above 80 dB; poor discrimination predicts poor satisfaction.**
- **Hearing aids work best one-to-one and worst in noisy groups.**
- **Binaural aids improve sound localisation and discrimination.**
- **ACHIEVE (2023): 48% slower cognitive decline with hearing aids in the at-risk subgroup only.**
- **Vision aids: magnifier, lighting intensifiers without glare, tinted glasses, night light, large print** — always with training.
- **Self-care classics: long-handled reacher, sock aid, dressing stick, built-up cutlery, raised toilet seat, shower chair, grab bars.**
- **Assistive devices alone do not prevent falls.**
- **WHO 4P framework: Policy, Products, Provision, Personnel.**
- **India: ADIP (since 1981, revised 2024), ALIMCO, Rashtriya Vayoshri Yojana, PMD-VK and DDRCs.**

Q10. Artificial limb — rehabilitation of the elderly amputee

High-Yield Points

A. Definition

1. **A prosthesis is a device that replaces a missing body part**, in contrast to an **orthosis**, which is applied to the external surface of the body to **provide support, improve function, or restrict or promote movement.** Their **design, construction and fitting require particular skill and technological expertise**, and well-resourced rehabilitation centres include **prosthetists and orthotists** in the multidisciplinary team.

B. Aetiology and Epidemiology

2. **Most lower-limb amputations in older people are a consequence of peripheral vascular disease.** Limb-threatening ischaemia is only occasionally due to a sudden embolic event; **most patients have a long period of worsening ischaemia before amputation. Many are diabetic with coexisting cardiac and vascular disease**, and others have **smoking-induced chronic lung disease.**
3. **Despite this, rehabilitation has much to offer older amputees, and many rise to the challenge of walking even with bilateral below-knee prostheses.**
4. **Elderly amputees are nonetheless a high-risk group: perioperative mortality 10–30%, two-year survival 40–50%, five-year survival 30–40%** — and **these rates have not changed significantly in the past 50 years despite better anaesthetic and surgical techniques.**

C. Level of Amputation — the critical surgical decision

5. **Preservation of the knee is critical** for **proprioceptive and neuromuscular control** and above all for **minimising energy expenditure**. **It takes 40% more energy to walk with an above-knee (transfemoral) than with a below-knee (transtibial) prosthesis.**
6. **But do not over-reach for the knee. Injudicious efforts to salvage a knee joint can produce an ischaemic stump that fails to heal and later requires a more proximal amputation. A second procedure is potentially disastrous — it increases anaesthetic risk, prolongs immobility, increases deconditioning and delays entry into rehabilitation.**

D. Pre-Prosthetic Phase

7. **Stump management** uses **rigid removable dressings** to **reduce oedema, promote healing and protect against incidental trauma.**
8. **Early physiotherapy must strengthen the arms, abdominal muscles, lower back and the remaining leg** — not merely the stump, because transfers and frame work depend on the upper body.
9. **Prevent contracture from day one. For a below-knee amputee the full range of knee extension must be maintained** — therefore **avoid prolonged sitting without corrective exercises**, and perform **a minimum of 20–30 minutes of prone lying twice daily** to promote full extension. In transfemoral amputees, guard against **hip flexion and abduction contracture**.

10. **Stump conditioning: the skin contacting the prosthesis must become durable and toughened, best achieved by graded use of the prosthesis. Massaging the stump improves circulation and prevents adhesions during healing**, and patients should be encouraged to do it themselves.

E. Selecting the Patient for a Prosthesis

11. **“Irrespective of age, prescription of a lower limb prosthesis is almost always indicated, even in the presence of other major medical problems.”** Yet **older amputees are less likely than younger ones to receive a prosthesis, even after adjusting for comorbidity and functional status** — an ageism issue.
12. **The prosthesis facilitates transfers, standing and walking, and has cosmetic value** — the transfer benefit alone justifies fitting in many frail patients.
13. **The old criterion is invalid.** It used to be argued that an older amputee should demonstrate **crutch-walking capacity before being offered a prosthesis. However, walking without bearing weight on the amputated side has a higher energy cost than walking on the prosthesis, so this criterion is invalid.**
14. **Respect patient choice. Some refuse a prosthesis and accept wheelchair mobility, but most older people — particularly those with few comorbidities — are happy with their prosthesis and use it well. A prosthesis should never be prescribed without considering each patient’s unique needs and wishes, and the demands of using it must be fully explained.**

F. Components of the Prosthesis

A modern trans-tibial prosthesis has three parts: a socket, a shank, and an ankle-and-foot mechanism.

Component	Key facts
Socket	**The major determinant of comfort and stability.** Designed to **transfer most of the weight onto the patellar tendon**; **a good fit is critical.** The stump is **oedematous early and shrinks over time**, so **temporary sockets are needed until shrinkage is complete before a permanent socket is cast. Plastic laminate is the commonest material.** May incorporate a **suction device for suspension**; with a **non-suction socket an interface (stump socks or resilient plastic) is required**
Shank	**Exoskeletal** — traditional, willow or lightweight balsa wood covered with laminated plastic. **Endoskeletal** — modern, a **central pillar of carbon fibre or lightweight metal** bearing body weight, surrounded by a **soft cover approximating the feel of a normal limb**
Ankle–foot	**Rigid, or an ankle allowing movement in one or more planes.** Prescription depends on **the level of activity and the condition of the stump**; **there is no evidence that any one design is inherently superior. Advantages of a rigid ankle: lightness, low initial and maintenance cost, easy fitting, good appearance.** The **SACH (solid ankle–cushion heel) foot** has a **rigid ankle, compressible heel and light foot**, is **particularly suited to frail, less active patients who do not take long steps**, gives **long service**, and is **almost always used for below-knee limbs**

16. **Prosthesis hygiene: stump socks should be washed daily with mild soap and warm water, rinsed thoroughly and dried flat**, and **the inner surface of the socket cleaned each evening with a warm soapy cloth.**

G. Gait Training and Protecting the Remaining Limb

17. **Once a comfortable, stable and functional limb has been provided, the next stage is teaching the patient to walk on it properly** — progressive weight transfer in parallel bars, then frame, then sticks.
18. **Protect the remaining limb — it is the one that matters most.** When peripheral vascular disease causes amputation, **the remaining limb is often significantly ischaemic: 15–20% undergo contralateral amputation within two years and about 40% within four years. Viability can be enhanced by revascularisation surgery and by minimising vascular risk factors (poor diabetic control, smoking). Promote foot hygiene and avoid trauma, particularly by wearing appropriate footwear.**
19. **Comprehensive rehabilitation involves more than the provision of a prosthesis. Comorbidity, prosthetic component selection, and resettlement with tenuous or absent social supports all present formidable challenges.**

H. Phantom Limb Phenomena

20. **Phantom limb sensation is normal and non-painful after amputation.** Initially it can be **so deceptive that the patient inadvertently attempts to walk on or scratch the missing limb. Over time the limb is felt to retract or "telescope" into the stump.**
21. **Phantom pain is a separate but perhaps related phenomenon affecting 50–80% of amputees.** Pathophysiology is poorly understood, with **central and peripheral nervous system factors plus psychological factors** implicated. **It is distinguished from stump pain because it is localised in the space vacated by the amputation**, and is described as **burning, crushing or lancinating**; it may be **continuous or intermittent**, and the limb may be **perceived as twisted or deformed.**
22. **Management of phantom pain is difficult.** The first step is **explaining the nature of the phenomenon to the patient. Patients with chronic pain before amputation have a higher incidence of phantom pain, but attempts to control pain before and during surgery do not consistently reduce it. There is little RCT evidence; reported improvement rates are little better than placebo. Anaesthetic and surgical techniques (local anaesthesia, sympathectomy, cordotomy) are as disappointing as pharmacological approaches. Tricyclic antidepressants and sodium-channel blockers are often used for their efficacy in neuropathic pain but are of no proven benefit. TENS provides modest relief at best**, and patients need **ongoing psychological help to develop coping strategies.**
23. **Nutrition detail:** when calculating BMI in an amputee, add back the missing body-part percentage — **single below-knee 6.0%, single at-knee 9.0%, single above-knee 15.0%, single arm 6.5%, single arm below elbow 3.6%.**

Latest Updates (2023–Present)

1. VA/DoD Clinical Practice Guideline for Rehabilitation of Individuals with Lower Limb Amputation (2024)

This **2024 update of the 2017 guideline** directly modifies textbook practice:

- **"For prosthetic ambulators, we suggest prescribing microprocessor knee units over non-microprocessor knee units for reducing falls, optimizing functional mobility and improving patient satisfaction"** (Weak for).

- **“We suggest energy storing and return (ESAR) or microprocessor-controlled foot and ankle components over solid ankle cushioned heel (SACH) feet to improve ambulation and patient satisfaction”** (Weak for) — though **there is insufficient evidence to prefer any specific ESAR or microprocessor foot over another.**
- **“For patients with transfemoral amputation who meet eligibility criteria, we suggest osseointegration as an option to improve prosthesis use”** (Weak for).
- **“We suggest intraoperative placement of a perineural catheter for postoperative delivery of local anaesthetic to reduce pain following amputation surgery.”**
- **Insufficient evidence for or against targeted muscle reinnervation** or other peripheral nerve surgery for phantom limb pain, and **insufficient evidence for or against neurostimulation** (peripheral nerve or spinal cord stimulation) **or neuroablation** (cryoneurolysis, radiofrequency ablation).
- **“We suggest using patient-reported and performance-based measures with acceptable psychometric properties to assess function”** (Weak for).

2. Osseointegration — promising but not for the typical elderly amputee

Osseointegration is **a procedure in which a metal implant is directly anchored to the residual bone and attached to a prosthetic limb by a transcutaneous connector**, an alternative to the socket-mounted prosthesis that has gained acceptance over the past 30 years. The VA/DoD **“weak for” recommendation rests on very low confidence in the evidence quality**, and on the fact that **many clinicians are unfamiliar with the procedure because of limited access and training**; the **2022 VA/DoD upper limb guideline found insufficient evidence** either way. Crucially, **candidates should not have diabetes, peripheral vascular disease or metabolic disease** — which **excludes the majority of elderly dysvascular amputees**. A **consensus statement on prehabilitation and rehabilitation of osseointegration patients** was published in **2025**, noting that **protocols vary by implant system and institution**.

3. Updated physiotherapy guidance and health economics

The **British Association of Chartered Physiotherapists in Limb Absence Rehabilitation (BACPAR)** published the **3rd edition of its “Clinical guidelines for the pre- and post-operative physiotherapy management of adults with lower limb amputations” in 2025**, incorporating new evidence and a **2024 Delphi consensus**. A **2025 exchange in the Canadian Prosthetics & Orthotics Journal** on the **health economic evaluation of microprocessor versus non-microprocessor knees** underlines that **real-world cost-effectiveness data now drive reimbursement decisions** — the practical barrier to microprocessor knees in most health systems, including India, is **cost, not evidence**.

4. Functional classification now drives component prescription (Medicare K-levels)

K0 — no ability or potential to ambulate or transfer safely; a prosthesis does not enhance mobility
K1 — transfers and ambulation on level surfaces at fixed cadence (household ambulator) → **SACH or single-axis foot**
K2 — limited community ambulator, can negotiate low-level barriers (kerbs, stairs, uneven ground) → **multi-axial or flexible-keel foot; increasing evidence for microprocessor knees**
K3 — community ambulator with variable cadence → **ESAR foot, microprocessor or hydraulic knee**
K4 — high impact; child, athlete or active adult

Most elderly dysvascular amputees are K1–K2, and the current shift is that **K2 patients now benefit from components once reserved for K3**.

5. India — the artificial limb landscape

ALIMCO is India's largest government-run prosthetic manufacturer, producing affordable artificial limbs, mobility aids and assistive devices distributed **free or subsidised under the ADIP Scheme**.

BMVSS (Bhagwan Mahaveer Viklang Sahayata Samiti), the "Jaipur Foot" organisation, completed 50 years in 2025. It **rehabilitated 88,467 people in 2022–23 and fitted 40,078 limbs in 2024–25**, and has provided **over 2.5 million artificial limbs, calipers and other aids free of charge across India and 27 other countries**.

The **Jaipur Limb uses an HDPE polymer custom-made socket linked to the specially designed Jaipur Foot, which is distinct from the SACH foot used in Western technology.** The **Jaipur Knee, developed with Stanford University**, is generally added for above-knee amputees. **A below-knee amputee with a Jaipur Foot can walk on even or uneven ground, run, sit, squat, climb, swim and dance** — an **above-knee amputee retains most of these functions**. It is a **rapid-fit limb, fabricated and fitted mostly within one or two days**, **light, durable and provided completely free**, with **no prior appointment needed** (Aadhaar card and disability certificate required), including at **on-the-spot fitment camps**. The **Jaipur/Stanford knee costs about US$20 to manufacture in India**.

In India, **around 45,000 legs are estimated to be amputated every year**, with major risk factors for diabetic amputation being **lower-extremity ischaemia, neuropathy, previous foot ulcer, elevated HbA1c and retinopathy**.

*Exam relevance: the **ability to squat and sit cross-legged is the culturally decisive functional outcome in India**, and this is precisely where the Jaipur Foot outperforms the SACH foot.*

Current Practice Update

Issue	Textbook Position	Current Evidence (2023–present)
Prosthetic foot	**SACH foot is almost always used for below-knee limbs**; no design inherently superior	**VA/DoD 2024 suggests ESAR or microprocessor feet over SACH** — SACH remains reasonable for **K1 household ambulators and low-resource settings**
Prosthetic knee	Not detailed	**Microprocessor knees suggested over non-microprocessor knees** to **reduce falls** and improve mobility and satisfaction — cost is the limiting factor
Attachment method	Socket-based only	**Osseointegration is a "weak for" option in selected transfemoral amputees** — but **excluded in diabetes and peripheral vascular disease**
Perioperative pain	Pre-emptive analgesia does not consistently prevent phantom pain	**Intraoperative perineural catheter** for postoperative local anaesthetic is now suggested to reduce post-amputation pain
Phantom pain drugs	TCAs and sodium-channel blockers of no proven benefit; TENS modest at best	Essentially unchanged; **insufficient evidence for targeted muscle reinnervation, neurostimulation or neuroablation**
Patient selection	Prosthesis "almost always indicated irrespective of age"; crutch-walking criterion invalid	Reinforced; **K-level functional classification** now guides *which components*, not *whether* to fit
Outcome measurement	Not specified	**Validated patient-reported and performance-based outcome measures now recommended**
Physiotherapy protocol	General principles	**BACPAR 3rd edition (2025)** provides updated pre- and post-operative guidance
India	Not covered	**ALIMCO/ADIP and BMVSS Jaipur Foot** — free, rapid-fit, squat-capable limbs; **~45,000 amputations/year**

Exam One-Liners

- **Prosthesis replaces a body part; orthosis supports, improves function, or restricts/promotes movement.**
- **Peripheral vascular disease is the commonest cause of lower-limb amputation in the elderly.**
- **Perioperative mortality 10–30%; 2-year survival 40–50%; 5-year survival 30–40%** — unchanged for 50 years.
- **Above-knee walking costs 40% more energy than below-knee** — save the knee where healing allows.
- **A failed knee salvage requiring re-amputation is disastrous.**
- **Rigid removable dressings** reduce oedema, promote healing and protect the stump.
- **Prone lying 20–30 minutes twice daily** prevents knee flexion contracture in the trans-tibial amputee.

- **Early physiotherapy targets the arms, abdomen, back and remaining leg.**
- **The crutch-walking prerequisite is invalid** — walking without weight-bearing costs more energy than walking on the prosthesis.
- **Three components of a trans-tibial prosthesis: socket, shank, ankle-foot.**
- **The socket determines comfort and stability** and loads the **patellar tendon**; **temporary sockets are needed until stump shrinkage is complete.**
- **Exoskeletal shank = willow/balsa with laminated plastic; endoskeletal = carbon-fibre central pillar with a soft cover.**
- **SACH foot = rigid ankle, compressible heel, light** — ideal for the **frail, short-stepping patient**.
- **Wash stump socks daily and dry flat; clean the socket interior nightly.**
- **15–20% lose the other leg within 2 years and ~40% within 4 years.**
- **Phantom sensation is normal and non-painful; the limb "telescopes" over time.**
- **Phantom pain affects 50–80%; it is felt in the space the limb occupied**, unlike stump pain.
- **VA/DoD 2024: microprocessor knees over non-microprocessor knees; ESAR/microprocessor feet over SACH.**
- **Osseointegration is contraindicated in diabetes and PVD.**
- **India: Jaipur Foot allows squatting, sitting cross-legged and walking on uneven ground.**

Master Revision Sheet — Numbers to Memorise

Topic	Number
Global rehabilitation need	**2.41 billion** (one-third of the world); **+63% since 1990**; **>50% unmet in LMICs**
Assistive technology need	**2.5 billion now; 3.5 billion by 2050; ~1 billion denied access; only 1 in 10 have appropriate products**
Wheelchairs	**80 million need one; only 5–35% have one; 6.5% of people ≥65 report needing one**
Home fall-hazard modification	**26% fall reduction overall; 38% in high-risk people** (Cochrane 2023)
Stroke therapy dose	**≥3 hours/day on ≥5 days/week** (NICE NG236, 2023)
Stroke recovery timeline	Most return in **month 1**; little after **month 3**; CIMT works at **3–9 months**
Knee OA in elderly Indians	**~47%** pooled prevalence
STEP 9 (semaglutide, knee OA)	Weight **−13.7% vs −3.2%**; WOMAC pain **−41.7 vs −27.5**
GLA:D programme dose	**12 supervised sessions over 6 weeks** + 2–3 education sessions
ACHIEVE (hearing aids)	**48% slower cognitive decline** in the at-risk subgroup only
Hearing aid satisfaction	Best at **55–80 dB** loss; partial help **>80 dB**
Blindness in India (≥50 years)	**1.99%**; cataract **66.2%**; **~93% avoidable**
Amputee survival	Perioperative mortality **10–30%**; 2-year **40–50%**; 5-year **30–40%**
Above-knee energy cost	**40% more** than below-knee
Contralateral amputation	**15–20% at 2 years; ~40% at 4 years**
Phantom pain	**50–80%** of amputees
Berg Balance Scale	**0–20** wheelchair; **21–40** aided walking; **41–56** independent
Team size	**4–7** members
LASI wave-1 (India, ≥60 y)	ADL dependency **7.4%**; vision **13.7%**; hearing **9.6%**; cognition **9.3%**; falls **5.7%**; incontinence **4.0%**
Institutional care	**0.1–1% in LMICs** vs **7.5% in Switzerland**
India amputations	**~45,000 legs per year**

MD GERIATRIC MEDICINE

Infectious Diseases & Respiratory Medicine

Sepsis • Septic Shock • COPD • Oxygen Therapy • CPAP • MDR-TB • Malaria

High-Yield 15-Mark Answers • Textbook-anchored • Updated to August 2026

Contents

1. Sepsis in the older person — definition, epidemiology, presentation, diagnosis, management, prognosis
2. Septic shock, SIRS and MODS — etiopathogenesis, investigations, treatment
3. Recent advances in the management of sepsis
4. COPD management — recent recommendations (GOLD 2026)
5. Tailoring oxygen requirements and delivery systems in COPD with severe hypoxaemia and infective exacerbation
6. Role of CPAP in diseases of the elderly
7. Diagnosis and management of multidrug-resistant tuberculosis (DOTS-Plus / PMDT)
8. Malaria — treatment, drug classes, contraindications, side effects, efficacy and prophylaxis

Sourcing note. Questions 1–6 are anchored in *Pathy's Principles and Practice of Geriatric Medicine, 6th ed.* (Ch. 39 Pneumonia, Ch. 41 COVID-19, Ch. 42 COPD & Asthma, Ch. 45 Sleep Apnoea, Ch. 23 DIC) and *Essentials of Clinical Geriatrics, 7th ed.* (Ch. 12 Infections in Older Adults). The formal **Sepsis-3 definitions, SIRS/SOFA criteria and MODS** are not set out in either uploaded textbook; these, together with **MDR-TB regimens (Q7)** and **malaria chemotherapy (Q8)**, which are also not covered in the uploaded texts, have been drawn from WHO, SCCM/ESICM, GOLD, BTS, NTEP-India and peer-reviewed journals, as flagged at the head of the relevant answers.

Q1. Define sepsis. Discuss its epidemiology, clinical presentation, diagnosis, management and prognosis in the older person.

Textbook coverage: Pathy's and Essentials describe infection in old age (altered presentation, blunted fever, gram-negative predominance, atypical features) and sepsis as a complication of pneumonia, biliary disease and DIC, but do **not** give the formal consensus definition. The definition below is Sepsis-3 (JAMA 2016), retained by the Surviving Sepsis Campaign 2026.

15–20 High-Yield Points

Definition

1. **Sepsis** is **life-threatening organ dysfunction caused by a dysregulated host response to infection** (Sepsis-3, 2016). Organ dysfunction = an acute rise in the **SOFA score by 2 or more points**.
2. **Septic shock** = sepsis with **persisting hypotension requiring vasopressors to keep MAP ≥ 65 mmHg** plus **lactate > 2 mmol/L** after adequate fluid resuscitation. The old terms *severe sepsis* and *septicaemia* are obsolete.

Epidemiology

3. Roughly **49 million cases and 11–13 million sepsis-related deaths** occur worldwide each year; it is a leading cause of death globally (SSC 2026).
4. **Older adults carry most of the burden** — those aged ≥ 65 years account for about **60–85% of all sepsis cases** and the incidence rises roughly **100-fold** from young adulthood to the ninth decade.
5. In India, sepsis is the commonest reason for ICU admission; reported ICU mortality from severe sepsis is **34–58%**, higher than Western cohorts, driven by late presentation, **multidrug-resistant gram-negative organisms** and limited critical-care access.
6. **Predisposing factors in the elderly:** immunosenescence and inflammaging, diminished cell-mediated immunity, protein–energy malnutrition, multimorbidity (diabetes, heart failure, CKD, COPD, dementia), **prostatic obstruction**, swallowing disorders and aspiration, pressure ulcers, indwelling catheters and institutional residence.
7. **Commonest sources:** respiratory tract (pneumonia is the single largest source), **urinary tract**, skin and soft tissue, intra-abdominal/biliary (acute cholecystitis, cholangitis), and device-related infection. **Gram-negative bacilli are more frequent at every site** than in the young.

Clinical Presentation

8. **Presentation is atypical and non-specific.** The **febrile response is blunted or absent in up to 30–50%**; **hypothermia** is an ominous sign, and **absence of leucocytosis does not exclude bacterial infection**.
9. **Delirium or acute confusion is frequently the only presenting feature**, especially in pre-existing dementia. Other geriatric presentations: falls, being ‘off legs’, new incontinence, anorexia, tachypnoea and unexplained functional decline.
10. A textbook axiom: **acute unexplained functional deterioration should alert the physician to an acute infectious process**, and one should **suspect infection (including TB) whenever an older patient is inexplicably failing**.
11. **Tachypnoea (RR > 20–22/min) is the earliest and most reliable sign**; hypotension is a late sign because of stiff vasculature and pre-existing hypertension — a ‘normal’ BP of 110/70 may represent shock in a chronic hypertensive.

Diagnosis and Investigations

12. Sepsis is a **clinical diagnosis** and **must not be ruled in or out on a single biomarker**. Screen with **NEWS/NEWS2, MEWS or SIRS criteria**; calculate **SOFA** in the ICU.
13. **Baseline work-up:** haemogram with differential, **serum lactate**, CRP ± procalcitonin, blood urea and creatinine (mg/dL), electrolytes, liver function, coagulation profile and D-dimer (DIC screen), arterial blood gas, blood sugar.
14. **Two sets of blood cultures before antibiotics**, plus site-directed cultures (urine, sputum, pus, CSF), chest radiograph, ultrasound abdomen/CT for source, and echocardiography if endocarditis is suspected.
15. **Key geriatric pitfall: asymptomatic bacteriuria is very common** in older people — a positive urine culture alone must never be accepted as the source, or a genuine pneumonia or intra-abdominal source will be missed.

Management

16. **Hour-1 bundle:** measure lactate, take blood cultures, give **broad-spectrum antibiotics immediately (ideally within 1 hour)** in septic shock or probable sepsis, start **30 mL/kg balanced crystalloid** for hypotension or lactate ≥ 4 mmol/L, and add **vasopressors** if hypotension persists.

17. **Antibiotics:** cover gram-negatives empirically (third-generation cephalosporin ± aminoglycoside per the textbook; locally, a beta-lactam/beta-lactamase inhibitor or carbapenem where ESBL prevalence is high). **Dose-adjust for renal function** — sarcopenia falsely lowers creatinine, so estimate GFR (Cockcroft–Gault) rather than trusting creatinine, and monitor aminoglycoside levels. De-escalate at 48–72 hours.
18. **Haemodynamics: noradrenaline is first-line**; add vasopressin if doses escalate, then adrenaline; add **IV hydrocortisone** in shock requiring ongoing vasopressors. Fluids must be given cautiously and reassessed — reduced cardiac and renal reserve makes the elderly prone to pulmonary oedema; use dynamic measures (passive leg raise, stroke-volume variation) and capillary refill rather than large fixed boluses.
19. **Source control within 6 hours** — drain abscess, remove infected catheter/line, decompress obstructed biliary or urinary tract, debride necrotic tissue.
20. **Geriatric supportive care bundle:** delirium prevention (orientation, sleep, avoid anticholinergics/benzodiazepines), **LMWH thromboprophylaxis**, PPI stress-ulcer prophylaxis if risk factors, **early enteral nutrition within 72 h**, early mobilisation and physiotherapy, oral care, pressure-ulcer prevention, glycaemic control (start insulin at glucose ≥ 180 mg/dL) and medication review with withdrawal of nephrotoxins.
21. **Goals of care must be addressed early (within 72 hours)**, framed by Pathy's functional categorisation (**robust / frail / moderately demented / end of life**): decide ceilings of treatment, escalation to ICU, resuscitation status and time-limited trials, incorporating advance directives (in India, valid under *Common Cause* 2018 as modified in 2023).

Prognosis

22. **Mortality rises steeply with age** — roughly 1.5–2 times that of younger patients for the same severity; but **frailty, baseline functional status and comorbidity predict outcome better than chronological age**, so age alone must never be used to deny critical care.
23. **Post-sepsis syndrome** is the rule in survivors: new physical disability, **accelerated cognitive decline**, depression and anxiety, sarcopenia, high 30-day readmission and a **one-year mortality of 30–40%**. Institutionalisation after the illness is common.

Current Practice Update

Textbook Position (Pathy's 6th ed. / Essentials 7th ed.)	Current Evidence & Guidelines (2023–2026)
Sepsis discussed descriptively as a complication of pneumonia, cholecystitis and DIC; no formal definition given.	**Sepsis-3 definition** (infection + SOFA rise ≥ 2) is retained by the **SSC 2026 guidelines**; 'severe sepsis' is abandoned.
Blunted fever and absent leucocytosis emphasised as diagnostic traps — still fully valid.	Unchanged, and reinforced: SSC 2026 states sepsis **cannot be ruled in or out by any single biomarker or test**.
Empirical therapy: 'consider a third-generation cephalosporin and/or aminoglycoside because gram-negative infection is commoner'.	Empirical cover is now **risk-stratified**: cover MDR pathogens only if specific risk factors exist; **routine antifungal and routine anaerobic cover are advised against**.
Aminoglycoside levels and renal function must be monitored.	Retained; SSC 2026 adds **therapeutic drug monitoring on a case-by-case basis** and **prolonged (extended) infusion of beta-lactams after a loading dose (strong recommendation)**.
Fluid resuscitation not addressed in detail for the older patient.	**Balanced crystalloids over 0.9% saline**; 30 mL/kg over 3 h with individualisation; **starches and gelatins are not to be used**; dynamic measures preferred.
qSOFA had been widely taught as the screening tool of choice.	SSC 2026 **strongly recommends NEWS, NEWS2, MEWS or SIRS over qSOFA** as the single screening tool in hospital.
MAP target not specified.	MAP target **65 mmHg** generally, but in patients **aged 65 years and over an initial MAP of 60–65 mmHg** is suggested — a directly geriatric recommendation.
Vitamin C, immunoglobulin and blood purification not addressed.	SSC 2026 **suggests against IV vitamin C, IV immunoglobulin, vitamin D, blood purification and polymyxin B haemoperfusion**.

Latest Updates (2023–Present)

- **Surviving Sepsis Campaign 2026 guidelines** (*Crit Care Med*, March 2026) — 129 statements, **46 entirely new**, from a 69-member panel across 23 countries with 38% from low- and middle-income settings.

- **Tiered antibiotic timing** (new): **within 1 hour** for septic shock and for probable/definite sepsis; for **possible** sepsis without shock, a time-limited period of rapid investigation with antibiotics **within 3 hours**; and **defer antibiotics** where the likelihood of infection is low.
- **Prolonged beta-lactam infusion** after a loading dose is now a **strong** recommendation (BLING III, MERCY trials).
- **Peripheral vasopressor initiation** is suggested rather than delaying until central access is obtained — practical in Indian emergency departments.
- **Respiratory support: high-flow nasal cannula (HFNC) is preferred over conventional oxygen**, and **HFNC over non-invasive ventilation as initial therapy**, in sepsis-associated hypoxaemic respiratory failure; awake proning is suggested in the non-intubated.
- **Corticosteroids are suggested for septic shock**; **terlipressin, levosimendan and beta-blockers are suggested against**; evidence remains insufficient for methylene blue and midodrine.
- **Restrictive transfusion strategy (strong)**; insulin started at glucose ≥ **180 mg/dL (strong)**; **LMWH preferred over unfractionated heparin (strong)** for VTE prophylaxis.
- **Survivorship is a new pillar**: pharmacist-led medication reconciliation at transitions, written and verbal sepsis education before discharge, physical rehabilitation after > 48 h of ventilation, mental-health support and post-critical-illness follow-up clinics — highly relevant to geriatric practice.
- Blood-culture yield falls sharply once antibiotics are started — a 2025 systematic review showed a 38% relative fall in sensitivity within a median of 70 minutes of the first dose, reinforcing pre-antibiotic sampling; a **single blood-culture set is acceptable in low-resource settings**.
- German S3 sepsis guideline update (July 2025) similarly emphasised individualised early therapy and **structured post-discharge aftercare**.

EXAMINER'S TIP — verbatim mark-earning lines

- "Sepsis is organ dysfunction, not just infection — Sepsis-3 replaced SIRS-based criteria in 2016 and SSC 2026 retains it."
- "In the elderly, delirium is the fever of the young — an acute confusional state in an older patient is sepsis until proved otherwise."
- "Absence of fever and absence of leucocytosis never exclude bacterial sepsis in old age; hypothermia carries a worse prognosis than pyrexia."
- "Never diagnose urosepsis on a urine culture alone — asymptomatic bacteriuria is present in a third of institutionalised elders."
- "SSC 2026 introduced the first age-specific haemodynamic target: MAP 60–65 mmHg in patients aged 65 years and above."
- "Frailty, not chronological age, predicts survival — age alone is never a justification for withholding critical care."

Q2. Define septic shock, SIRS and MODS. Discuss the etiopathogenesis, investigations and treatment.

Textbook coverage: Pathy's covers the coagulation limb in detail in Ch. 23 (Disseminated intravascular coagulation) — sepsis is the commonest cause of DIC, occurring in 20–80% of severe cases with a mortality of 20–50%. The consensus definitions of SIRS, septic shock and MODS are not stated in the uploaded texts and are taken from ACCP/SCCM (1992), Sepsis-3 (JAMA 2016) and SSC 2026.

15–20 High-Yield Points

Definitions

1. **SIRS** (ACCP/SCCM 1992) = **two or more** of: temperature > 38°C or < 36°C; heart rate > 90/min; respiratory rate > 20/min or $PaCO_2$ < 32 mmHg; total leucocyte count > 12,000/µL or < 4,000/µL or > 10% band forms. It is **non-specific** — pancreatitis, burns, trauma and surgery also produce SIRS.
2. **Septic shock** = a subset of sepsis with **circulatory, cellular and metabolic abnormalities** profound enough to increase mortality substantially. Clinically: **vasopressors needed to maintain MAP ≥ 65 mmHg AND lactate > 2 mmol/L despite adequate fluid resuscitation**. **Hospital mortality exceeds 40%.**
3. **MODS** = **progressive but potentially reversible dysfunction of two or more organ systems** arising from an acute insult, such that homeostasis cannot be maintained without intervention. **Primary MODS** results directly from the insult; **secondary MODS** develops later as a consequence of the host response.
4. **SOFA score** grades six systems 0–4 each: **respiratory** (PaO_2/FiO_2), **coagulation** (platelets), **hepatic** (bilirubin), **cardiovascular** (MAP/vasopressor dose), **CNS** (Glasgow Coma Scale) and **renal** (creatinine/urine output). A rise of ≥ 2 points defines sepsis.

Etiopathogenesis

5. **Trigger: PAMPs** (lipopolysaccharide, peptidoglycan, flagellin) and **DAMPs** (mitochondrial DNA, HMGB1, histones released from injured cells) are recognised by **Toll-like receptors** on monocytes and endothelium.
6. **Hyperinflammatory limb:** TLR ligation activates **NF-κB** and the inflammasome, releasing **TNF-α, IL-1β, IL-6 and IL-8** — the 'cytokine storm' that produces fever, capillary leak and myocardial depression.
7. **Endothelial injury:** shedding of the **endothelial glycocalyx**, loss of tight junctions and upregulated adhesion molecules cause **capillary leak, tissue oedema and microcirculatory failure**, with maldistribution of flow despite a normal or high cardiac output.
8. **Vasoplegia:** induction of **iNOS** generates excess nitric oxide; ATP-sensitive potassium channels open and **vasopressin stores are depleted**, producing catecholamine-resistant vasodilatation.
9. **Coagulopathy:** monocyte and endothelial **tissue factor** expression drives thrombin generation, while the natural anticoagulants (**antithrombin, protein C, protein S**) are consumed and fibrinolysis is suppressed by PAI-1 — giving **microvascular thrombosis with simultaneous bleeding**, i.e. DIC.
10. **The paradox of DIC** (Pathy's Ch. 23): the clinical picture is one of bleeding, while morbidity and mortality result from **organ damage due to microvascular thrombosis** — AKI and ARDS. Recognising and treating the underlying cause matters far more than replacing factors; giving factors and platelets without treating the cause simply provides more substrate for thrombosis.
11. **Cellular/mitochondrial dysfunction:** nitric-oxide-mediated inhibition of the respiratory chain produces **cytopathic hypoxia** — oxygen is delivered but cannot be utilised, hence lactate rises despite adequate perfusion.
12. **Immunoparalysis (compensatory anti-inflammatory response):** lymphocyte and dendritic-cell apoptosis, **reduced monocyte HLA-DR expression**, IL-10 excess — responsible for late deaths from secondary and opportunistic infection and for viral reactivation (CMV, HSV).
13. **Why the elderly do worse: inflammaging** gives a raised baseline IL-6 and TNF-α; **immunosenescence** impairs pathogen clearance; reduced cardiac, renal, respiratory and cerebral reserve means a small insult exhausts compensation; and polypharmacy (beta-blockers masking tachycardia, ACE inhibitors and diuretics worsening hypotension, NSAIDs precipitating AKI) blunts both the response and its recognition.

Investigations

14. **Confirm and grade organ dysfunction:** haemogram, **serum lactate and repeat lactate**, ABG (metabolic acidosis, raised anion gap, PaO_2/FiO_2 ratio), creatinine and urine output, bilirubin and transaminases, GCS.
15. **DIC screen** (Pathy's, Table 23): **APTT prolonged > 10 s beyond normal; PT prolonged > 5 s; thrombin time prolonged > 10 s; fibrinogen usually < 1 g/L; platelets usually < 50,000/µL; D-dimer > 1000 ng/mL**. Serial trends

matter more than a single set.

16. **Microbiology:** two sets of blood cultures before antibiotics, urine, sputum/endotracheal aspirate, pus, CSF; pathogen-specific rapid molecular tests on a case-by-case basis.
17. **Source imaging:** chest radiograph, ultrasound abdomen and chest (chest ultrasound is particularly suitable in the geriatric patient), CT abdomen/thorax, echocardiography. **Biomarkers** — CRP, procalcitonin (better for de-escalation than initiation) — support but never replace clinical judgement.

Treatment

18. **Resuscitation:** 30 mL/kg **balanced crystalloid** in the first 3 hours for hypoperfusion, then individualised; **noradrenaline first-line**, started peripherally if necessary; vasopressin added on escalation; adrenaline third; **dobutamine added** if there is myocardial dysfunction with persistent hypoperfusion despite adequate filling.
19. **Anti-infective therapy and source control** as in Q1 — immediate broad-spectrum antibiotics in shock, ideally within one hour, and source control ideally within 6 hours.
20. **Organ support:** lung-protective ventilation at **6 mL/kg ideal body weight with plateau pressure ≤ 30 cmH_2O** in ARDS, higher PEEP in moderate-to-severe ARDS, prone positioning > 12 h/day; renal replacement therapy only for definite indications; **sodium bicarbonate only if pH ≤ 7.2 with AKIN stage 2–3**.
21. **Treatment of DIC:** treat the cause; transfuse only if bleeding or before a procedure — platelets if < 50,000/µL with bleeding, fresh frozen plasma for prolonged PT/APTT, cryoprecipitate or fibrinogen concentrate if fibrinogen < 1 g/L. In the **chronic/thrombotic form** (malignancy-associated), **low-molecular-weight heparin is the treatment of choice**, with DOACs a recognised alternative; warfarin is notoriously difficult in this setting.
22. **Adjuncts and de-escalation:** IV hydrocortisone in vasopressor-dependent shock; stress-ulcer prophylaxis with a PPI if risk factors; LMWH thromboprophylaxis; early enteral nutrition; **active fluid removal with diuretics after the acute resuscitation phase**; daily review for antimicrobial de-escalation and for the shortest effective duration.

Current Practice Update

Textbook Position (Pathy's 6th ed. / Essentials 7th ed.)	Current Evidence & Guidelines (2023–2026)
SIRS/severe sepsis terminology and the classical inflammatory paradigm.	Replaced by **Sepsis-3**; SIRS survives only as a **screening tool** — and SSC 2026 places it **above qSOFA** for that purpose.
DIC treated by supporting the underlying cause and cautious product replacement (Ch. 23).	Unchanged and endorsed; **DOACs (rivaroxaban, apixaban)** now recognised alongside LMWH in the chronic thrombotic form.
Sepsis conceived largely as an overwhelming inflammatory response.	Now understood as a **biphasic** process — hyperinflammation followed by **immunoparalysis**, with distinct transcriptomic **phenotypes** (α, β, γ, δ) that differ in outcome and treatment response.
Lactate clearance targeted as a resuscitation endpoint.	SSC 2026 suggests serial lactate **plus capillary refill time**; fluids should **not** be continued simply until lactate normalises (ANDROMEDA-SHOCK).
Renal replacement therapy for sepsis-associated AKI not specified.	**Suggested against** RRT in the absence of a definitive indication (STARRT-AKI, AKIKI); either continuous or intermittent modality when indicated.

Latest Updates (2023–Present)

- **SSC 2026** reaffirms noradrenaline first-line over dopamine (high certainty), epinephrine and selepressin; **suggests against terlipressin**; and adds vasopressin on escalation with epinephrine third.
- **Sepsis phenotypes and endotypes** — transcriptomic and clinical clustering now guides trial enrichment, with the aim of matching corticosteroid or immunomodulator therapy to the biologically appropriate subgroup rather than to all comers.
- **Sepsis-induced coagulopathy (SIC) score** and the ISTH overt-DIC score are increasingly used for earlier recognition than the classical DIC picture; anticoagulant therapy remains reserved for selected patients.
- **Selective digestive decontamination** is suggested (moderate certainty) in mechanically ventilated patients in units with a low prevalence of antimicrobial resistance — a recommendation of limited applicability in most Indian ICUs.
- **Active fluid removal** after the acute phase is a new suggestion, reflecting recognition that a positive cumulative fluid balance independently predicts mortality.

- **Beta-blockers (esmolol, landiolol) are suggested against** in septic shock following the negative STRESS-L trial.

EXAMINER'S TIP — verbatim mark-earning lines

- "SIRS is sensitive but not specific; SOFA is specific but needs laboratory data — hence SIRS/NEWS to screen, SOFA to define."
- "Septic shock is a vasopressor-plus-lactate definition, not a blood-pressure definition; mortality exceeds 40%."
- "The paradox of DIC: the patient bleeds, but dies of microvascular thrombosis."
- "Cytopathic hypoxia explains why lactate remains high despite adequate oxygen delivery — the defect is mitochondrial, not circulatory."
- "Late deaths in sepsis are deaths of immunoparalysis, not of cytokine storm."
- "In the elderly, a beta-blocked patient will not mount a tachycardia — tachypnoea and delirium are the surviving warning signs."

Q3. Discuss the recent advances in the management of sepsis.

15–20 High-Yield Points

Recognition and Diagnostics

1. **Screening tools have been reordered:** SSC 2026 **strongly recommends NEWS, NEWS2, MEWS or SIRS in preference to qSOFA** as a single in-hospital screening instrument, because qSOFA lacks sensitivity and detects patients too late.
2. **Institutional systems**, not just drugs, are now recommended: mandatory **sepsis performance-improvement programmes** (strong recommendation) and **'code sepsis' or sepsis-huddle protocols**; screening is also suggested **pre-hospital, in the ambulance**.
3. **Rapid molecular diagnostics** — multiplex PCR panels, T2 magnetic resonance and metagenomic next-generation sequencing — allow pathogen and resistance-gene identification in hours; recommended for **selected patients** guided by local resistance patterns and stewardship input, not for everyone.
4. **Host-response transcriptomic assays** (e.g. SeptiCyte, IMX-BVN) that distinguish bacterial from viral and from non-infective inflammation are promising but **evidence remains insufficient** for a recommendation.
5. **Artificial-intelligence early-warning systems** using continuous EHR data are being deployed to predict deterioration hours before clinical recognition; validation in older, multimorbid populations is still limited.

Antimicrobial Strategy

6. **Timing has been graded rather than universal:** antibiotics within **1 hour** for septic shock and probable sepsis, within **3 hours** for 'possible' sepsis without shock after rapid investigation, and **deferred** if infection is unlikely — a deliberate correction of the over-treatment that blanket one-hour rules produced.
7. **Prolonged/extended infusion of beta-lactams** after a loading dose is now a **strong recommendation**, following **BLING III** and **MERCY** and their meta-analysis, which showed a mortality benefit through improved time-above-MIC.
8. **Narrower empirical therapy:** routine **anaerobic cover** and routine **empirical antifungals** are advised against; MDR cover only where specific risk factors exist (known colonisation, prior MDR infection, prolonged broad-spectrum exposure, prolonged stay in a high-prevalence unit).
9. **Procalcitonin** has moved from the front door to the exit: clinical evaluation alone is suggested to **start** antibiotics, but **procalcitonin plus clinical evaluation** is suggested to decide when to **stop**. **Shorter courses are preferred** where source control is adequate.
10. **New agents against resistant gram-negatives** — ceftazidime–avibactam, meropenem–vaborbactam, imipenem–relebactam, **cefiderocol**, ceftolozane–tazobactam and (emerging) aztreonam–avibactam and cefepime–taniborbactam — are of particular importance in India, where carbapenem-resistant *Klebsiella* and *Acinetobacter* dominate ICU sepsis.

Haemodynamic Resuscitation

11. **Balanced crystalloids over 0.9% saline** (BaSICS, PLUS and their meta-analyses), with **starches and gelatins prohibited** and albumin reserved for those already given large crystalloid volumes or with cirrhosis.
12. **The liberal-versus-restrictive fluid debate has been closed to a draw:** after the initial 30 mL/kg, **CLASSIC** and **CLOVERS** showed no mortality difference, so SSC 2026 permits either strategy, individualised to the patient (heart failure, renal reserve) and the health system.
13. **Peripheral vasopressors** are now suggested rather than delaying noradrenaline until central access is obtained — earlier MAP restoration and less fluid overload.
14. **Capillary refill time** is endorsed as an adjunct perfusion target (ANDROMEDA-SHOCK), and fluids should **not** simply be continued until lactate normalises.
15. **Age-specific target:** in patients ≥ **65 years, an initial MAP of 60–65 mmHg** is suggested — the first explicitly geriatric haemodynamic recommendation in the guideline's history, reflecting the harm of vasopressor excess in stiff, atheromatous vasculature.

Adjunctive and Organ-Support Therapies

16. **Therapies now recommended against** after negative trials: **IV vitamin C** (VITAMINS, VICTAS, **LOVIT** — which showed possible harm), **IV immunoglobulin, vitamin D** (VIOLET), **blood purification and polymyxin B haemoperfusion** (EUPHRATES), **levosimendan** (LeoPARDS) and **beta-blockers** (STRESS-L).
17. **Corticosteroids** are suggested for septic shock; the practical residue of APROCCHSS/ADRENAL is hydrocortisone 200 mg/day in vasopressor-dependent shock. Evidence for **methylene blue** and **angiotensin II** in refractory

vasoplegia remains insufficient for a recommendation.

18. **Respiratory support has shifted to HFNC:** HFNC over conventional oxygen, HFNC over NIV as initial therapy, and a trial of **awake proning** in the non-intubated — largely a legacy of COVID-19-era evidence.
19. **Restrictive transfusion (strong)**, insulin initiation at ≥ **180 mg/dL (strong)** rather than tight control, **LMWH over unfractionated heparin (strong)**, and **pharmacological prophylaxis alone** rather than adding mechanical prophylaxis.
20. **Sepsis survivorship is now formally part of sepsis management**: goals-of-care discussion within 72 hours, pharmacist-led medication reconciliation at every transition, screening for social and economic needs, written and verbal patient/family education, post-ICU follow-up services, physical rehabilitation after > 48 hours of ventilation and mental-health support. For geriatrics this converts sepsis care into a comprehensive geriatric assessment problem, not merely an ICU one.
21. **Health-system equity** is explicit: single blood-culture sets are acceptable where resources are limited, and 38% of the guideline panel practised in low- and middle-income countries — making the 2026 document more directly applicable to Indian practice than any previous edition.

Current Practice Update

Textbook Position (Pathy's 6th ed. / Essentials 7th ed.)	Current Evidence & Guidelines (2023–2026)
Universal ‘antibiotics within one hour for everyone’.	**Tiered by certainty and shock** — 1 h for shock/probable sepsis, 3 h for possible sepsis, defer if infection unlikely.
Bolus dosing of beta-lactams.	**Prolonged infusion after a loading dose — strong recommendation** (BLING III, MERCY).
Vitamin C / thiamine / hydrocortisone (‘metabolic resuscitation’) widely trialled after 2017.	**Abandoned** — SSC 2026 suggests against IV vitamin C on the basis of LOVIT and pooled data.
0.9% saline as default resuscitation fluid.	**Balanced crystalloids preferred**; saline retained specifically for sepsis with traumatic brain injury.
qSOFA as the bedside screen.	**NEWS/NEWS2/MEWS/SIRS strongly preferred over qSOFA.**
Care ends at ICU or hospital discharge.	**Post-sepsis survivorship** — rehabilitation, cognition, mental health, medication reconciliation — is now a formal component of the guideline.

Latest Updates (2023–Present)

- **SSC 2026** (*Crit Care Med* 2026;54:725–812) — the first full revision since 2021: 129 statements, 46 new, with a dedicated survivorship section and quick-reference bedside graphics.
- **BLING III** (*JAMA* 2024) and **MERCY** (*JAMA* 2024) with subsequent meta-analysis established continuous/extended beta-lactam infusion.
- **CLOVERS** (*NEJM* 2023) and **CLASSIC** (*NEJM* 2022) — neither restrictive fluids with early vasopressors nor liberal fluids proved superior, ending a long-running controversy.
- **LOVIT** (*NEJM* 2022) — high-dose vitamin C associated with a higher risk of death or persistent organ dysfunction, closing the metabolic-resuscitation era.
- **STRESS-L** (*JAMA* 2023) — landiolol did not reduce organ dysfunction in septic shock and signalled possible harm.
- **German S3 guideline update (2025)** and the **WHO 2023–2030 global sepsis resolution implementation reports** emphasise national sepsis registries, quality bundles and antimicrobial stewardship.
- Continuing directions: **machine-learning early-warning scores**, **immunophenotype-guided immunotherapy** (GM-CSF, interferon-gamma, anti-PD-1 for immunoparalysis), **extracorporeal cytokine adsorption** (not currently recommended), and **bacteriophage therapy** for extensively drug-resistant gram-negative sepsis — all still investigational.

EXAMINER'S TIP — verbatim mark-earning lines

- “The direction of travel in sepsis since 2021 is de-escalation: fewer empirical antibiotics, less fluid, fewer adjuncts, and more system-level recognition.”
- “The single strongest new pharmacological recommendation is prolonged beta-lactam infusion after a loading dose.”
- “Every major immunomodulatory adjunct trialled since 2017 — vitamin C, IVIG, vitamin D, haemoadsorption — is now recommended against.”
- “Sepsis does not end at discharge: post-sepsis syndrome is now a guideline-mandated part of management.”
- “The 2026 guideline contains the first age-stratified haemodynamic target in critical care — MAP 60–65 mmHg at 65 years and above.”

Q4. Discuss the management of COPD with reference to recent recommendations.

15–20 High-Yield Points

Definition, Burden and Assessment

1. COPD is characterised by **airflow obstruction that is not fully reversible**, shows little variability over time, is usually progressive, and reflects small-airway disease and emphysema. Diagnosis requires a **post-bronchodilator FEV_1/FVC < 0.70** (or below the lower limit of normal, which is preferred in the elderly).
2. COPD is the **fifth largest cause of disability worldwide**; worldwide prevalence is 4–10%, but **12–15% in the elderly**. Despite this, it is **persistently under- and mis-diagnosed in older adults**, largely because breathlessness is attributed to age, obesity or cardiac disease.
3. **Severity of airflow obstruction** (post-bronchodilator FEV_1/FVC < 0.7): Stage 1 mild, $FEV_1 \geq 80\%$; Stage 2 moderate, 50–79%; Stage 3 severe, 30–49%; Stage 4 very severe, < 30% predicted.
4. **Assessment must be multidimensional**, not spirometric alone: breathlessness (**mMRC**), health status (**CAT**), exercise tolerance (shuttle walk or six-minute walk test), **BMI**, oxygen saturation if FEV_1 < 50% or signs of cor pulmonale, screening for **anxiety and depression (HADS)**, and the exacerbation rate.
5. **Comorbidity is the rule in the older COPD patient** and is classified as: **co-causal** (shared smoking risk — ischaemic heart disease, heart failure, lung and bladder cancer, CKD, aortic aneurysm); **coexisting** (diabetes, hypertension, benign prostatic hypertrophy, glaucoma, OSA); and **complicating** (anxiety/depression, **osteoporosis, sarcopenia**, arrhythmias, dysphagia, pulmonary embolism).
6. **A critical geriatric prescribing trap:** sarcopenia blunts the rise in serum creatinine, so **moderate (GFR 30–60) and even severe renal impairment go unrecognised**, leading to overdose of renally cleared drugs during exacerbations. Umeclidinium and aclidinium are safe in renal failure as they are hepatically metabolised to inactive derivatives.

Management of Stable Disease

7. **Smoking cessation is the only intervention that slows the age-related decline in FEV_1**. Use nicotine replacement, bupropion or varenicline; **quit rates in older adults are as good as or better than in the young**. **E-cigarettes are not recommended** as a cessation aid.
8. **Vaccination:** annual influenza and pneumococcal vaccination for all; current guidance adds **RSV vaccination and COVID-19 vaccination**, and pertussis-containing Tdap.
9. **Pulmonary rehabilitation** improves dyspnoea, health status and exercise tolerance, and secondarily mood and quality of life; it should be part of integrated management **from the early stages**, not reserved for end-stage disease.
10. **Self-management plans** covering recognition and early treatment of exacerbations are vital; as disease progresses, there must be an opportunity for **advance care planning and discussion of end-of-life wishes**.

Pharmacological Therapy

11. **On-demand short-acting bronchodilators alone should be discouraged in the elderly** because they lead to adverse drug reactions and overdosing; regular **long-acting** therapy is preferred.
12. **LAMA and LABA** are the foundation: both improve symptoms, health status and exacerbation rate, and **LAMAs are clearly superior to short-acting ipratropium**. Escalate to **LABA + LAMA** for persistent breathlessness.
13. **Inhaled corticosteroids** are added for frequent exacerbators or those with asthma-like features. They increase the risk of **non-fatal pneumonia**, but this does not outweigh benefit in appropriately selected patients. **Blood eosinophil count guides the decision** — ICS considered above 300 cells/µL, avoided below 100 cells/µL.
14. **Second-line options:** slow-release **theophylline** at low dose (poor therapeutic index, drug interactions with macrolides and quinolones); **roflumilast** (PDE-4 inhibitor) for frequent exacerbators with chronic bronchitis and FEV_1 < 50% — monitor for weight loss, diarrhoea and mood disturbance, with limited safety data in the elderly; **high-dose acetylcysteine** as a mucolytic/antioxidant; nebulised bronchodilators where inhaler technique cannot be corrected.
15. **Maintenance oral corticosteroids should generally be avoided** — risks outweigh benefits; where repeated courses are used, **screen and treat for osteoporosis**, which should in any case be routine in elderly COPD.

Oxygen, Exacerbations and Palliation

16. **Long-term oxygen therapy (LTOT)** for ≥ 15 hours a day improves survival in severe COPD with persistent hypoxaemia. Prescribe during clinical stability, to non-smokers, when **PaO_2 < 7.3 kPa**, or **PaO_2 7.3–8.0 kPa with secondary polycythaemia, pulmonary hypertension, peripheral oedema or nocturnal hypoxaemia**. Screen by referring patients with saturation < 93%, or < 95% with possible cor pulmonale.

17. **Exacerbation management:** increased bronchodilators; **oral corticosteroids reduce length of stay and treatment failure**; **antibiotics where sputum is purulent** (commonest organisms *Haemophilus influenzae*, *Streptococcus pneumoniae*, *Moraxella catarrhalis*) — but antibiotics should be withheld if there is no sputum purulence. Controlled oxygen targeting **SpO_2 88–92%**; **NIV if pH < 7.35** despite maximal medical therapy, which reduces mortality, intubation and length of stay.
18. **Prognosis:** about one-third of admitted patients are readmitted within 30 days and 15% die by three months; roughly **50% of patients on LTOT survive three years**. In older adults the main predictors of mortality are **disability, low BMI, lung function and use of LTOT**.
19. **Palliative care:** patients judged to be in their last year of life should be placed on an end-of-life register and offered **opioids and benzodiazepines** for refractory breathlessness. The textbook is explicit that the move from an active to a palliative approach **all too often comes too late**.
20. **Nutrition and sarcopenia:** hypercatabolism and accelerated muscle loss characterise severe exacerbations. **Nutritional support improves respiratory and peripheral muscle strength but not lung-function indices**, and should be combined with exercise in a comprehensive programme for the frail patient.

Current Practice Update

Textbook Position (Pathy's 6th ed. / Essentials 7th ed.)	Current Evidence & Guidelines (2023–2026)
Fixed FEV_1-based staging (GOLD 1–4) drives treatment; algorithm based on mMRC/CAT and exacerbations (Pathy's Fig. 42.1).	**GOLD 2026** uses the **ABE groups**: A = low symptoms/low risk; B = high symptoms (mMRC ≥ 2 or CAT ≥ 10); **E = exacerbators**. Spirometric grade informs prognosis, not initial drug choice.
'Frequent exacerbations' defined as ≥ 2 exacerbations and/or 1 hospitalisation in the previous year.	GOLD 2026 lowers the threshold: escalation is recommended after **even one moderate exacerbation**, since a single event predicts future risk.
Initial therapy may be a single long-acting bronchodilator in most symptomatic patients.	**Group B and Group E now start on LABA + LAMA dual therapy**; monotherapy is reserved for Group A.
ICS added on clinical grounds (asthma features, frequent exacerbations).	**Blood eosinophil count is the biomarker of choice** — ICS added in Group E if **eosinophils > 300 cells/μL**; avoided below 100.
Theophylline is a recognised option when symptoms persist.	**GOLD 2026 does not recommend methylxanthines** because of side effects.
Pneumococcal and influenza vaccines recommended.	Expanded: **RSV vaccination from age 50** (lowered from 60 in the 2026 report), plus COVID-19 and Tdap.
No biologic therapy available for COPD.	**Dupilumab** (anti-IL-4Rα, approved 2024) and **mepolizumab** (anti-IL-5, approved May 2025) are now licensed add-ons for inadequately controlled **eosinophilic COPD**; **ensifentrine** (dual PDE3/PDE4 inhibitor) is a new inhaled maintenance option.
Corticosteroid course for exacerbation continued 7–14 days depending on severity.	**Systemic corticosteroids for up to 5 days** and **antibiotics for 5 days** in moderate/severe exacerbations.

Latest Updates (2023–Present)

- **GOLD 2026 report** (published November 2025; sixth major revision) — incorporates 330 new references, completely revised chapters 4 and 5, updated screening and case-finding, and a **new chapter on artificial intelligence and emerging technologies** in COPD detection and management.
- **New concept of 'disease activity'** — reducing reversible biological activity (using blood eosinophils as a type-2 inflammation biomarker) is now framed as a treatment goal alongside symptom control; strategies include smoking cessation, pulmonary rehabilitation, pharmacotherapy and lung-volume reduction.
- **Expanded multimorbidity and frailty chapter** — GOLD 2026 explicitly contextualises COPD within multimorbidity and frailty, which brings the guideline much closer to geriatric practice.
- **Dupilumab** — BOREAS (*NEJM* 2023) and NOTUS (*NEJM* 2024) showed 30% and 34% reductions in moderate/severe exacerbations on top of triple therapy in patients with eosinophils ≥ 300 cells/μL; FDA approval September 2024.

- **Mepolizumab** — MATINEE (*NEJM* 2025) showed a 21% reduction in exacerbations and a 35% reduction in exacerbation-related emergency visits/hospitalisation; FDA approval May 2025 for eosinophils ≥ 150 cells/µL.
- **Ensifentrine** — first-in-class inhaled dual PDE3/PDE4 inhibitor with both bronchodilator and anti-inflammatory action (ENHANCE-1 and -2), included in GOLD as an option where roflumilast is poorly tolerated.
- **Tezepelumab** (anti-TSLP) failed its primary endpoint in the overall COPD population in COURSE (phase 2a) but showed signal at higher eosinophil counts.
- **Indian context:** COPD is the second commonest cause of death in India (ICMR India State-Level Disease Burden Initiative); **biomass and solid-fuel smoke exposure** is a major non-smoking cause, particularly in older rural women, and COPD care is delivered through the **NP-NCD programme and Ayushman Arogya Mandirs**. Access to biologics remains negligible outside tertiary private practice.

EXAMINER'S TIP — verbatim mark-earning lines

- "The GOLD ABE assessment replaced ABCD in 2023 — Group E collapses the old C and D because exacerbation risk, not symptom burden, drives therapy."
- "In 2026 a single moderate exacerbation is now sufficient to justify treatment escalation."
- "Blood eosinophil count is the only routinely available biomarker guiding ICS and biologic use in COPD."
- "COPD is now a biologic-treatable disease: dupilumab (2024) and mepolizumab (2025) for the eosinophilic phenotype."
- "In the elderly, sarcopenia masks renal impairment — always estimate GFR before dosing renally cleared drugs in an exacerbation."
- "Only three interventions alter mortality in COPD: smoking cessation, long-term oxygen therapy in chronic hypoxaemia, and lung volume reduction in selected emphysema."

Q5. Discuss the tailoring of oxygen requirements and delivery systems for a COPD patient with severe hypoxaemia and an infective exacerbation.

15–20 High-Yield Points

Principles

1. **Oxygen is a drug**: it must be prescribed with a **target saturation range**, a device and a flow rate, and signed for on the drug chart like any other medicine.
2. **The therapeutic target is a saturation range, not a flow rate**. In COPD with an infective exacerbation the aim is **SpO_2 88–92%** pending arterial blood gases — adjust upward to 94–98% only if the ABG shows a normal $PaCO_2$ and there is no history of previous hypercapnic failure requiring ventilation.
3. **Why the ceiling matters:** excess oxygen in the chronic CO_2 retainer produces hypercapnia by **reversal of hypoxic pulmonary vasoconstriction with worsened V/Q mismatch** (the dominant mechanism), the **Haldane effect**, and to a lesser extent depression of hypoxic respiratory drive — leading to **respiratory acidosis, narcosis and death**. Pathy's warns of depressed hypoxic drive, increased coronary and cerebral arterial resistance and worsened acidosis, and advises target saturations in this range.
4. **The opposite error is equally lethal: oxygen must never be withheld from a severely hypoxaemic patient** for fear of retention. Hypoxaemia kills faster than hypercapnia; treat the hypoxaemia and manage the CO_2 with ventilatory support.
5. **Baseline saturations are lower in the very old** — clinically stable people over 70 may sit at 92–94% and require no oxygen; know the patient's stable baseline before treating a number.

Initial Assessment

6. **Immediate assessment:** respiratory rate, level of consciousness, ability to speak, accessory muscle use, SpO_2, and an **arterial blood gas within 30–60 minutes** of starting oxygen — and repeated 30–60 minutes after every significant change in FiO_2.
7. **Classify the failure** from the ABG: **Type 1** (PaO_2 < 8 kPa / 60 mmHg with normal or low $PaCO_2$) versus **Type 2** (PaO_2 < 8 kPa with $PaCO_2$ > 6.5 kPa / 50 mmHg). Then distinguish **acute hypercapnic failure** (low pH, normal bicarbonate) from **acute-on-chronic** (low pH with raised bicarbonate and base excess) — the bicarbonate tells you how chronic the retention is.
8. **Look for the treatable precipitant simultaneously:** purulent sputum and consolidation, pneumonia (which in older adults is frequently afebrile and non-productive), pneumothorax, pulmonary embolism, heart failure, arrhythmia, sedative drugs and aspiration.

Choosing the Delivery System

9. **Venturi (high-flow, fixed-performance) masks are the device of choice in COPD** because they deliver a **predictable, constant FiO_2 independent of the patient's inspiratory flow** — start at **24% at 2–3 L/min** or **28% at 4 L/min**. If the respiratory rate exceeds 30/min, **increase the flow through the Venturi barrel by up to 50%** to maintain the entrained FiO_2 (the barrel colour, not the flow, sets the concentration).
10. **Nasal cannulae (1–2 L/min)** are an acceptable alternative once the patient is stable and the target range is reliably held: better tolerated, allow eating, talking and expectoration, and reduce delirium and claustrophobia in the older patient — but they are **variable-performance**, with FiO_2 depending on minute ventilation and mouth-breathing.
11. **Simple face mask (5–10 L/min)** delivers roughly 40–60% and is **generally unsuitable in COPD**; below 5 L/min there is a risk of CO_2 rebreathing within the mask.
12. **Reservoir (non-rebreathing) mask at 15 L/min** gives 60–90% and is reserved for **critical illness or life-threatening hypoxaemia (SpO_2 < 85%)** as an emergency bridge — step down to a Venturi mask as soon as the gas is available.
13. **Humidification** should be added for flows above 4 L/min, for prolonged therapy and where secretions are tenacious, to preserve mucociliary clearance — important where the cough reflex is already blunted by ageing.

Escalation

14. **High-flow nasal cannula (HFNC)** delivers heated, humidified gas at 30–60 L/min with a precisely titrated FiO_2, washes out anatomical dead space, generates a small PEEP effect and improves secretion clearance. It has proved **safe and effective in exacerbated COPD in both normocapnic and hypercapnic respiratory failure**, and is particularly valuable **where there is a do-not-intubate order or where intermediate care is needed outside the ICU**.

15. **Non-invasive ventilation (NIV/BiPAP) is the key intervention in acute hypercapnic failure:** indicated when **pH < 7.35 with $PaCO_2$ > 6.5 kPa despite maximal medical therapy** and controlled oxygen. **NIV reduces mortality, need for intubation and length of hospital stay.** Start at IPAP 10–12 and EPAP 4–5 cmH_2O, titrating IPAP upward to 20–25 by response, with oxygen entrained to keep SpO_2 88–92%; repeat the gas at 1 and 4 hours.
16. **Invasive ventilation** is considered for pH < 7.25 with deteriorating consciousness, NIV failure or intolerance, cardiovascular instability or inability to protect the airway — but the decision must be made **on premorbid function, frailty and previously stated wishes, not chronological age**, and a ceiling of care should ideally be documented before deterioration.

Concurrent Treatment of the Exacerbation and Discharge

17. **Treat the exacerbation in parallel:** nebulised salbutamol and ipratropium **driven by air, not oxygen**, in the CO_2 retainer (with nasal oxygen alongside at 1–2 L/min); systemic corticosteroids; and **antibiotics where sputum is purulent** — beta-lactam ± inhibitor, doxycycline or a fluoroquinolone, with anti-pseudomonal cover (ciprofloxacin, anti-pseudomonal cephalosporin or aminoglycoside) if bronchiectasis coexists.
18. **Additional supportive measures:** chest physiotherapy and secretion clearance, VTE prophylaxis, correction of dehydration and hypokalaemia, delirium prevention, review of sedatives and opioids, and nutritional support — hypercatabolism and muscle loss are hallmarks of severe exacerbation in the elderly.
19. **Wean the oxygen deliberately:** step down device and FiO_2 as the patient improves, reassess on air before discharge, and do **not** assess for LTOT during the acute episode — LTOT must be prescribed during a period of clinical stability, generally **at least 6–8 weeks after the exacerbation**, in a non-smoker.
20. **Practical geriatric issues with home oxygen:** long tubing and concentrators are a **falls hazard**; there are safety concerns where there is cognitive impairment or poor manual dexterity; fire risk mandates that the patient and household are non-smokers; care-home residents using overnight concentrators may need portable cylinders for daytime rooms; and staff and carers require training. Issue **oxygen alert cards** recording previous blood-gas results to guide future emergency treatment.

Current Practice Update

Textbook Position (Pathy's 6th ed. / Essentials 7th ed.)	Current Evidence & Guidelines (2023–2026)
Target saturation quoted in the text as 89–95% for the CO_2 retainer.	**BTS and GOLD standardise on 88–92%** pending blood gases in patients at risk of hypercapnic failure; 94–98% for those not at risk.
High-flow oxygen described as recently proven safe and effective in exacerbated COPD.	**HFNC is now mainstream** — recommended over conventional oxygen in hypoxaemic failure, valuable in do-not-intubate patients, and increasingly used to prevent post-extubation failure.
NIV for pH < 7.35 despite maximal medical therapy.	Unchanged and reinforced by BTS/ICS guidance: NIV is **first-line** for acute hypercapnic acidosis; delay in starting it is a recognised cause of avoidable death.
LTOT criteria: PaO_2 < 7.3 kPa, or 7.3–8.0 kPa with cor pulmonale/polycythaemia/nocturnal hypoxaemia.	Unchanged, but **LOTT** confirmed **no benefit in moderate or exertional desaturation alone**, and assessment must be deferred until stability.
Oxygen prescribed to correct hypoxaemia.	Modern emphasis on avoiding **hyperoxia**: SSC 2026 permits either conservative (SpO_2 90–93%) or liberal (≥ 96%) targets in sepsis, and observational data link hyperoxaemia in COPD exacerbation to increased mortality.

Latest Updates (2023–Present)

- **BTS Guideline for oxygen use in adults in healthcare and emergency settings** remains the reference standard: 94–98% for most acutely ill adults, **88–92% where there is risk of hypercapnic respiratory failure**, with a fall of more than 3% within the target range prompting reassessment.
- **BTS/ICS guideline for ventilatory management of acute hypercapnic respiratory failure** continues to define pH < 7.35 with hypercapnia as the NIV threshold, and emphasises that **NIV should be commenced within 60 minutes** of the qualifying blood gas.
- **HFNC in hypercapnic COPD** — meta-analyses (2023–2025) show HFNC to be **non-inferior to NIV** for mild-to-moderate hypercapnic exacerbations, with better comfort and tolerance; it is increasingly used as a rescue for

NIV intolerance and in the frail patient with a ceiling of care.

- **SSC 2026** suggests **HFNC over conventional oxygen therapy, and HFNC over NIV as initial therapy**, in sepsis-associated acute hypoxaemic respiratory failure — directly applicable to the COPD patient with pneumonic sepsis.
- **Pulse oximetry accuracy** is now a recognised safety issue: SSC 2026 notes SpO_2 is **less accurate in shock, in darker skin tones and at saturations below 90% or above 97%**. In an Indian population this is a strong argument for confirming with arterial blood gases rather than relying on the oximeter alone.
- **GOLD 2026** advises systemic corticosteroids for up to 5 days and antibiotics for 5 days in moderate-to-severe exacerbations, and starting maintenance long-acting bronchodilator therapy before discharge, with ICS added at discharge for patients with an exacerbation and raised eosinophils.

EXAMINER'S TIP — verbatim mark-earning lines

- "Oxygen is a drug — prescribe a target range, not a flow rate."
- "In COPD, aim 88–92% pending blood gases; do not withhold oxygen from a severely hypoxaemic patient for fear of retention."
- "The Venturi mask is the device of choice because it is fixed-performance — the barrel colour sets the FiO2, and the flow only ensures the total gas flow exceeds peak inspiratory demand."
- "Hypercapnia on oxygen is mainly V/Q mismatch from reversal of hypoxic pulmonary vasoconstriction, plus the Haldane effect — loss of hypoxic drive is the smallest component."
- "NIV for pH below 7.35 reduces mortality, intubation rate and length of stay — delay in starting it is a recognised avoidable cause of death."
- "Never assess for long-term oxygen therapy during an exacerbation — wait 6–8 weeks of clinical stability."
- "Drive nebulisers with air, not oxygen, in the CO2 retainer."

Q6. Discuss the role of CPAP in diseases of the elderly.

15–20 High-Yield Points

Principle and Mechanism

1. **CPAP delivers a single continuous positive pressure throughout the respiratory cycle** through a nasal or oro-nasal interface. It acts as a **pneumatic splint** for the upper airway, **recruits collapsed alveoli and increases functional residual capacity**, reduces the work of breathing and, by raising intrathoracic pressure, **reduces left ventricular preload and afterload**.
2. **CPAP is not a ventilator.** It provides no inspiratory pressure support and therefore does not augment tidal volume or clear carbon dioxide — **hypercapnic respiratory failure requires bilevel NIV (BiPAP), not CPAP**. This distinction is a favourite viva question.

Obstructive Sleep Apnoea — the Principal Indication

3. **OSA is defined** by an **apnoea–hypopnoea index (AHI) > 5/hour** with excessive daytime sleepiness or neurocognitive/behavioural disturbance not attributable to another cause.
4. **OSA is far commoner in old age:** approximately **50% of older people have an AHI > 5 and 20% have an AHI > 15**, compared with about 10% of younger adults. The rise is explained by **increased upper-airway collapsibility** with age — loss of pharyngeal dilator muscle tone and altered reflexes, **edentulism**, increased pharyngeal fat deposition and post-menopausal hormonal change (after which the sex difference disappears).
5. **The classical triad is unreliable in the elderly:** the proportion of OSA patients who do **not** snore rises after 50; daytime sleepiness is confounded by comorbidity and drugs; and the Epworth Sleepiness Scale includes driving items that are unsuitable for many older people. Suspect OSA instead from **nocturia, morning headache, non-restorative sleep, unexplained falls, refractory hypertension and worsening cognition**.
6. **Polysomnography is the gold standard**; simplified home respiratory polygraphy is cheaper but **less satisfactory a priori in the elderly** because of the high background frequency of restless legs and other sleep disorders and the practical difficulty of accommodating equipment at home — each case must be judged individually.
7. **CPAP is the treatment of choice for OSA.** It abolishes snoring and apnoeas, improves gas exchange and normalises sleep architecture, and in older adults improves alertness, cognitive processing, memory and executive function.
8. **Adherence is the principal limitation — 40–50% of patients do not use the device adequately**. Importantly, **compliance in older patients is no worse than in the young**, including in series of patients with dementia or cognitive impairment; practical difficulties such as applying the mask are solved by patient and **caregiver training**.
9. **Adjunctive measures:** weight reduction in the obese, positional therapy, avoidance of alcohol and sedatives (especially benzodiazepines and Z-drugs), treatment of nasal obstruction, and mandibular advancement devices where dentition allows.

Cardiovascular and Neurocognitive Indications

10. OSA is epidemiologically linked in older people to **hypertension, ischaemic stroke, heart failure, atrial fibrillation and ischaemic heart disease** — and in advanced age these cardiovascular manifestations may be the **only** presentation of OSA. A prospective study showed that **long-term CPAP reduced cardiovascular mortality in elderly OSA patients**.
11. **Heart failure:** Pathy's lists **sleep-disordered breathing as a comorbidity of heart failure that increases mortality risk, with CPAP as the key management**. CPAP reduces preload and afterload, improves left ventricular ejection fraction and reduces sympathetic activation.
12. **Acute cardiogenic pulmonary oedema** is the other major acute indication: CPAP at 5–10 cmH_2O improves oxygenation, reduces the work of breathing and **reduces the need for intubation**, and is often preferable to intubation in the frail elderly patient.
13. **Cognition and dementia:** sleep-disordered breathing is associated with Alzheimer-type dementia and with agitation in institutionalised patients with dementia. CPAP is associated with improved neurocognitive performance and, on imaging, with favourable changes in cortical thickness and default-mode-network connectivity.
14. **Depression, epilepsy and falls:** untreated OSA in the elderly is correlated with worsening depressive symptoms, poor seizure control and **frequent falls** — all potentially modifiable by effective CPAP therapy.

Other Uses, Contraindications and Practical Issues

15. **Other clinical roles:** obesity hypoventilation syndrome (though bilevel NIV is preferred if hypercapnia is established), **type 1 hypoxaemic respiratory failure** including pneumonia and COVID-19, **post-operative**

atelectasis and prevention of post-extubation failure, and chest-wall trauma. In multiple sclerosis and other neurological disease, CPAP and BiPAP address the OSA that commonly underlies fatigue.

16. **Contraindications:** reduced consciousness and inability to protect the airway, facial trauma or surgery, **undrained pneumothorax**, vomiting or high aspiration risk, haemodynamic instability, severe epistaxis, and recent upper gastrointestinal surgery. **Established hypercapnic acidosis is a contraindication to CPAP in favour of BiPAP.**
17. **Complications:** mask pressure sores over the nasal bridge (a particular hazard on thin, fragile, ageing skin), nasal dryness, epistaxis, conjunctivitis from leak, gastric distension and aspiration, claustrophobia and **precipitation or worsening of delirium**.
18. **Geriatric-specific practical points:** humidification is essential; the interface must be fitted around dentures and facial contours; arthritis and poor dexterity affect strap handling; cognitive impairment demands caregiver involvement; and CPAP therapy should be introduced with a **structured education and early-follow-up programme**, since adherence in the first week predicts long-term use.
19. **Ethical framing:** in the frail or severely demented patient, CPAP may be entirely appropriate as a **ceiling of care** — the maximum agreed level of respiratory support short of intubation — and this should be documented in advance.
20. **Unresolved question, as stated in the textbook:** what remains to be established is whether CPAP in seniors corrects the symptoms caused by OSA and modifies cardiovascular comorbidity to the same degree as in younger patients.

Current Practice Update

Textbook Position (Pathy's 6th ed. / Essentials 7th ed.)	Current Evidence & Guidelines (2023–2026)
CPAP is the treatment of choice for OSA; compliance is the main obstacle, and its effect on symptoms and cardiovascular comorbidity in seniors remains to be evaluated.	Position broadly unchanged. Randomised trials (SAVE, RICCADSA, ISAACC) failed to show **secondary cardiovascular prevention** in unselected non-sleepy patients, but this is now attributed largely to **poor adherence (~3 h/night)**; adherence-adjusted analyses and 2025 meta-analyses show mortality benefit with good use.
Weight loss and positional therapy are the recommended adjunct measures.	**Pharmacotherapy has entered the field: tirzepatide** was FDA-approved in December 2024 for moderate-to-severe OSA with obesity — the first drug ever licensed for OSA.
Cognitive effects of CPAP in older adults described as small and inconsistent.	A 2025 randomised trial demonstrated **improved cortical thickness and default-mode-network connectivity** with 12 months of CPAP, though measurable cognitive gain in normal-cognition patients remained modest.
Home respiratory polygraphy regarded as not the best option a priori in the elderly.	Home sleep apnoea testing is now widely accepted in patients with **high pre-test probability and no significant comorbidity**; polysomnography remains necessary in complex or comorbid older patients.
AHI used as the severity metric.	Newer physiological metrics — **hypoxic burden, ventilatory burden and arousal burden** — predict cardiovascular mortality better than AHI and are moving into risk stratification.

Latest Updates (2023–Present)

- **Tirzepatide (SURMOUNT-OSA, *NEJM* 2024; FDA approval December 2024)** — a dual GIP/GLP-1 receptor agonist reduced AHI by 25–29 events/hour versus placebo at 52 weeks, both in patients on PAP and in those unwilling or unable to use it, with nearly half achieving remission or mild disease. It is an **adjunct to, not a replacement for**, CPAP.
- **Benjafield et al., *Lancet Respiratory Medicine* 2025** — systematic review and meta-analysis of randomised and confounder-adjusted non-randomised studies found positive airway pressure therapy associated with **reduced all-cause and cardiovascular mortality** in OSA, reconciling the negative RCTs with real-world data.
- **Mazzotti et al., *JAMA Network Open* 2024** — in older adults specifically, PAP therapy was associated with lower mortality and cardiovascular risk.
- **ERJ Open Research 2025 review** — blood-pressure lowering with CPAP is seen chiefly in **uncontrolled and treatment-resistant hypertension**, and MACE reduction depends on **good long-term adherence**; RCTs in primary prevention and in the highest-risk groups are still lacking.

- **Hypoglossal nerve stimulation** (Inspire) is an established alternative for CPAP-intolerant patients with moderate-to-severe OSA, BMI below approximately 35 and no complete concentric collapse; it has been used successfully in selected fit older adults.
- **Telemonitoring and auto-titrating CPAP** with remote adherence data and automated feedback have become standard, and are especially valuable for older patients in whom clinic attendance is difficult — relevant to the Indian context where sleep services are concentrated in metropolitan centres.
- **Indian context:** OSA prevalence in Indian urban adults is 9–13%, with a distinctive phenotype of **OSA at lower BMI** owing to craniofacial structure and central adiposity; cost of devices, absence of insurance cover for CPAP and limited polysomnography capacity remain the principal barriers to treatment in older Indians.

EXAMINER'S TIP — verbatim mark-earning lines

- "CPAP splints the upper airway; BiPAP ventilates. Hypercapnic failure needs BiPAP, hypoxaemic failure and OSA need CPAP."
- "Half of all people over 65 have an AHI above 5 — OSA is a geriatric syndrome, not a middle-aged male disease."
- "In older patients, suspect OSA from nocturia, morning headache, falls, resistant hypertension and cognitive decline — not from snoring and sleepiness."
- "CPAP adherence in older adults, including those with dementia, is no worse than in the young — the barrier is education and caregiver support, not age."
- "The negative cardiovascular RCTs (SAVE, RICCADSA, ISAACC) were adherence-limited trials, not evidence that CPAP does not work."
- "Tirzepatide (2024) is the first drug ever approved for obstructive sleep apnoea — an adjunct to CPAP, not a substitute."

Q7. Discuss the diagnosis and management of multidrug-resistant tuberculosis (DOTS-Plus regime).

Textbook coverage: Pathy's (Ch. 38, Respiratory infections) covers tuberculosis in old age — higher infection rates in those over 65, nursing-home outbreaks, atypical presentation with less fever, sweating and haemoptysis, more dyspnoea, fewer cavitary lesions, lower tuberculin and IGRA positivity, and sputum smear/culture as the gold standard — and Essentials covers LTCF screening and preventive therapy. **Neither textbook covers MDR-TB regimens or the DOTS-Plus/PMDT programme.** The management sections below are drawn from the WHO Consolidated Guidelines on Tuberculosis Module 4 (2025), the WHO Operational Handbook, and the Government of India NTEP/PMDT guidelines.

15–20 High-Yield Points

Definitions and Burden

1. **MDR-TB** = resistance to at least **isoniazid and rifampicin**. **RR-TB** = rifampicin resistance with or without other resistance; the two are managed together as **MDR/RR-TB**. **Pre-XDR-TB** = MDR/RR-TB plus resistance to any **fluoroquinolone**. **XDR-TB** = MDR/RR-TB plus fluoroquinolone resistance plus resistance to at least one of bedaquiline or linezolid (2021 revised definition).
2. **DOTS-Plus** is the programmatic strategy for managing MDR-TB within the DOTS framework, requiring the five DOTS elements plus **quality-assured drug-susceptibility testing, second-line drug supply, standardised regimens, active drug-safety monitoring (aDSM) and a recording/reporting system**. In India it is delivered as **PMDT (Programmatic Management of Drug-Resistant TB)** under the **National TB Elimination Programme (NTEP)**.
3. **India carries the largest MDR/RR-TB burden in the world** — roughly a quarter of global cases — with about 1.1–1.3 lakh incident MDR/RR-TB cases annually. Globally there are approximately 4 lakh MDR/RR-TB cases each year.
4. **Mechanisms of resistance**: spontaneous chromosomal mutations (*rpoB* for rifampicin, *katG* and *inhA* for isoniazid, *gyrA/gyrB* for fluoroquinolones, *rrs* and *eis* for injectables, *Rv0678* for bedaquiline) selected by **inadequate treatment** — monotherapy, poor adherence, erratic drug supply, poor-quality drugs and unsupervised private-sector prescribing.

Why the Elderly are Special

5. In older people TB is usually **reactivation of remote infection** as immunosenescence weakens cell-mediated containment; rates in institutional and nursing-home populations exceed those in the community, and staff as well as residents can spread infection.
6. **Presentation is atypical:** older patients are **less likely to report fever, sweating and haemoptysis, more likely to present with dyspnoea**, and have a **lower likelihood of radiographic cavitation** — with more lower-lobe, miliary and extrapulmonary disease. **Suspect tuberculosis whenever an older patient is inexplicably failing.**
7. **Immunological tests are less reliable:** both tuberculin skin testing and IGRAs show **lower positivity in the elderly** despite comparable exposure. A simply positive or negative Mantoux neither confirms nor excludes active disease, and IGRA performance in the very old — most of whom have had lifetime contact with the organism — is not well documented.
8. **Treatment tolerance is poorer:** age-related decline in renal and hepatic clearance, **polypharmacy and QT-prolonging drug interactions**, pre-existing peripheral neuropathy, cataract and hearing loss, and low body weight all raise the risk of adverse events on second-line drugs.

Diagnosis

9. **Universal drug-susceptibility testing is now mandatory** — every diagnosed TB patient in India must have at least rifampicin resistance determined at diagnosis, and no patient should be started on first-line therapy without it.
10. **Rapid molecular tests are the front line: CBNAAT / Xpert MTB/RIF and Xpert Ultra** and **Truenat MTB/Rif Dx** (an indigenous Indian chip-based platform used at peripheral level) detect *M. tuberculosis* and rifampicin resistance within two hours. Specimens: sputum (two samples), or induced sputum, gastric aspirate or bronchoalveolar lavage in the frail or non-expectorating older patient.
11. **Line probe assay (LPA): first-line LPA** detects *rpoB*, *katG* and *inhA* mutations (rifampicin and isoniazid, including high- versus low-level isoniazid resistance); **second-line LPA** detects fluoroquinolone and second-line injectable resistance. **Xpert MTB/XDR** gives resistance to isoniazid, fluoroquinolones, ethionamide and amikacin in a single cartridge.
12. **Culture and phenotypic DST** on liquid medium (MGIT, 2–3 weeks) remains the reference standard and is essential for **bedaquiline, linezolid, clofazimine and delamanid** susceptibility, for treatment monitoring and for regimen modification. **Targeted next-generation sequencing** is now WHO-endorsed for comprehensive resistance prediction directly from sputum.

13. **Baseline pre-treatment evaluation (PTE):** weight and BMI, haemogram, blood sugar (diabetes screening is mandatory), renal and liver function, serum electrolytes including **potassium, magnesium and calcium**, thyroid-stimulating hormone, **HIV testing**, chest radiograph, **ECG for QTc**, visual acuity and colour vision, audiometry where an injectable is contemplated, and a pregnancy test in women of child-bearing age.

Treatment Regimens

14. **Six-month BPaLM is the preferred regimen** for MDR/RR-TB and pre-XDR-TB: **B**edaquiline + **Pa**retomanid + **L**inezolid (600 mg once daily) + **M**oxifloxacin for 6 months, extendable to 9 months. In TB-PRACTECAL, BPaLM achieved **89% treatment success compared with 52% for the standard of care**. Moxifloxacin is dropped (giving **BPaL**) where fluoroquinolone resistance is documented. **Not for pregnant or breastfeeding women, or those with more than 1 month's prior exposure to these drugs unless resistance is excluded.**
15. **Nine-month all-oral bedaquiline-containing regimen** for MDR/RR-TB in whom fluoroquinolone resistance has been **excluded** and who have no extensive disease: 4–6 months of bedaquiline + levofloxacin/moxifloxacin + ethionamide (or 2 months of linezolid) + ethambutol + high-dose isoniazid + pyrazinamide + clofazimine, followed by 5 months of levofloxacin + clofazimine + ethambutol + pyrazinamide.
16. **Longer individualised all-oral regimen (18–20 months)** for extensive, XDR or intolerant disease, built from: **Group A** — levofloxacin/moxifloxacin, **bedaquiline, linezolid** (all three included); **Group B** — **clofazimine, cycloserine/terizidone** (one or both added); **Group C** — ethambutol, delamanid, pyrazinamide, imipenem–cilastatin or meropenem (with amoxicillin–clavulanate), amikacin/streptomycin, ethionamide/prothionamide, p-aminosalicylic acid — added to complete at least four effective drugs. **Injectable-free regimens are now the norm**; kanamycin and capreomycin have been withdrawn.
17. **Isoniazid mono-resistant (Hr) TB:** rifampicin + ethambutol + pyrazinamide + levofloxacin for 6 months (6 REZ-Lfx).

Monitoring, Adverse Effects and Support

18. **Monitoring:** monthly sputum smear and culture (the response is judged on culture conversion), monthly weight, monthly haemogram and renal/liver function, **ECG for QTc at baseline, weeks 2, 4, 8, 12 and monthly thereafter** (bedaquiline, moxifloxacin, clofazimine and delamanid are all QT-prolonging), TSH on ethionamide and PAS, monthly visual acuity/colour vision on ethambutol and linezolid, and audiometry if an injectable is used.
19. **Key adverse effects: linezolid** — myelosuppression, peripheral and optic neuropathy, lactic acidosis (the dominant toxicity, requiring dose reduction to 300 mg or interruption); **bedaquiline** — QT prolongation, hepatotoxicity; **pretomanid** — hepatotoxicity, peripheral neuropathy; **clofazimine** — skin hyperpigmentation, ichthyosis, QT prolongation; **cycloserine** — psychosis, depression, seizures (a particular hazard in the older brain); **fluoroquinolones** — tendinopathy, dysglycaemia, delirium, QT prolongation. Manage through **active drug-safety monitoring (aDSM)** with mandatory reporting.
20. **Adjunctive and programmatic care: pyridoxine** with linezolid, cycloserine and high-dose isoniazid; nutritional support — under NTEP through the **Ni-kshay Poshan Yojana** direct benefit transfer (enhanced to Rs 1000 per month in 2024) and the **Pradhan Mantri TB Mukt Bharat Abhiyaan** with Ni-kshay Mitra community adoption; airborne infection control; contact screening and TB preventive treatment for contacts; treatment support through **DOT providers, video-observed therapy and 99DOTS**; and screening/management of diabetes, HIV, alcohol use and undernutrition.
21. **Geriatric-specific management:** review and reduce polypharmacy before starting, screen for and stop other QT-prolonging drugs (macrolides, antipsychotics, antiemetics, antiarrhythmics), correct hypokalaemia and hypomagnesaemia, adjust doses for weight and renal function, monitor for delirium on cycloserine and fluoroquinolones, arrange family or caregiver-supported adherence, and involve the multidisciplinary team for nutrition, mobility and depression.
22. **Outcomes:** historically MDR-TB treatment success was only 50–60% with 18–24-month injectable regimens; with the all-oral shorter regimens success now approaches **85–90%**, with far lower loss to follow-up. Outcomes in the elderly remain poorer because of comorbidity, drug intolerance and higher all-cause mortality.

Current Practice Update

Textbook Position (Pathy's 6th ed. / Essentials 7th ed.)	Current Evidence & Guidelines (2023–2026)
Pathy's: sputum acid-fast stain and culture remain the gold standard for diagnosis; detection rates are not clearly lower in the elderly.	Still valid for confirmation, but **rapid molecular testing (CBNAAT/Truenat) is now the mandated first diagnostic test**, with universal DST for every notified patient.

Textbook Position (Pathy's 6th ed. / Essentials 7th ed.)	Current Evidence & Guidelines (2023–2026)
Pathy's: tuberculin and IGRA positivity are lower in older patients with similar exposure.	Confirmed; WHO now emphasises that these tests **have no role in diagnosing active TB** and are used only for infection/preventive-treatment decisions.
Classical DOTS-Plus: 18–24-month regimens containing an **injectable** (kanamycin, capreomycin).	**Injectable-containing regimens are obsolete.** Kanamycin and capreomycin have been removed from all WHO regimens because of ototoxicity and inferior outcomes.
Standardised long regimens for all MDR-TB patients.	**Six-month BPaLM is the preferred regimen** for MDR/RR-TB and pre-XDR-TB, with a 9-month all-oral regimen where fluoroquinolone resistance is excluded and longer regimens reserved for complex disease.
XDR-TB defined as MDR plus fluoroquinolone plus second-line injectable resistance.	**Redefined (2021):** MDR/RR-TB plus fluoroquinolone resistance plus resistance to bedaquiline or linezolid; the older category becomes **pre-XDR-TB**.
Linezolid 1200 mg daily used in early BPaL studies.	**Linezolid 600 mg once daily** for both BPaLM and BPaL — the ZeNix trial showed equal efficacy with markedly less myelosuppression and neuropathy.

Latest Updates (2023–Present)

- **WHO Consolidated Guidelines on Tuberculosis, Module 4: Treatment and Care (15 April 2025)** — all previous guidance consolidated into a single document, with a landmark new recommendation for a novel all-oral 6-month regimen, **BDLLfxC** (**b**edaquiline, **d**elamanid, **l**inezolid, **l**evo**fx**oxacin, **c**lofazimine), for MDR/RR-TB with or without fluoroquinolone resistance — derived from the **endTB** trial. Modified 9-month regimens are recommended where fluoroquinolone resistance is excluded.
- **India:** the Union Health Ministry **approved introduction of BPaLM under NTEP on 6 September 2024**, with a countrywide time-bound rollout and structured capacity building. The 2024 National Technical Expert Group also recommended **extending bedaquiline and delamanid to all age groups** and replacing ethionamide with **linezolid in the 9-month shorter oral regimen**.
- **Xpert MTB/XDR and targeted next-generation sequencing** were WHO-endorsed (2023–2024) for rapid detection of resistance to isoniazid, fluoroquinolones, ethionamide, amikacin and beyond; NTEP has begun deploying CBNAAT MTB/XDR at equipped laboratories.
- **TB-PRACTECAL** (*NEJM* 2022) and **ZeNix** (*NEJM* 2022) provided the evidence base for BPaLM and for the reduced linezolid dose; **endTB** (*NEJM* 2024) established three further 9-month all-oral bedaquiline/delamanid-based regimens.
- **Drug-susceptible TB:** the 4-month **HPMZ** regimen (high-dose rifapentine, isoniazid, moxifloxacin, pyrazinamide; Study 31/A5349) is WHO-recommended and is being evaluated for Indian programmatic adoption — relevant because shortening the susceptible regimen reduces the pool from which resistance emerges.
- **India's TB elimination target of 2025** (five years ahead of the SDG target) has been supported by **Ni-kshay Poshan Yojana**, **Pradhan Mantri TB Mukt Bharat Abhiyaan** and the **100-day intensified TB elimination campaign (December 2024)**, with an emphasis on active case-finding in the vulnerable, including the elderly and residents of old-age homes.
- **TB preventive treatment** has moved to shorter rifamycin-based courses — **3HP** (weekly rifapentine + isoniazid for 12 doses), **1HP** (daily rifapentine + isoniazid for 1 month) and **4R** — which are far better tolerated than 6–9 months of isoniazid in older, polypharmacy-burdened patients.

EXAMINER'S TIP — verbatim mark-earning lines

- "MDR-TB is resistance to isoniazid and rifampicin; pre-XDR adds fluoroquinolone resistance; XDR (2021 definition) adds bedaquiline or linezolid resistance."
- "BPaLM — bedaquiline, pretomanid, linezolid, moxifloxacin for six months — achieved 89% success versus 52% with standard care in TB-PRACTECAL, and was approved for NTEP rollout in India in September 2024."
- "All WHO-recommended MDR-TB regimens are now injectable-free; kanamycin and capreomycin have been withdrawn."
- "Linezolid 600 mg once daily — the ZeNix trial showed equal efficacy to 1200 mg with far less myelosuppression and neuropathy."
- "Every MDR-TB regimen contains QT-prolonging drugs — ECG monitoring and correction of potassium and magnesium are mandatory, especially in the polypharmacy-burdened elderly."
- "Group A drugs are levofloxacin/moxifloxacin, bedaquiline and linezolid — all three are included unless contraindicated."
- "In the older patient, suspect tuberculosis when he or she is inexplicably failing — fever, sweats, haemoptysis and cavitation are all less common."

Q8. Discuss malaria — treatment, drug classes, contraindications, side effects, efficacy and prophylaxis.

Textbook coverage: Malaria is **not covered as a disease entity** in Pathy's Principles and Practice of Geriatric Medicine (6th ed.) or Essentials of Clinical Geriatrics (7th ed.); antimalarials appear only incidentally — chloroquine and hydroxychloroquine as drugs that may worsen myasthenia gravis and cause painless myopathy (Ch. 56, Ch. 57), and hydroxychloroquine as a failed COVID-19 therapy (Ch. 41). The answer below is therefore constructed from the **WHO Guidelines for Malaria (13 August 2025)**, the **National Drug Policy on Malaria (NCVBDC/NVBDCP, Government of India)** and current peer-reviewed literature.

15–20 High-Yield Points

Aetiology and Burden

1. Malaria is caused by five species of *Plasmodium* — ***P. falciparum*** (severe disease and death), ***P. vivax*** (relapsing, dominant in India), *P. ovale*, *P. malariae* and ***P. knowlesi*** (zoonotic, South-East Asia) — transmitted by the female *Anopheles* mosquito.
2. **Global burden:** the World Malaria Report 2025 estimates **282 million cases and 610,000 deaths in 2024**, an increase of about 9 million cases on the previous year, driven by drug and insecticide resistance, climate change, conflict and funding contraction.
3. **India:** cases have fallen from around 7.5 crore at Independence to about **2.5 lakh confirmed cases and 86 deaths in 2024** — a fall of more than 97%. India **exited the WHO 'High Burden to High Impact' group in 2024** and targets **zero indigenous cases by 2027 and certification of elimination by 2030**. In 2024 India still accounted for about 73% of the WHO South-East Asia Region's estimated cases.
4. **Why malaria matters in the geriatric patient:** loss of acquired immunity in previously endemic areas, blunted fever, presentation as **delirium, falls or unexplained deterioration** rather than classical paroxysms, a higher risk of severe disease with **renal failure, ARDS, hypoglycaemia and cerebral malaria**, and greater vulnerability to antimalarial toxicity through polypharmacy, QT prolongation and impaired renal and hepatic clearance.

Drug Classes

5. **Quinoline blood schizonticides: chloroquine**, **amodiaquine**, **quinine**, **mefloquine**, **lumefantrine**, **piperaquine**, **pyronaridine** — act by inhibiting **haem detoxification** in the parasite food vacuole.
6. **8-Aminoquinolines (tissue schizonticides/hypnozoiticides and gametocytocides): primaquine** and **tafenoquine** — the only drugs that achieve **radical cure** by eradicating *P. vivax* and *P. ovale* hypnozoites; both cause **haemolysis in G6PD deficiency**.
7. **Artemisinins: artesunate, artemether, dihydroartemisinin, arteether** — endoperoxide bridge generates free radicals on contact with haem iron. They are the **most rapidly acting antimalarials**, killing ring stages and reducing parasite biomass 10,000-fold per cycle, but have a very short half-life, hence the mandatory combination with a longer-acting partner drug.
8. **Antifolates: sulfadoxine–pyrimethamine** (sequential blockade of dihydropteroate synthase and dihydrofolate reductase), **proguanil**. **Antibiotic antimalarials: doxycycline, clindamycin** (apicoplast inhibitors, slow-acting, always used as partners). **Naphthoquinone: atovaquone** (cytochrome bc1 inhibitor), used with proguanil.

Treatment

9. **Uncomplicated *P. falciparum* malaria is treated with an artemisinin-based combination therapy (ACT) for 3 days** — the artemisinin clears the parasite biomass and the partner drug eliminates the residuum. WHO recommends six ACTs: artemether–lumefantrine, artesunate–amodiaquine, artesunate–mefloquine, artesunate–sulfadoxine–pyrimethamine, dihydroartemisinin–piperaquine and artesunate–pyronaridine.
10. **India's National Drug Policy on Malaria** uses **AS+SP (artesunate 3 days + sulfadoxine–pyrimethamine on day 1) as first-line for *P. falciparum* across most of the country**, plus **primaquine 0.75 mg/kg single dose on day 2** as a gametocytocide. In the **north-eastern states**, where SP resistance is established, first-line is **artemether–lumefantrine**.
11. ***P. vivax*: chloroquine 25 mg base/kg over 3 days** (10–10–5 mg/kg) for the blood stage, plus **primaquine 0.25 mg/kg daily for 14 days** for radical cure in India (WHO now advises a higher total dose — 7 mg/kg, given as 0.5 mg/kg/day for 14 days or 1 mg/kg/day for 7 days). Where chloroquine resistance exists, an ACT is used instead.
12. **Severe malaria — the single most important therapeutic point: intravenous artesunate is the drug of choice in all patients, including the elderly, pregnant women in all trimesters and children**, at 2.4 mg/kg at 0, 12 and 24 hours and then daily. It **reduced mortality by 35% compared with quinine in SEAQUAMAT and by 22.5% in**

AQUAMAT. Give at least 24 hours of parenteral therapy, then complete with a full oral ACT course once the patient can swallow.

13. **Supportive management of severe malaria:** hourly glucose monitoring (hypoglycaemia is common, and quinine worsens it by stimulating insulin release), cautious fluid resuscitation with careful attention to pulmonary oedema in the elderly, renal replacement therapy for acute kidney injury or blackwater fever, treatment of seizures, exclusion of concomitant bacterial sepsis, and **avoidance of routine exchange transfusion, corticosteroids and heparin**, none of which improve outcome.

14. **Reducing transmissibility:** in low-transmission areas a **single low dose of primaquine 0.25 mg/kg** should be given with an ACT to patients with *P. falciparum* malaria to kill gametocytes; **G6PD testing is not required** at this dose.

Contraindications, Side Effects and Efficacy

15. **Primaquine and tafenoquine are contraindicated in G6PD deficiency** (haemolysis), in pregnancy, in infants under 6 months and in women breastfeeding infants under 6 months. **G6PD status must guide radical-cure prescribing**; where testing is unavailable, weekly primaquine 0.75 mg/kg for 8 weeks under supervision is the compromise regimen.

16. **Key adverse effects by drug: chloroquine** — pruritus (especially in dark skin), retinopathy on prolonged use, **myopathy and worsening of myasthenia gravis**, QT prolongation; **quinine** — **cinchonism** (tinnitus, deafness, headache, nausea, visual disturbance), **hypoglycaemia**, hypotension on rapid infusion, QT prolongation; **mefloquine** — neuropsychiatric reactions, contraindicated in epilepsy and psychiatric illness; **sulfadoxine–pyrimethamine** — Stevens–Johnson syndrome, agranulocytosis, sulfa allergy; **doxycycline** — photosensitivity, oesophagitis (a real hazard in the older patient — take upright with water), contraindicated in pregnancy; **artemisinins** — generally very well tolerated, with a recognised **post-artesunate delayed haemolysis** at 1–3 weeks requiring follow-up haemoglobin; **piperaquine and halofantrine** — marked QT prolongation.

17. **Geriatric prescribing cautions:** the cumulative QT burden of chloroquine, quinine, mefloquine, piperaquine and lumefantrine on top of existing antipsychotics, antiemetics, macrolides and antiarrhythmics; dose adjustment of quinine in renal and hepatic impairment; risk of hypoglycaemia in the diabetic on sulphonylureas or insulin; and interactions of artemether–lumefantrine with CYP3A4 inhibitors and inducers.

18. **Efficacy and resistance:** ACTs remain highly efficacious, with cure rates above 95% where the partner drug is effective. However, **artemisinin partial resistance** (delayed parasite clearance, mediated by *Pfkelch13* mutations) is entrenched in the Greater Mekong subregion and has now emerged independently in **Eritrea, Rwanda, Uganda, Ethiopia and Tanzania**; **chloroquine resistance in *P. falciparum* is universal in India**, and sulfadoxine–pyrimethamine resistance is established in the north-east. Efficacy is monitored through **therapeutic efficacy studies every 24 months** at sentinel sites.

Prophylaxis and Prevention

19. **Chemoprophylaxis for travellers to endemic areas** (Government of India recommendations): **short-term (< 6 weeks) — doxycycline 100 mg daily**, started 2 days before travel and continued 4 weeks after leaving; **long-term (> 6 weeks) — mefloquine 250 mg weekly**, started 2–3 weeks before and continued 4 weeks after. **Atovaquone–proguanil** (daily, from 1–2 days before to 7 days after) is the best-tolerated option internationally and the preferred choice for short trips and for older travellers because of its short post-travel tail and few interactions.

20. **Terminal prophylaxis** with primaquine (or a single dose of tafenoquine) after leaving an area endemic for *P. vivax* or *P. ovale*, subject to G6PD status, prevents late relapse.

21. **Vector control and personal protection** remain the backbone: **long-lasting insecticidal nets (LLINs)**, **indoor residual spraying**, larval source management, repellents, screened housing and full-sleeved clothing after dusk. Under India's programme these are delivered through **ASHA-led surveillance with rapid diagnostic tests and radical treatment at village level**.

22. **Vaccines** — **RTS,S/AS01 (Mosquirix)** and **R21/Matrix-M** are WHO-recommended for children in areas of moderate-to-high transmission; the latter is manufactured by the Serum Institute of India. **Neither has any role in adult or geriatric practice**, and neither is used in India, where transmission is low and the goal is elimination.

23. **Programmatic framework in India:** the **National Framework for Malaria Elimination (2016–2030)** and the **National Strategic Plan (2023–2027)**, delivered by the **National Centre for Vector Borne Diseases Control (NCVBDC, formerly NVBDCP)**, categorise districts **0 to 3** by annual parasite incidence, with Category 0 districts (zero indigenous cases) entering **prevention of re-establishment** and eligible for subnational verification. By end-2025, **160 districts across 23 states/UTs had reported zero indigenous cases**.

Current Practice Update

Textbook Position (Pathy's 6th ed. / Essentials 7th ed.)	Current Evidence & Guidelines (2023–2026)
Chloroquine and hydroxychloroquine appear in the uploaded textbooks only as causes of myopathy and as aggravators of myasthenia gravis.	These remain valid geriatric cautions, and are the reason to avoid chloroquine in a patient with known neuromuscular disease — but chloroquine now has **no role in *P. falciparum* treatment in India** because of universal resistance.
Hydroxychloroquine trialled for COVID-19 with early observational promise (Ch. 41).	Definitively abandoned — randomised trials showed no benefit and an **excess of cardiovascular events, mainly QT prolongation**.
Quinine was long the standard for severe malaria.	**Intravenous artesunate has replaced quinine** in every patient group (SEAQUAMAT, AQUAMAT); quinine is now only a fallback where artesunate is unavailable.
Primaquine 0.25 mg/kg for 14 days for *P. vivax* radical cure (Indian programme).	**WHO 2025 now recommends a higher total dose of 7 mg/kg** — 0.5 mg/kg/day for 14 days or 1 mg/kg/day for 7 days — and offers **tafenoquine as a single-dose alternative** in patients with ≥ 70% G6PD activity receiving chloroquine.
G6PD status assessed clinically or not at all.	**Qualitative near-patient G6PD tests** are now recommended to guide relapse-prevention regimens (strong recommendation).
Malaria regarded as an endemic disease of high burden in India.	India is now in the **elimination endgame** — exited the WHO HBHI group in 2024, targeting **zero indigenous cases by 2027** and certification by 2030.

Latest Updates (2023–Present)

- **WHO Guidelines for Malaria, 13 August 2025 consolidated edition** — ACT for 3 days for all uncomplicated falciparum malaria; ACT or chloroquine for non-falciparum species in chloroquine-susceptible areas and **ACT where chloroquine resistance exists**; **G6PD status must guide primaquine or tafenoquine**; **tafenoquine recommended as an alternative to primaquine** for relapse prevention in patients aged 2 years and above with ≥ 70% G6PD activity who receive chloroquine.
- **Single low-dose primaquine (0.25 mg/kg) with an ACT** in low-transmission settings to reduce transmissibility (2024 recommendation) — directly relevant to India's elimination strategy, though under-used because it confers no individual clinical benefit.
- **Artemisinin partial resistance** (WHO, January 2025) — independent emergence of *Pfkelch13* validated markers in East Africa (Eritrea, Rwanda, Uganda, Ethiopia, Tanzania) alongside **histidine-rich protein 2/3 (*pfhrp2/3*) gene deletions** that cause false-negative rapid diagnostic tests, prompting a shift towards non-HRP2 or combination RDTs.
- **New and emerging agents: ganaplacide–lumefantrine** (a non-artemisinin combination in phase 2/3), **KAF156**, **artefenomel**, **cipargamin (KAE609)** targeting PfATP4, and **triple ACTs** (e.g. artemether–lumefantrine–amodiaquine; DHA–piperaquine–mefloquine, DeTACT trial 2022–2024) designed to protect partner drugs against resistance.
- **Monoclonal antibodies** — **L9LS** and **CIS43LS** against the circumsporozoite protein conferred high-level protection over a malaria season in phase 2 trials (*NEJM* 2022–2024), pointing towards a passive-immunisation strategy for high-risk groups and outbreak control.
- **Vector-control innovations:** dual-active-ingredient (chlorfenapyr and pyriproxyfen) LLINs against pyrethroid-resistant vectors, spatial repellents, attractive targeted sugar baits, and gene-drive research — alongside the growing threat of ***Anopheles stephensi*** invading urban Africa, a vector originally of Indian cities.
- **India-specific:** the WHO South-East Asia Regional Office confirmed India is on track for the Global Technical Strategy target of a 75% reduction in cases by 2025 against a 2015 baseline; **Mizoram and Tripura** remain the last Category 3 (high-burden) states, and forest-dwelling, tribal and migrant populations are the principal residual reservoirs.

EXAMINER'S TIP — verbatim mark-earning lines

- “Intravenous artesunate is the treatment of choice for severe malaria in every patient group — it reduced mortality by 35% versus quinine in SEAQUAMAT.”
- “India's first-line for falciparum malaria is artesunate plus sulfadoxine–pyrimethamine, except in the north-eastern states where artemether–lumefantrine is used.”
- “Only the 8-aminoquinolines — primaquine and tafenoquine — kill hypnozoites and achieve radical cure; both demand G6PD testing.”
- “A single low dose of primaquine, 0.25 mg/kg, is gametocytocidal and does not require G6PD testing.”
- “Artemisinin partial resistance means delayed parasite clearance, not treatment failure — the failure comes when the partner drug is also resistant.”
- “Quinine causes cinchonism and hyperinsulinaemic hypoglycaemia; artesunate causes delayed haemolysis at one to three weeks — always recheck the haemoglobin.”
- “India exited the WHO High Burden to High Impact group in 2024 and targets zero indigenous cases by 2027, certification by 2030.”
- “RTS,S and R21 vaccines are for children in moderate-to-high transmission settings only — they have no place in Indian geriatric practice.”

PREVENTIVE GERIATRICS

Immunization & Cancer Screening in the Elderly

MD Geriatric Medicine — Examination Compendium

Seven Compiled Question–Answer Sets

15-Mark Format with Current Practice Updates

Primary sources: Pathy's Principles and Practice of Geriatric Medicine, 6th edition (2022) • Essentials of Clinical Geriatrics, 7th edition

Updates: Peer-reviewed literature, international guidelines and society recommendations, 2023 – mid-2026, with Indian context integrated throughout (API, ICMR, IAG, LASI, NP-NCD).

Contents

1.	Immunization schedule for a fit elderly patient, with evidence-based reasons	15 marks
2.	Current guidelines and status of vaccination in older persons, with recent evidence	15 marks
3.	Travel-related vaccination in the elderly — CDC guidelines	15 marks
4.	Cancer screening in the elderly — currently accepted strategies	15 marks
5.	Screening for colorectal cancer	15 marks
6.	Mammography — screening the older woman	15 marks
7.	Prostate-specific antigen — to screen or not to screen?	15 marks
8.	**Rapid Revision Grid** — key numbers, trials and cut-offs	Appendix

How to use this compendium

Every answer follows the same architecture: **high-yield textbook points → Latest Updates (2023-present) → Current Practice Update comparison table**. The comparison table is the highest-value section for the examiner — it demonstrates that you know both the textbook position and where current evidence has moved beyond it. The **Rapid Revision Grid** at the end compiles every number and trial name in this compendium onto a few pages for last-night revision.

Question 1. Immunization schedule for a fit elderly patient, with evidence-based reasons.

Primary source: Pathy's, 6th ed. — "Preventive Geriatrics"; Essentials of Clinical Geriatrics, 7th ed.

A. High-Yield Points (Textbook Content)

PRINCIPLES

1. **Immunization is primary prevention** in geriatrics — the aim is to prevent infection-related **hospitalisation, functional decline and death**, not merely infection.
2. **Immunosenescence** makes older adults respond less well to vaccines; hence **higher-dose, adjuvanted, conjugate or recombinant** preparations are preferred in this age group.
3. Pathy's recommends tailoring vaccination to the patient's **functional category** — *Fit, Frail, Moderately demented, End of life* — and to **probable life expectancy**. A **fit elderly patient receives the complete schedule.**

INFLUENZA VACCINE

4. Give **annually to all older patients in every category**, ideally in **early autumn** (pre-monsoon / winter in India).
5. **More than 90% of deaths** from pneumonia and influenza during epidemics occur in people **aged ≥65 years**.
6. Vaccination reduces the risk of influenza by about **50%** and influenza-related mortality by about **70%**; it also reduces hospitalisation and is **cost-effective**.
7. In nursing homes it is only about **40% effective against clinical illness**, but is **much more effective in preventing pneumonia, hospitalisation and death**. Vaccinating **>80% of residents prevents outbreaks**.
8. **Adjuvanted influenza vaccine** is especially valuable in institutionalised elderly and those with **diabetes, heart failure or respiratory failure**.
9. **Vaccinate healthcare staff and close family contacts** — this reduces respiratory infection and mortality among the elderly they care for (**"cocooning"**).

PNEUMOCOCCAL VACCINE

10. Textbook schedule: **one dose PCV13 followed by PPSV23 one year later**, after age 65. If **PPSV23 was already given**, only **PCV13** is needed. Pathy's states there is **no recommendation on re-immunisation**; other tables quote a repeat **every 3–5 years**.
11. Antibody response is **weaker than in young adults**, but the vaccine still reduces **invasive pneumococcal disease and pneumococcal bacteraemia** in high-risk elders.
12. **CAPITA trial** (community-dwelling >65 years): PCV13 reduced **vaccine-type pneumococcal pneumonia by 45%**. A large **Japanese nursing-home RCT** showed PPSV23 reduced **pneumonia incidence and mortality**.
13. Pneumococcal plus annual influenza vaccine should be offered to **all patients with COPD**.

TETANUS–DIPHTHERIA

14. **Over half of all tetanus cases occur in people aged ≥60 years** — a strong argument for boosters in the elderly.
15. **Td booster every 10 years** (every **5 years** for a tetanus-prone wound). If the primary series is absent or uncertain: **0.5 mL Td IM at 0, 2 months and 6 months**. At least **one dose should be given as Tdap** if never received.

HERPES ZOSTER

16. Herpes zoster occurs in up to **half of individuals aged ≥85 years**; **post-herpetic neuralgia** affects **>40% of those over 60**.
17. **Zostavax (live, 2006) is only 30% protective; Shingrix (recombinant, 2016) is ~90% protective.** Give **2 doses of Shingrix to all adults ≥50 years**, **regardless of past chickenpox** and **even if Zostavax was received earlier**.

COVID-19 AND OTHERS

18. **SARS-CoV-2 vaccines reduce severe disease and mortality**; indicated for adults **>65 years**, healthcare professionals and **family members in contact with older individuals**.
19. **No vaccine is used for tuberculosis** in the elderly — **isoniazid 300 mg/day for 9–12 months** is given for **skin-test converters** in selected residents. Tuberculin reaction may be **falsely negative due to anergy**.
20. The textbook records **two controversies**: ecological data showing rising influenza/pneumonia hospitalisation despite rising vaccine uptake (**Simonsen et al.**), and continuing debate on the **cost-effectiveness of pneumococcal vaccine**.

QUICK SCHEDULE TABLE (TEXTBOOK-BASED)

Vaccine	Fit elderly schedule
Influenza	Every year, early autumn
Pneumococcal	PCV13 → PPSV23 one year later (textbook table: repeat every 3–5 years)
Tetanus (Td/Tdap)	Booster every 10 years; at least one dose as Tdap
Herpes zoster (RZV)	2 doses, once, age ≥50
COVID-19	As per current guidelines

B. Latest Updates (2023–Present)

1. RSV vaccine — the biggest addition to the elderly schedule. ACIP now recommends a single dose of **Arexvy, Abrysvo or mResvia** for unvaccinated adults; additional doses are not currently recommended. Vaccination is **universal at ≥75 years** and **risk-based at 60–74 years**; in April 2025 ACIP extended the risk-based recommendation down to **50–59 years** for those at increased risk of severe RSV disease (*CDC/ACIP, 2023–2025*). Evidence: **AReSVi-006** showed **82.6% efficacy** against RSV lower respiratory tract disease in adults ≥60, and **94.6%** in those with at least one underlying condition; cumulative efficacy over three seasons was **62.9%**, with **67.4%** against severe RSV-LRTD, falling to **48% in the third season** (*Lancet Respiratory Medicine, 2025*).

2. Pneumococcal schedule simplified to a single dose. ACIP issued recommendations for the **21-valent pneumococcal conjugate vaccine (PCV21)** in adults in 2024, and adults who previously completed PCV13 + PPSV23 after age 65 may receive one dose of PCV20 or PCV21, or no further vaccine. The sequential PCV13 → PPSV23 pathway is being replaced by **one dose of PCV20 or PCV21** (*MMWR, 2024–2025*).

3. Influenza — product preference and the DANFLU-2 result. For age ≥65, **HD-IIV3, RIV3 or aIIV3 is preferred**; any age-appropriate vaccine may be used if these are unavailable (*CDC Adult Immunization Schedule, 2025–26*). However, **DANFLU-2**, the largest randomised influenza vaccine trial ever conducted, found that high-dose vaccine did **not** significantly reduce hospitalisation for influenza or pneumonia compared with standard dose (*NEJM, December 2025*). A prespecified secondary analysis of **332,438 adults ≥65** showed cardiorespiratory hospitalisation of **2.25% with high-dose versus 2.38% with standard dose**, driven mainly by fewer cardiovascular hospitalisations (**1.30% vs 1.40%**) (*JAMA Cardiology, 2025*).

4. Zoster vaccine and dementia — a major new off-target signal. A Welsh natural experiment using a date-of-birth eligibility cut-off found that live zoster vaccination reduced new dementia diagnoses by about **one-fifth over 7 years** (*Nature, 2025*). A follow-up showed reduced mild cognitive impairment and fewer dementia deaths among those already living with dementia (*Cell, 2025*). AS01-adjuvanted shingles and RSV vaccines were also associated with reduced 18-month dementia risk in **436,788 matched individuals** (*npj Vaccines, 2025*). Zoster vaccination has additionally been linked to a **23% lower risk of cardiovascular events** including stroke, heart failure and coronary heart disease (*European Heart Journal, 2025*).

5. COVID-19 policy has shifted. For the 2025–26 season ACIP moved COVID-19 vaccination from a universal recommendation to **shared clinical decision-making**, with an annually updated formulation (*MMWR, 2025*).

6. India — API Consensus Guidelines (essential for the Indian viva). The Association of Physicians of India, with other societies, released an exhaustive **adult immunization consensus guideline in 2024**, emphasising "bridging childhood vaccines" in adult care. The **2026 Update** involves **23 national and regional associations** and adopts a **life-course immunization framework** based on age bands and risk profiles rather than disease category. Key India-specific changes:

- **PCV20** included as the **preferred single-dose option**, replacing sequential PCV13 → PPSV23
- **RZV for all adults ≥50 years** irrespective of prior shingles, and for **immunocompromised adults ≥18 years** (cancer, CKD, CVD, transplant, biologics), as **2 doses 2–6 months apart**
- **Annual influenza vaccination for all adults over 50**, framed as **chronic-disease risk reduction**, not just seasonal protection
- **Standardised adult immunization cards** with written records and linkage to electronic health records
- HPV positioned as **preventive oncology**, gender-neutral, with a 3-dose schedule in older and immunocompromised adults

> **Practical Indian note**
>
> Adult vaccines are **not part of the Universal Immunization Programme or NPHCE**; they remain largely **out-of-pocket**, which is the single biggest barrier to uptake.

C. Current Practice Update

Aspect	Pathy's / Essentials (Textbook)	Current Evidence (2023–2026)

Pneumococcal	PCV13 → PPSV23 one year apart; no re-immunisation advice	**Single dose PCV20 or PCV21**; sequential schedule obsolete; API 2026 prefers PCV20
Influenza product	Standard vaccine; adjuvanted vaccine in institutionalised elderly	**HD-IIV3 / RIV3 / aIIV3 preferred ≥65**; but **DANFLU-2 neutral** for primary hospitalisation endpoint; **trivalent** formulations (B/Yamagata dropped)
RSV	Not part of the schedule	**New core elderly vaccine** — single dose, universal ≥75, risk-based 50–74
Zoster	Shingrix ~90% protective; prevents zoster and PHN	Same, **plus** signals for reduced **dementia, MCI and cardiovascular events**
COVID-19	Recommended for all >65	**Shared clinical decision-making**; annually updated formulation
Framework	Vaccination by **functional category / life expectancy**	**Life-course immunization** by age band + risk profile (API 2026); standardised **adult immunization card**
Age threshold	Most recommendations start at **65**	Several now start at **50** (zoster, pneumococcal, risk-based RSV, influenza in API 2026)

Examiner's Tip

Open with the framing line: "*In a fit older adult the objective of immunization is not merely to prevent infection but to prevent hospitalisation, functional decline and death — and because of immunosenescence, the elderly need higher-antigen, adjuvanted or recombinant preparations rather than standard formulations.*"
Then give the schedule as a table. Close with: "*The five core vaccines in a fit older adult today are influenza, pneumococcal, herpes zoster, RSV and COVID-19, with tetanus boosters every ten years.*"
Quote three numbers if nothing else: **>90% of influenza deaths occur above 65; Shingrix 90% vs Zostavax 30%; RSV vaccine efficacy 82.6% in the first season.**

Question 2. Current guidelines and status of vaccination in older persons, with recent evidence.

Primary source: Pathy's, 6th ed.; Essentials of Clinical Geriatrics, 7th ed. Current guidelines cited separately.

A. High-Yield Points

I. TEXTBOOK BASELINE

1. **Vaccination is the single most cost-effective primary preventive measure in geriatrics.** Pathy's advises tailoring it to the patient's **functional category** and **probable life expectancy**.
2. **Immunosenescence and inflammaging** reduce vaccine response. Pathy's notes that **frailty impairs vaccine-induced antibody response** and increases post-vaccination influenza infection in community-dwelling elders — hence **adjuvanted and high-antigen preparations** are preferred.
3. **Influenza vaccine — yearly, in all categories.** Over **90% of pneumonia and influenza deaths** in epidemics occur in those ≥65. It reduces influenza risk by **~50%** and influenza mortality by **~70%**.
4. In **nursing homes**, efficacy against clinical illness is only **~40%**, but protection against **pneumonia, hospitalisation and death is much better**; vaccinating **>80% of residents prevents outbreaks**.
5. **Vaccinate healthcare staff, relatives and regular visitors** — reduces respiratory infection and mortality in residents. Pathy's states this explicitly.
6. **Pneumococcal vaccine** — textbook schedule is **PCV13 followed by PPSV23 one year later after 65**; PPSV23 alone is the practice in the UK, USA, Germany, Canada, Australia and Japan. **CAPITA** showed PCV13 cut vaccine-type pneumococcal pneumonia by **45%**; a Japanese nursing-home RCT showed PPSV23 reduced **pneumonia and mortality**.
7. **Tetanus** — **Td booster every 10 years** (5 years for a tetanus-prone wound); **>50% of tetanus cases occur in people ≥60**; at least one booster should be **Tdap**.
8. **Herpes zoster** — **Shingrix ~90% protective** versus **Zostavax ~30%**; two doses for all ≥50 regardless of chickenpox history. Essentials records for the live vaccine: **HZ HR 0.45, ophthalmic HZ HR 0.37, HZ hospitalisation HR 0.35**.
9. **Essentials (Nichol 2007):** influenza vaccination in community-dwelling elders gave **HR 0.75 for pneumonia/influenza hospitalisation** and **HR 0.52 for death**. The textbook also records the counter-argument (**Simonsen**) that hospitalisation rates rose despite rising coverage, and the ongoing debate on **pneumococcal cost-effectiveness**.
10. Both textbooks predate — and therefore do not cover — **RSV vaccines, PCV20/PCV21, preferential high-dose influenza vaccine, off-target effects of zoster vaccine, and Indian adult immunization guidelines.**

II. CURRENT GUIDELINE STATUS (GLOBAL)

11. **Influenza — annual for all older adults.** For age ≥65, **HD-IIV3, RIV3 or aIIV3 is preferred**; if none is available, any age-appropriate vaccine may be used. All formulations are now **trivalent** (B/Yamagata removed).
12. **Pneumococcal — the sequential schedule is obsolete.** On **23 October 2024** ACIP recommended a **single dose of PCV for all PCV-naïve adults aged ≥50 years**, using **PCV20 or PCV21 alone**, or PCV15 in series with PPSV23. The age threshold was formally lowered from **65 to 50 years**.
13. **RSV — now a core elderly vaccine.** First approved May 2023 for adults ≥60. Current position: **universally recommended at ≥75 years**, **risk-based at 60–74 years**, extended in April 2025 to **risk-based 50–59**. A **single dose only**.
14. **Herpes zoster** — RZV two doses for all ≥50 years, now emphasised also for **immunocompromised adults ≥18 years**, given **2–6 months apart**.
15. **COVID-19 — policy has narrowed.** For 2025–26, ACIP moved from a universal recommendation to **shared clinical decision-making**.
16. **Td/Tdap unchanged** — booster every 10 years, with at least one lifetime Tdap.
17. **Individualisation is the new theme.** The AGS position (*JAGS, 2025*) is that guidelines list recommendations by chronological age, but **biological sex, frailty, functional status and multimorbidity** modify vaccine responses and outcomes, and clinicians must increasingly account for this heterogeneity — as well as overcome barriers to uptake in long-term care.

III. CURRENT STATUS IN INDIA

18. **There is no unified national guideline for geriatric immunization in India**; society recommendations are heterogeneous and evidence-limited. Coverage among Indian older adults is **<3% for most vaccines**, with barriers including absent national guidelines, poor awareness, cost, accessibility and **ageism** (*Indian Academy of Geriatrics*

position statement, 2026).

19. **LASI Wave 1 (2017–18, n = 31,464 aged ≥60) coverage figures — high-yield numbers: diphtheria–tetanus 2.75%**, typhoid 1.84%, hepatitis B 1.82%, **influenza 1.59%**, **pneumococcal 0.74%**; coverage was highest in the most affluent groups. Male sex, urban residence, wealthier household, more schooling and existing medical conditions predicted uptake.
20. **Guideline development in India is now rapid:** API issued the first exhaustive **Indian Consensus Guideline on Adult Immunization in 2024**. The **2026 Update** involves **23 associations** and adopts a **life-course immunization framework**, with **PCV20 preferred**, **RZV for all ≥50**, **annual influenza ≥50**, and standardised **adult immunization cards**. The **Indian Academy of Geriatrics** published a dedicated **position statement on vaccination for older adults ≥60 years** in 2026.
21. **Programmatic gap:** adult vaccines are **not included in the Universal Immunization Programme or NPHCE**, so they remain **out-of-pocket** — the main reason for low uptake.

B. Latest Updates (2023–Present)

1. RSV vaccine efficacy and durability. AReSVi-006 showed **82.6% efficacy** against RSV lower respiratory tract disease in adults ≥60, and **94.6%** in those with at least one underlying condition. Cumulative efficacy over three seasons was **62.9%**, with **67.4%** against severe RSV-LRTD, falling to **48% in the third season**. The authors conclude protection lasts **at least three RSV seasons** despite waning, and that the **optimal revaccination strategy is still undefined** (*Lancet Respiratory Medicine, 2025*). For RSVpreF (Abrysvo), the **RENOIR** trial has reported efficacy across **two seasons** (*NEJM, 2023*).

2. DANFLU-2 — the largest randomised influenza vaccine trial ever conducted. In **332,438 adults ≥65** randomised to high-dose versus standard-dose vaccine, high-dose did **not** significantly reduce hospitalisation for influenza or pneumonia — a **neutral primary endpoint** (*NEJM, December 2025*). In a prespecified secondary analysis, cardiorespiratory hospitalisation was **2.25% vs 2.38%**, driven mainly by fewer **cardiovascular hospitalisations (1.30% vs 1.40%)**, described as exploratory in a neutral trial (*JAMA Cardiology, 2025*). A further analysis found fewer cardiorespiratory, cardiovascular and influenza hospitalisations with high-dose vaccine **regardless of diabetes status** (*JAMA Internal Medicine, 2026*).

3. Zoster vaccine and dementia — the most discussed geriatric vaccine finding of the decade. Using the Welsh date-of-birth eligibility cut-off (born before versus after 2 September 1933, with vaccine uptake jumping from **0.01% to 47.2% across one week**), live zoster vaccination reduced new dementia diagnoses by about **one-fifth over 7 years** (*Nature, 2025*). A 2025 extension found HZ vaccination also reduced **mild cognitive impairment diagnoses** and, among people already living with dementia, **deaths due to dementia**, with effects not confined to one dementia subtype (*Cell, 2025*). In **436,788 propensity-matched individuals**, AS01-adjuvanted shingles and RSV vaccines were associated with **lower 18-month dementia risk** (*npj Vaccines, 2025*). Zoster vaccination has also been linked to a **23% lower risk of cardiovascular events** (*European Heart Journal, 2025*).

4. Pneumococcal — simplification plus wider serotype coverage. The ACIP expansion to adults ≥50 was formally published in *MMWR 2025;74(1):1–8*. Supporting data include the **STRIDE-8** trial of PCV21 in high-risk adults, with immunogenicity comparable to PCV15 and PPSV23.

5. India-specific evidence base. An *Indian Journal of Medical Research* rapid review (2024) documented **influenza vaccine uptake of ≤2%** in adults ≥45 years and hepatitis B uptake of only **55–64% even among healthcare workers** in Delhi and Mumbai. Indian advocacy documents highlight two influenza peaks (monsoon and winter) creating **year-round exposure**, a case-fatality rate of **20–25% for invasive pneumococcal disease in ≥65 years** versus 5–10% in younger adults, and a **lifetime herpes zoster risk of ~30%, rising to ~50% by age ≥85**.

C. Current Practice Update

Vaccine / Issue	Pathy's & Essentials	Current guidelines (2023–2026)
Age to start	Most recommendations begin at **65**	Several now begin at **50** — pneumococcal, zoster, risk-based RSV, influenza (API 2026)
Pneumococcal	PCV13 → PPSV23 one year apart	**Single dose PCV20 or PCV21**; ACIP age lowered 65 → 50 (Oct 2024); PCV21 covers ~84% of IPD serotypes
Influenza product	Standard vaccine; adjuvanted in institutionalised or chronic disease	**HD-IIV3 / RIV3 / aIIV3 preferred ≥65**; trivalent; **DANFLU-2 primary endpoint neutral**, benefit mainly cardiovascular and exploratory
RSV	Not mentioned	**New core elderly vaccine** — single dose, universal ≥75, risk-based 50–74; 82.6% season 1, 62.9% cumulative over 3 seasons

Zoster	Prevents zoster and PHN only	Same, **plus** reduced **dementia, MCI, dementia deaths and cardiovascular events**
COVID-19	Recommended for all >65	**Shared clinical decision-making** (2025–26); annually updated formulation
Framework	By **functional category and life expectancy**	**Life-course immunization**; **adult immunization card**; individualisation by **frailty and function** (JAGS 2025)
India	Not addressed	API 2024 → **API 2026 Update (23 societies)**; **IAG position statement 2026**; coverage still **<3%**, outside UIP/NPHCE

Examiner's Tip

This question rewards **structure over content**. Answer in three tiers: *(i) the biology — immunosenescence and frailty blunt vaccine response; (ii) the global guideline position, vaccine by vaccine; (iii) the Indian status, which is the gap.*
The line that earns marks: "*Globally the framework has shifted from chronological age to life-course, risk-profile-based immunization; in India the problem is not the guideline but the programme — adult vaccines lie outside both the UIP and NPHCE, so coverage remains under 3%.*"

Extra Points to Remember

LASI coverage figures are the single most examinable Indian data set: DT 2.75%, typhoid 1.84%, hepatitis B 1.82%, influenza 1.59%, **pneumococcal 0.74%** — remember the order and remember that pneumococcal is lowest.
Three trial names to have ready: CAPITA (pneumococcal), AReSVi-006 (RSV), DANFLU-2 (high-dose influenza).
One conceptual line: vaccination in old age is a *frailty-modified* intervention — the frailer the patient, the greater the potential benefit from preventing hospitalisation, but the weaker the immunological response.

Question 3. Travel-related vaccination in the elderly — CDC guidelines.

CDC Yellow Book / ACIP guidance. The uploaded textbooks contain no travel-medicine vaccination chapter; Pathy's does note that air travel spreads influenza, TB, measles, SARS/MERS and COVID-19 through close cabin proximity, and that schistosomiasis must be considered in returning travellers.

A. High-Yield Points

PRINCIPLES

1. **Age alone is not a contraindication to travel or to vaccination.** The pre-travel consultation is a **risk assessment**, not a fixed schedule.
2. Vaccine selection depends on **destination, duration, season, itinerary, planned activities, type of accommodation and purpose of travel**, plus the patient's **health status and current medications**.
3. **Immunosenescence** lowers seroconversion and shortens duration of protection, so older travellers need **earlier vaccination, longer lead time, and sometimes serological confirmation**.
4. **Ideal timing: pre-travel consultation 4–6 weeks before departure**, because most travel vaccines need multiple doses and two weeks for protective immunity. For **last-minute travellers**, accelerated schedules can be used.
5. CDC groups travel vaccines into three categories — **Routine, Required and Recommended**.

CATEGORY 1 — ROUTINE VACCINES

6. **Catch up routine age-appropriate vaccines first.** Influenza and pneumococcal vaccines are recommended for older persons whether or not they travel; travel-specific vaccines are discussed case by case, weighing vaccine risk against the risk of acquiring the disease during travel.
7. Ensure **influenza (annual), pneumococcal, Td/Tdap (10-yearly), herpes zoster (RZV), RSV and COVID-19** are up to date. **Influenza is the most common vaccine-preventable infection in travellers** — remember the **Southern Hemisphere season (April–September)** and **year-round transmission in the tropics**.
8. **MMR** — older adults born before 1957 are usually presumed immune; still important for travel to measles-outbreak areas.

CATEGORY 2 — REQUIRED VACCINES (INTERNATIONAL HEALTH REGULATIONS)

9. **Yellow fever (YF)** is the only vaccine **required under IHR**. The certificate becomes valid **10 days after vaccination** and is now **valid for life**. **India requires a YF certificate from travellers arriving from endemic countries**, failing which **6 days' quarantine** applies.
10. **Age ≥60 years is a CDC "precaution", not a contraindication, for yellow fever vaccine** — the single most examinable point. CDC lists the precautions to YF vaccine as age 6–8 months, **age ≥60 years**, asymptomatic HIV with moderate immunosuppression, pregnancy and breastfeeding.
11. **Why the precaution:** the incidence of **YEL-AND (neurologic disease)** is 0.8 per 100,000 doses overall but rises to **2.2 per 100,000 doses in people aged ≥60**. **YEL-AVD (viscerotropic disease)** mimics wild-type yellow fever with **multi-organ failure and ~48% mortality**. A meta-analysis found serious adverse events **3 times higher** in the elderly, **5 times higher above 70 years**, and viscerotropic disease **6 times higher** than in those under 60.
12. **Practical rule:** give YF vaccine to an older traveller **only if truly going to a high-risk endemic area**; if exposure risk is low, issue a **Medical Letter of Exemption (waiver)** on official letterhead, signed and stamped, plus advice on strict **mosquito-bite avoidance**.
13. **Meningococcal ACWY is required by Saudi Arabia for Hajj and Umrah** — highly relevant to Indian elderly pilgrims. Must be given **≥10 days before arrival**; valid for **3–5 years** depending on vaccine type.
14. **Polio** — a booster dose of **IPV** is required for travellers leaving certain polio-affected countries, documented on the International Certificate of Vaccination.

CATEGORY 3 — RECOMMENDED BY DESTINATION

15. **Hepatitis A** — recommended for almost all travellers to developing countries. Older Indians often have **natural immunity from childhood exposure**, so **pre-vaccination serology (anti-HAV IgG) is cost-effective**. Risk of **fulminant hepatitis rises sharply with age**.
16. **Typhoid** — two options: **Vi polysaccharide (injectable, inactivated)** and **Ty21a (oral, live)**. Prefer the **injectable Vi vaccine** in older or immunosuppressed travellers.
17. **Hepatitis B** — for long stays, medical tourism, dialysis abroad, dental or surgical care, or unscreened blood transfusion risk.
18. **Rabies** — pre-exposure prophylaxis for long stays, rural travel, or poor access to immunoglobulin. Now a **2-dose regimen (day 0 and 7)**. Important in the elderly because **post-exposure rabies immunoglobulin is often unavailable abroad**.

19. **Japanese encephalitis** — for stays **≥1 month in rural or agricultural endemic areas** during transmission season. Note that **advancing age is itself a risk factor for symptomatic JE and poor outcome**.
20. **Cholera (CVD 103-HgR)** is a **live oral vaccine** — used only for high-risk relief or outbreak settings and best avoided in the immunosuppressed. **Tick-borne encephalitis** for forested Central and Eastern Europe.

SPECIAL PRECAUTIONS IN OLDER TRAVELLERS

21. **Avoid or carefully weigh live vaccines** — yellow fever, Ty21a oral typhoid, oral cholera, MMR, varicella — especially with **steroids, biologics, malignancy, or thymus disorders** (an absolute contraindication for YF).
22. **Check drug interactions:** proton pump inhibitors and antibiotics inactivate **oral typhoid and cholera vaccines**; **mefloquine** is avoided with conduction defects, bradyarrhythmias, psychiatric illness and epilepsy; **doxycycline** causes pill oesophagitis and photosensitivity; **atovaquone-proguanil** needs a creatinine check.
23. **Non-vaccine measures matter more in the elderly** — insect-bite avoidance, food and water precautions, VTE prophylaxis on long flights, medication supply in hand baggage with a doctor's letter, travel insurance covering pre-existing disease, and a **travel health kit**.

B. Latest Updates (2023–Present)

1. CDC Yellow Book 2026 edition is now current (published 2025). Its structure includes *The Pre-Travel Consultation*, *Vaccination and Immunoprophylaxis — General Principles*, *Medication and Vaccine Interactions in Travel Medicine*, and *Yellow Fever Vaccine and Malaria Prevention Information, by Country*. There is **no longer a standalone "Older Adults" chapter**; guidance for older travellers is now embedded within **"Travelers with Chronic Illnesses"** and **"Immunocompromised Travelers"**, reflecting a shift from **chronological age to comorbidity- and immune-status-based risk assessment** (*CDC Yellow Book 2026*).

2. COVID-19 and travel. The Yellow Book 2026 advises that travellers at high risk of severe COVID-19 — specifically **frail older adults with serious comorbid conditions** and the severely immunocompromised — be counselled to be up to date on vaccination, told when and where to seek care abroad, and made aware of treatment options. **Pemivibart (Pemgarda)** is available as pre-exposure prophylaxis for some severely immunocompromised people, and antivirals require a **drug–drug interaction check** (*CDC Yellow Book 2026*).

3. Yellow fever durability in older adults. A 10-year follow-up of travellers vaccinated at **≥60 years** showed **waning neutralising antibody** despite the lifelong-validity certificate — so serology may be considered before repeat high-risk travel rather than routine revaccination (*Journal of Travel Medicine, 2021*).

4. RSV vaccination is now part of the pre-travel review for older adults, since travel and cruise settings are recognised amplifiers of respiratory viral transmission (*ACIP, 2023–2025*).

5. Indian context. The **API Indian Consensus Guidelines on Adult Immunization 2026 Update** adopt a **life-course framework** and recommend a **standardised adult immunization card** — practically useful for documenting travel and routine vaccines together in the older Indian traveller (*API, 2026*).

C. Current Practice Update

Issue	Older / traditional position	Current CDC position
Yellow fever certificate	Booster every **10 years**	**Valid for life** after a single dose (IHR amendment); boosters only for special groups
Age ≥60 and YF vaccine	Often treated as a contraindication	A **precaution** — give only if genuine high-risk exposure; otherwise issue a **medical waiver letter**
Rabies pre-exposure	3 doses (day 0, 7, 21/28)	**2-dose schedule (day 0, 7)** for most travellers
Structure of guidance	Dedicated "Older Adults" chapter	Age-based chapter removed; folded into **chronic illness / immunocompromised** chapters — risk judged by **comorbidity and immune status, not age**
Hepatitis A in Indian elderly	Vaccinate all travellers	**Check anti-HAV IgG first** — most older Indians are already immune
Respiratory vaccines pre-travel	Influenza ± pneumococcal	Influenza + **PCV20/PCV21** + **RSV** + COVID-19 as appropriate

> **Examiner's Tip**
>
> Structure the answer on the **CDC three-category framework — Routine, Required, Recommended**. This alone signals that you know the CDC system rather than reciting a vaccine list.
> The mark-earning line: "*In the older traveller, age ≥60 is a precaution — not a contraindication — to yellow fever vaccine, because YEL-AND rises from 0.8 to 2.2 per 100,000 doses and viscerotropic disease carries almost 50% mortality; if the itinerary is low-risk, the correct action is a medical waiver letter with strict bite-avoidance advice, not vaccination.*"
> Finish with the Indian angle: **Hajj/Umrah meningococcal ACWY, and anti-HAV IgG serology before hepatitis A**

vaccine.

Question 4. Cancer screening in the elderly — currently accepted strategies.

Primary source: Pathy's, 6th ed. — "Preventive Geriatrics"; Essentials of Clinical Geriatrics, 7th ed. — "Prevention".

A. High-Yield Points (Textbook Content)

GENERAL PRINCIPLES

1. **Screening is secondary prevention** — detection of disease in the preclinical phase. Essentials states that **the value of screening depends on the availability of an effective intervention and the likelihood that the intervention will change the clinical course**.
2. In the elderly, three questions must be answered before screening: **Is life expectancy long enough to benefit? Will the patient accept diagnostic work-up and treatment? Do benefits outweigh harms?**
3. **The "10-year rule"** — most guidelines advise continuing screening only if **life expectancy is 10 years or more**, because that is the **lag time to mortality benefit** for breast and colorectal cancer.
4. **Cancer biology may differ in old age**: although incidence and prevalence rise with age, **the rate of growth may be slower**, which increases the risk of **overdiagnosis**.
5. Essentials gives sobering figures: **the mean age of detection of the commonest cancers is about 68 and the mean age of death about 72**. For breast cancer, median age at diagnosis is ~61 and at death ~69.
6. **Pathy's screens by functional category, not chronological age — Robust, Frail, Moderately demented, End of life**.

PATHY'S SCREENING TABLE (HIGH-YIELD FOR VIVA)

Test	Robust	Frail	Moderately demented	End of life
Breast examination	Yearly	Yearly	Yearly	—
Mammography	Every 1–2 years **up to 80**	Consider every 1–2 years **up to 75**	Consider every 1–2 years **up to 70**	—
Pap smear	Consider **1–3 smears if never had one**	—	—	—
Faecal occult blood	Yearly	Consider yearly	Consider yearly	—
Colonoscopy	Consider every 10 years	—	—	—
PSA	Discuss pros and cons with patient	Discuss with patient	Discuss with caregiver	—

BREAST CANCER

7. **Mammography is the mainstay**, and research has shown it to be **effective up to age 84**.
8. **There is no agreement on when to stop.** ACS: **annual from 55, or transition to biennial**, continuing if **overall health is good and life expectancy ≥10 years**. NCCN: **annual, with no stopping age**. USPSTF (textbook version): **biennial up to 74**.
9. **Self-examination and clinical breast examination are contested — NCCN recommends both, ACS recommends breast self-awareness if the woman is interested**, and **USPSTF recommends against both**.
10. Few studies describe how women decide to stop; many **discontinue after a serious health event**, recognising that benefit falls with remaining life expectancy.

CERVICAL CANCER

11. **Stop screening at 65** if there have been **three or more consecutive negative Pap tests, or at least two consecutive negative HPV-and-Pap tests within the past 10 years, with the most recent within 5 years**.
12. **Women who had a hysterectomy for benign disease can stop.**
13. **Continue in older women with inadequate or unknown screening history, prior high-grade lesion or cervical cancer, in-utero DES exposure, or immunocompromise.** Essentials warns that **cervical cancer remains a serious risk in older women** and that risk rises with longer inter-test intervals — highly relevant in India, where most older women have **never been screened even once**.

COLORECTAL CANCER

14. **Structural examination of the colon is one of the most effective means of preventing colorectal cancer**, because polyps can be found and removed before malignant change.
15. Start at **50 (USPSTF, NCCN)** or **45 (ACS)**. All three recommend **individualised decisions in adults aged 76–85**;

USPSTF recommends against screening above 85.

16. Options: **FOBT or FIT-DNA every 3 years, flexible sigmoidoscopy every 5 years; colonoscopy is more thorough and is the preferred option**. **Structural tests detect polyps; stool tests detect cancers.**
17. **Limit screening in frail individuals and patients with dementia.** Essentials notes that **most clinicians would not be enthusiastic about subjecting an 85-year-old to colonoscopy**.

PROSTATE CANCER

18. **No major organisation — ACS, AUA, USPSTF, AAFP or ACPM — supports routine PSA testing.** ACS: from **age 50**, informed discussion if **life expectancy ≥10 years**; NCCN: PSA **only if life expectancy ≥10 years**; **USPSTF recommends against screening in men 70 and older**.
19. **The core problem is overdiagnosis** — many prostate cancers grow slowly and would never cause harm, yet surgery or radiation produces **incontinence and impotence** that seriously affect quality of life.

LUNG CANCER AND TESTS NOT RECOMMENDED

20. **Low-dose CT reduces lung cancer mortality by about 20%** compared with chest X-ray in current or former heavy smokers. **ACS: annual LDCT age 55–74, ≥30 pack-years, quit within 15 years, good health, after shared decision-making. NCCN: stop at 77. USPSTF (textbook version): 55–80.**
21. **Stop LDCT** once the person has **not smoked for 15 years**, or **develops a health problem that substantially limits life expectancy or the ability or willingness to undergo curative lung surgery**. **LDCT is never an alternative to smoking cessation.**
22. **Not recommended at any age:** USPSTF gives a **grade D against routine ovarian cancer screening**.

B. Latest Updates (2023–Present)

1. Breast — USPSTF revised in April 2024. The Task Force now recommends **biennial screening mammography for women aged 40 to 74 (B recommendation)**; for **women 75 years or older the evidence remains insufficient (I statement)**, as it does for supplemental ultrasound or MRI in dense breasts (*JAMA/USPSTF, 2024*). The change was estimated to **save 19% more lives** than the previous "start at 50" position. **The key geriatric point is unchanged: above 75, the decision is individualised on life expectancy and comorbidity, not on evidence.**

2. Cervical — the biggest change is self-collection. USPSTF recommends **cytology every 3 years at 21–29 and HPV testing every 5 years at 30–65**, and **for the first time endorses self-collected HPV samples**, which are as accurate as clinician-collected and increase uptake in under-screened groups; it continues to **recommend against screening women over 65 who have had adequate prior screening** (*USPSTF, 2024–25*). The **American Cancer Society updated its guideline in December 2025** — screening from age 25 with **primary HPV testing every 5 years through 65**, with **self-collected vaginal specimens acceptable** and new guidance on when to safely exit screening. HRSA's Women's Preventive Services Guidelines were updated in January 2026 to prefer **primary hrHPV testing every 5 years for ages 30–65**.

3. Colorectal — start age lowered. USPSTF (2021, still current) recommends screening from **45 to 75 years**, with **selective screening at 76–85 (grade C)**. The elderly-specific decision point is unchanged, but the entry age has moved from 50 to 45.

4. Lung — eligibility broadened. USPSTF (2021) now recommends **annual LDCT from age 50 to 80** in adults with a **≥20 pack-year history** who currently smoke or quit within 15 years — a lower age and lower pack-year threshold than the textbook figures.

5. Prostate — unchanged but clarified. USPSTF (2018): **shared decision-making at 55–69 (grade C)** and **against PSA screening at 70 and above (grade D)**.

6. India — a population-based programme now exists, but it stops at 65. NPCDCS was **renamed the National Programme for Prevention and Control of Non-Communicable Diseases (NP-NCD) in May 2023**. Under this programme, **oral, breast and cervical cancer screening is free at Health and Wellness Centres for adults aged 30 to 65**, with community health workers identifying eligible individuals; **ICMR recommends clinical breast examination every 1–3 years** as the practical starting point in India. The **Operational Framework for Management of Common Cancers** notes that **breast, cervix and oral cancers together account for about 34% of all cancers in India**, and recommends **oral cancer screening for all adults over 30**.

7. Indian coverage data. As of July 2025, **10.18 crore of an eligible 25.42 crore women aged ≥30** had been screened for cervical cancer through **Ayushman Arogya Mandirs**, primarily with **visual inspection with acetic acid (VIA)** at sub-centres and PHCs, with VIA-positive cases referred upward (*Ministry of Health, 2025*). However, a 2025 review found that **fewer than 2% of eligible women aged 30–49 had actually been screened** for cervical or breast cancer at the time of the 2019–21 national survey (*Asian Pacific Journal of Cancer Prevention, 2025*).

8. Resource-stratified Indian guidance. The **National Cancer Grid** consensus guidelines recommend **VIA for**

women 30–65, one to three times in a lifetime, in low-resource settings; HPV testing every 10 years in enhanced-resource settings; and HPV testing every 5 years for ages 25–65 in high-resource settings.

9. Decision aids are now standard practice. Current geriatric oncology practice emphasises **life-expectancy estimation (e.g. ePrognosis calculators)**, **"lag time to benefit" framing**, and **explicit de-implementation of screening** when life expectancy falls below the lag time — with counselling framed around "*your other health conditions matter more than this test now*" rather than "*you are too old*".

C. Current Practice Update

Cancer	Textbook Position	Current Practice (2024–2026)
Breast	USPSTF biennial to **74**; ACS annual from 55; NCCN annual, no stop age	USPSTF 2024: **biennial 40–74 (B)**; **≥75 insufficient evidence (I)** — individualised on life expectancy
Cervical	Pap ± HPV to 65; stop if adequate prior screening	**Primary HPV every 5 years, 30–65**; **self-collected samples now endorsed**; still stop at 65 if adequately screened
Colorectal	Start **50** (USPSTF/NCCN) or 45 (ACS)	Start **45** for all; **45–75 recommended**, **76–85 selective**, **>85 not recommended**
Lung	LDCT **55–74/77/80**, **≥30 pack-years**	LDCT **50–80**, **≥20 pack-years**, quit within 15 years
Prostate	ACS/NCCN if life expectancy ≥10 years; USPSTF against ≥70	Unchanged — **SDM 55–69 (C)**, **against ≥70 (D)**
Framework	Screening by **functional category**	Same principle, operationalised through **life-expectancy calculators and lag-time-to-benefit**, with formal **de-implementation** counselling
India	Not addressed	**NP-NCD (renamed 2023)** offers free **oral, breast and cervical screening at 30–65** via **Ayushman Arogya Mandirs** using **VIA**; **no national programme covers those above 65**

Examiner's Tip

The opening sentence should establish the geriatric principle, not the cancer list: "*In the elderly, the decision to screen is governed not by chronological age but by three questions — is life expectancy longer than the lag time to benefit, will the patient accept work-up and treatment, and do benefits outweigh harms?*"
Then present **Pathy's four-category table** — it is the single highest-yield item in this topic.
Close on the Indian gap: "*India's NP-NCD programme screens for oral, breast and cervical cancer, but only up to age 65 — so no organised cancer screening exists for the Indian geriatric population at all.*"

Extra Points to Remember

The one-line summary of the whole topic: screening tests are chosen by *life expectancy*, stopped by *competing comorbidity*, and abandoned altogether when the patient would not accept the consequences of a positive result.
Two numbers that anchor everything: lag time to benefit ≈ **10 years** for breast and colorectal cancer; mean age of cancer detection ≈ **68**, mean age of cancer death ≈ **72**.
Never forget the distinction between screening and diagnosis — red-flag symptoms (bleeding, anaemia, weight loss, change in bowel habit) mandate investigation at *any* age, irrespective of every screening cut-off in this answer.

Question 5. Screening for colorectal cancer.

Primary source: Pathy's, 6th ed.; Essentials of Clinical Geriatrics, 7th ed.

A. High-Yield Points (Textbook Content)

RATIONALE

1. **The prevalence of carcinogenesis increases along the entire gastrointestinal tract with age**, driven by **mucosal irritation, inflammation, impaired antioxidant mechanisms and genetically damaged epithelium**.
2. GI cancers may cause **constipation, early satiety and bleeding in the early stages** — and **if caught and treated early they may be cured**.
3. **Colorectal cancer screening is unique because it prevents cancer, not merely detects it.** Pathy's states that **regular structural examination of the colon is one of the most effective means of preventing colorectal cancer**, because polyps can be found and removed before they turn malignant.

THE TESTS

4. **Faecal occult blood testing is rapid and easy, with a sensitivity of 82%.** If positive, **further diagnostic testing is recommended regardless of age**.
5. **Structural tests versus faecal tests — a key examination distinction:** structural examinations are **more likely to detect polyps, which can be removed, thereby preventing malignancy**; faecal tests are **more likely to detect malignancies early**, so that treatment carries less morbidity.
6. **Colonoscopy is the more thorough examination and is the preferred option for screening.** ACS guidance quoted in Pathy's is **endoscopic screening of the colon every decade over the age of 50**.
7. **Choice of test depends on extrinsic factors** — transportation, availability of a gastroenterologist, the patient's willingness to undergo one procedure over another, and the patient's health status.
8. **Screening intervals differ by test type, which is a recognised source of confusion for both patients and clinicians** — clinicians must explain the strengths and limitations of each.
9. Textbook-supported options: **FOBT or FIT-DNA every three years, or flexible sigmoidoscopy every five years**, with colonoscopy preferred.

AGE THRESHOLDS

10. **USPSTF and NCCN recommend starting at 50; ACS at 45** (textbook position).
11. **All three recommend that screening in adults aged 76–85 be an individual decision**, based on **overall health and prior screening history**.
12. **USPSTF recommends against screening above 85 (grade D).** In the Essentials table the grades are: **A for 50–75, C for 76–85, D for >85**, and **I (insufficient evidence) for CT colonography and faecal DNA testing**.
13. Pathy's also notes that **most guidelines add that screening can be stopped after the age of 80**.
14. Medicare frequencies quoted in Essentials: **FOBT every 12 months; flexible sigmoidoscopy every 48 months (or 120 months after a colonoscopy); colonoscopy every 120 months (every 24 months if high risk), or 48 months after a sigmoidoscopy; barium enema every 24–48 months**.

GERIATRIC-SPECIFIC CONSIDERATIONS

15. **Pathy's screens by functional category:** colonoscopy — **"consider every 10 years" in the Robust only**; FOBT — **yearly in the Robust, "consider yearly" in the Frail and the moderately demented**; **nothing at End of life**.
16. **Primary care physicians may choose to limit screening in Frail individuals or in patients with dementia.**
17. **Colonoscopy in patients over 80 carries a greater risk of complications, adverse events and higher morbidity** than in younger patients. It is also associated with **a higher chance of poor bowel preparation, resulting in inadequate imaging and lower completion rates**. It should be performed only **after careful consideration of individual risks and benefits**.
18. Essentials adds bluntly that **the issue of risk versus benefit becomes even more relevant when procedures like colonoscopy are considered**, and that **most clinicians would not be enthusiastic about subjecting an 85-year-old to colonoscopy**.
19. **The "10-year rule" applies:** the lag time to mortality benefit is roughly a decade, so screening is justified only when **life expectancy is 10 years or more**.
20. **Screening versus diagnosis — never confuse the two.** Pathy's notes that in a constipated older patient, **structural examination of the colon is dictated by the need for routine screening AND by red-flag signs — rectal bleeding, anaemia, unintentional weight loss, or a recent change in bowel habit. Red flags mandate diagnostic colonoscopy at any age, irrespective of screening cut-offs.**

B. Latest Updates (2023–Present)

1. Starting age has been lowered to 45. USPSTF (2021, still current) recommends screening from **45 to 75 years** (grade B for 45–49, grade A for 50–75), with **selective screening at 76–85 (grade C)** based on overall health, prior screening history and patient preference. The textbook's "start at 50" is now outdated; the geriatric decision point at 76–85 is unchanged.

2. Blood-based screening has arrived. On **29 July 2024** the FDA approved **Guardant Health's Shield blood test** for colorectal cancer screening in **average-risk adults aged 45 and older** — the **first blood test approved as a primary screening option**, meaning it can be offered like other non-invasive methods, and the first to meet Medicare coverage requirements. Approval was based on **ECLIPSE**, involving more than 20,000 patients, published in the *New England Journal of Medicine* in March 2024, showing **83% sensitivity for colorectal cancer and 90% specificity for advanced neoplasia**.

3. But the blood test has a critical weakness for geriatric practice. Shield's sensitivity for **advanced adenomas was only 13.2%**, and patients with advanced adenomas are precisely those at high risk of developing colorectal cancer. The NCI notes that neither FIT nor Cologuard is as accurate as colonoscopy, and that Shield detected only 13% of advanced precancerous polyps found at colonoscopy. **In other words, a blood test detects cancer but does not prevent it — it converts CRC screening from a preventive to a purely early-detection strategy.** For reference, Cologuard was approved in 2014 for ages ≥50 and in 2019 for 45–49, and Epi proColon in 2016.

4. NordICC — the only RCT of screening colonoscopy — has now reported long-term results, and they are sobering. In the 10-year report (*NEJM, 2022*), the risk of colorectal cancer was **0.98% in the colonoscopy group versus 1.20% in the no-screening group, an 18% risk reduction**, but **colorectal cancer mortality was not significantly reduced**. Uptake was only **42%**; in adjusted per-protocol analysis, incidence and mortality reductions rose to **31% and 50%** respectively (*Gastroenterology, 2023*).

5. The 13-year NordICC update (*Lancet*, May 2026 / DDW 2026) confirms the pattern. One colonoscopy significantly reduced colorectal cancer incidence but not mortality over 13 years, and colorectal cancer mortality was **lower in both study groups than had been assumed when the trial was designed**. The authors state that **the risk of colorectal cancer death is very low even without screening, and the hope of finding a screening benefit on colorectal cancer mortality with longer follow-up is so far not substantiated**. A quality caveat: **as many as 30% of endoscopists in NordICC fell short of the adenoma detection rate of 25%** that is the accepted quality indicator.

6. How this fits with the older trials. A standardised comparison found that **at 10 years all randomised trials showed significant reductions in colorectal cancer incidence**, but **two flexible sigmoidoscopy trials and NordICC showed no significant mortality reduction**, whereas PLCO saw fewer colorectal cancer deaths from year 2 (*JNCI, 2025*). Others argue the negative NordICC mortality result mainly reflects **insufficient follow-up time**, since in NORCCAP mortality differences only emerged after 9.5 years and were about four-fold greater at 12 years than at 10.

> **Practical implication for the elderly**
> The mortality benefit of colonoscopy is **later and smaller than previously believed**, which **strengthens rather than weakens the geriatric argument** — if the lag time to benefit may exceed 10–13 years, then screening an 80-year-old with competing comorbidity is even harder to justify, while the procedural harms (poor preparation, sedation risk, perforation) occur immediately.

7. Indian position. There is **no national colorectal cancer screening programme in India**. The NP-NCD programme delivered through Ayushman Arogya Mandirs covers only **oral, breast and cervical cancers, for ages 30–65**. CRC screening in India is therefore **opportunistic, private and largely confined to symptomatic or high-risk individuals** — making **red-flag-driven diagnostic colonoscopy**, rather than screening, the realistic pathway for most older Indian patients.

C. Current Practice Update

Aspect	Textbook Position	Current Practice (2021–2026)
Starting age	**50** (USPSTF, NCCN); 45 (ACS)	**45 for everyone** (USPSTF 2021, ACS)
Upper age	76–85 individualised; against >85; "stop after 80"	**Unchanged** — 45–75 recommended, 76–85 selective (C), >85 against (D)
Blood-based test	Not available; faecal DNA graded "I"	**Shield approved July 2024** as a primary screening option for ≥45; but **advanced adenoma sensitivity only 13.2%**
Stool tests	FOBT (sensitivity 82%); FIT-DNA every 3 years	**FIT annually** is now standard; **FIT-DNA (Cologuard) every 3 years**
Colonoscopy evidence	Inferred from cohort studies; "most effective	**NordICC (NEJM 2022, Lancet 2026): incidence**

	means of preventing CRC"	**reduced, mortality not** at 10 and 13 years; per-protocol 31% and 50%
Key uncertainty	Risk–benefit in the very old	**Lag time to mortality benefit may exceed 13 years** — raises the bar for screening the frail or very elderly
India	Not addressed	**No national CRC screening programme**; NP-NCD covers only oral, breast and cervical cancers at 30–65

Examiner's Tip

The distinguishing line for a high mark: "*Colorectal cancer screening is unique among cancer screening tests because a structural examination is genuinely preventive — polypectomy removes the precursor lesion — whereas stool and blood tests only detect established cancer earlier.*"
Then deliver the counterweight: "*However, NordICC, the only randomised trial of screening colonoscopy, showed reduced incidence but no significant mortality reduction at both 10 and 13 years — which means the lag time to benefit may exceed a decade, and that argues strongly against screening the frail or very elderly.*"
Always end with the red-flag caveat — examiners look for it: **bleeding, anaemia, weight loss or change in bowel habit demand diagnostic colonoscopy at any age.**

Extra Points to Remember

Structural vs faecal: structural = detects polyps = *prevents* cancer. Faecal/blood = detects cancer = *early detection only*.
NordICC numbers: 10-year incidence 0.98% vs 1.20% (18% reduction), **no mortality benefit**; uptake only 42%; per-protocol 31% incidence and 50% mortality reduction.
Shield blood test: 83% sensitivity for cancer but only **13.2% for advanced adenoma** — the number that explains why it cannot replace colonoscopy.
Colonoscopy in the over-80s: more complications, more sedation risk, and **poorer bowel preparation with lower completion rates**.

Question 6. Mammography — screening the older woman.

Primary source: Pathy's, 6th ed. — "Preventive Geriatrics"; Essentials of Clinical Geriatrics, 7th ed. — "Prevention".

A. High-Yield Points (Textbook Content)

RATIONALE

1. **Mammography is the mainstay of breast cancer screening.**
2. **Research has shown mammography to be effective up to age 84** — a key figure to quote.
3. Breast cancer is common and does shorten life in older women: Essentials records the **median age at diagnosis as about 61 and the median age at death as about 69**.
4. However, **cancer in older people may grow more slowly** even though incidence and prevalence rise with age — this is the basis of **overdiagnosis**.

GUIDELINE DISAGREEMENT (HIGH-YIELD)

5. **There is a lack of agreement among organisations about both the screening interval and when to stop.**
6. **ACS: annual mammography for women 55 and older**, or they may **transition to biennial**. Women should **continue if overall health is good and life expectancy is 10 years or more**.
7. **NCCN: annual mammography, with no guidelines for stopping.**
8. **USPSTF (textbook version): biennial screening mammography up to age 74**; evidence **insufficient for women 75 or older (I statement)**.
9. **Medicare (Essentials):** covers a **screening mammogram every 12 months for all women aged 40 and above**, plus **one baseline mammogram between 35 and 39**, and covers digital technologies.

CLINICAL BREAST EXAMINATION AND SELF-EXAMINATION

10. **There is no recommendation for or against breast self-examination, breast self-awareness, or clinician-performed clinical breast examination.**
11. **NCCN recommends both; ACS recommends breast self-awareness if the woman is interested; USPSTF recommends against both** (grade **D for teaching BSE**, grade **I for CBE**).
12. **Breast self-awareness may allow detection of lesions not seen on mammography, or interval cancers developing between mammograms** — the argument in its favour.

PATHY'S FUNCTIONAL-CATEGORY APPROACH

13. **Mammography by category:** every **1–2 years up to age 80** in the **Robust**; **consider** every 1–2 years **up to 75** in the **Frail**; **consider** every 1–2 years **up to 70** in the **Moderately demented**; **nothing at End of life**.
14. **Breast examination: yearly** in the Robust, Frail and Moderately demented; not at End of life.

STOPPING SCREENING

15. **Few studies describe how women decide to stop mammography.**
16. Some women and their providers, **recognising that the expected benefit of early detection declines with remaining life expectancy, consciously decide to discontinue screening following a serious health event** — a practically useful trigger point.
17. **The "10-year rule" applies:** the lag time to mortality benefit is about a decade, so continue only if **life expectancy ≥10 years**.

CONTROVERSY AND RELATED RECOMMENDATIONS

18. Essentials states that **analysis of mammography trial data illustrates just how confusing this literature can be**; one analysis suggests **screening after age 69 gives a small gain in life expectancy and is modestly cost-effective** (Kerlikowske, 1999).
19. **Harms** to weigh: **false positives, unnecessary biopsy, pain, anxiety, and overdiagnosis with overtreatment** — the last being especially serious in older women who tolerate treatment poorly.
20. Related USPSTF positions in Essentials: **BRCA testing — grade D against routine referral if family history is not suggestive, grade B for referral if it is**; **chemoprevention — grade D against routine tamoxifen or raloxifene at average risk, grade B for discussion in high-risk women at low risk of adverse effects**.
21. **Practical geriatric rule:** screening should stop when a woman **would not accept or could not tolerate a biopsy or subsequent cancer treatment** — if the result cannot change management, the test should not be done.

B. Latest Updates (2023–Present)

1. USPSTF revised its recommendation in April 2024. The Task Force now recommends **biennial screening mammography for women aged 40 to 74 (B recommendation)**, concludes the evidence is **insufficient for women 75 years or older (I statement)**, and also insufficient for **supplemental ultrasound or MRI in women with dense breasts** (*JAMA/USPSTF, 2024*). The change from the 2016 version is that **biennial screening now begins at 40 rather than being individualised for 40–49**; the change was estimated to **save 19% more lives** than starting at 50. Both **digital mammography and digital breast tomosynthesis ("3D mammography") are accepted as effective mammographic modalities**.

2. The 75-and-over question remains unresolved — but the observational data are more favourable than "insufficient evidence" suggests. No randomised trial of mammography included women over 74, so it is not known whether mammography helps them live longer; data suggest around **10 years of life expectancy** is needed for a mortality benefit, while the harms — pain, anxiety, complications of biopsy after a false positive, and overdiagnosis — are immediate. On the other hand, **mammography performs significantly better in older women**, with **higher sensitivity, specificity, cancer detection rate and positive predictive value, and lower recall and false-positive rates**; modelling suggests the **overdiagnosis rate is low and benefits outweigh risks until about age 90**. A systematic review of 36 studies in women 75+ found **mixed evidence**, with some studies showing a mortality benefit and others none, and called for better comparison groups and formal estimation of overdiagnosis given the substantial competing risk of death in this age group (*British Journal of Cancer, 2023*).

3. Counterpoint on overdiagnosis in the elderly. A meta-analysis of two RCTs including **33,268 women aged 70 or older** found a **28% relative reduction in breast cancer mortality (RR 0.72)**, but estimated the **proportion of overdiagnosis in older women at 19%**, higher than in younger women. A 2025 review in *Nature Reviews Clinical Oncology* argues the case for **active monitoring of selected screen-detected breast cancers in women ≥75**, drawing on the **COMET trial of active monitoring for low-risk DCIS (*JAMA*, 2025)** — a shift from "detect and treat" to "detect and observe" in the very old.

4. Artificial intelligence is the biggest technical change — MASAI is the landmark trial. MASAI is the **first randomised controlled trial of AI in breast cancer screening and the largest trial of AI in cancer screening generally**, involving **105,934 Swedish women**. Interim safety results (*Lancet Oncology*, 2023) showed a **44% reduction in screen-reading workload**; a *Lancet Digital Health* analysis showed a **29% increase in cancer detection without any increase in false positives**; and the full results (*Lancet*, 2026) showed **12% fewer interval cancers**, meaning fewer aggressive and advanced cancers diagnosed between screening rounds. **Sensitivity was 6.7 percentage points higher (80.5% vs 73.8%) at the same specificity (98.5%), with consistent results across age and breast-density subgroups.** Importantly, the authors state the findings **do not support replacing radiologists** — AI-supported screening still requires at least one human reader.

5. Breast density notification is now mandatory in the US. The FDA's updated MQSA rule (effective **September 2024**) requires that every mammogram report include a **breast density notification** to the patient. USPSTF, however, still finds the evidence **insufficient** to recommend supplemental ultrasound or MRI on the basis of density alone.

6. India — mammography is not the national strategy. ICMR recommends clinical breast examination every one to three years as the practical starting point in India, along with monthly self-examination; free **breast, oral and cervical cancer screening is offered at Health and Wellness Centres for adults aged 30 to 65**. Uptake remains very poor — **fewer than 2% of eligible women aged 30–49 had been screened** for cervical or breast cancer at the 2019–21 national survey. **For Indian geriatric practice this means: CBE, not mammography, is the realistic screening tool, and no programme covers women above 65 at all.**

C. Current Practice Update

Aspect	Textbook Position	Current Practice (2024–2026)
Starting age	USPSTF biennial from **50**; individualise 40–49	**Biennial from 40 to 74 (grade B)** — USPSTF 2024
Stopping age	ACS: continue if healthy, life expectancy ≥10 years; NCCN: no stop age; Pathy's: up to 80 / 75 / 70 by category	**Unchanged in principle**; USPSTF still **"insufficient evidence ≥75"** — individualised on life expectancy and comorbidity
Evidence in ≥75	"Effective up to 84"	**No RCT ever included women >74**; observational data favourable (better sensitivity/specificity, low recall), but **overdiagnosis ~19%** in the old
Modality	Film vs digital vs MRI — "insufficient evidence"	**Digital mammography and DBT both accepted**; supplemental US/MRI for dense breasts still **insufficient evidence**
Breast density	Not addressed	**Mandatory density notification** in mammogram reports (FDA, 2024)
Artificial intelligence	Not addressed	**MASAI RCT**: +29% detection, −44% reading workload, −12% interval cancers, +6.7% sensitivity at same

		specificity; **AI supports, does not replace, the radiologist**
Screen-detected disease in the very old	Detect and treat	Growing case for **active monitoring** of low-risk screen-detected lesions (COMET trial, 2025)
India	Not addressed	**CBE every 1–3 years (ICMR)**; free breast screening at 30–65 via Ayushman Arogya Mandirs; **no coverage above 65**; uptake <2%

Examiner's Tip

The paradox to state explicitly, because it earns marks: "*Mammography actually performs better in older women — higher sensitivity, higher specificity, higher positive predictive value and fewer recalls — yet the evidence for screening them is graded insufficient, because no randomised trial ever enrolled a woman over 74 and because competing mortality means much of what is detected is overdiagnosis*."
Give **Pathy's category cut-offs (80 / 75 / 70)** as the practical answer, then the current USPSTF position (**biennial 40–74; ≥75 insufficient evidence**).
Close on India: **CBE every 1–3 years per ICMR is the realistic tool, and no national programme screens women above 65.**

Question 7. Prostate-specific antigen — to screen or not to screen?

Primary source: Pathy's, 6th ed.; Essentials of Clinical Geriatrics, 7th ed.

A. High-Yield Points (Textbook Content)

THE CORE CONTROVERSY

1. **The biggest controversy in prostate screening is that some prostate cancers grow slowly and would never cause any problems.**
2. Because of an elevated PSA, **men may be diagnosed and treated with surgery or radiation for lesions that would never have caused symptoms or led to their death** — and **these treatments have side effects that can seriously affect a man's quality of life**.
3. Essentials frames it more broadly: **there is great controversy around the efficacy of active screening for prostate and breast cancer in older persons**, because although incidence rises with age, **the rate of tumour growth may be slower**.
4. **This is therefore a disease where screening can cause net harm** — the classic example of **overdiagnosis leading to overtreatment**.

WHAT THE GUIDELINES SAY (TEXTBOOK POSITIONS)

5. **No major scientific or medical organisation supports routine testing for prostate cancer** — including the **ACS, American Urological Association (AUA), USPSTF, American Academy of Family Physicians, and American College of Preventive Medicine**.
6. **ACS:** beginning at **age 50**, men at average risk with a **life expectancy of at least 10 years** should have **a conversation with their provider** about the benefits and limitations of PSA testing and make an **informed decision based on personal values and preferences**.
7. **NCCN:** focuses on men at **increased risk because of family history and race**, and states that **PSA testing should only be offered to men with a life expectancy of 10 or more years**.
8. **USPSTF (as quoted in Pathy's): recommends against prostate cancer screening in men 70 and older.**
9. **Older USPSTF grades (Essentials table): "I" (insufficient evidence) for men younger than 75** and **"D" (recommends against) for men aged 75 or older**.
10. **Note the policy discrepancy highlighted in Essentials: Medicare will pay for PSA screening even though the USPSTF does not recommend it.**
11. Essentials records that **the USPSTF recommendation not to screen men older than 75 "unleashed a firestorm of controversy"** — worth quoting in a viva.
12. All organisations **emphasise the need for the clinician to discuss possible benefits and side effects of treatment**, so that **men aged at least 50 (younger if at higher risk) can make informed decisions**.

PATHY'S FUNCTIONAL-CATEGORY APPROACH

13. **PSA by category: "Discuss pros and cons with the patient"** in the **Robust** and the **Frail**; **"Discuss pros and cons with the caregiver"** in the **Moderately demented**; **nothing at End of life**.
14. **Examination point:** PSA is the **only cancer screening test Pathy's carries into the demented category** — and even then the recommended action is **a conversation, not a test**. The intervention here is shared decision-making itself.

PRACTICAL POINTS IN THE OLDER MAN

15. **The "10-year rule" is central** — both ACS and NCCN make life expectancy of ≥10 years the entry criterion, because prostate cancer has a long natural history and the lag time to mortality benefit is long.
16. **Benign prostatic hyperplasia is a major confounder** — Pathy's notes prostatic hyperplasia is present in **one in three men over the age of 70**, and it raises PSA independently of cancer.
17. **Other non-malignant causes of a raised PSA:** prostatitis, urinary tract infection, urinary retention, catheterisation, recent instrumentation, digital rectal examination, ejaculation and cycling. **5-alpha-reductase inhibitors halve the PSA**, so the measured value must be doubled.
18. **The harm cascade must be explained explicitly:** PSA → biopsy (bleeding, pain, **sepsis**) → overdiagnosis → radical treatment.
19. **Treatment harms are geriatrically important:** Pathy's states that **prostate cancer treatment, including surgery, radiotherapy and brachytherapy, can cause urinary incontinence and erectile dysfunction**, and that **prostate cancer survivors report high levels of concern about sexual health**, particularly about satisfying their partners.

20. **The decisive geriatric question is not "how old is he?" but "if the PSA is raised, will he accept a biopsy, and if cancer is found, would he accept treatment?"** If the answer is no, the test should not be done.

B. Latest Updates (2023–Present)

1. ERSPC — the definitive trial — now has 23-year follow-up. The final ERSPC analysis (*NEJM*, 2025) covered a predefined core age group of **162,236 men aged 55–69** across eight European countries, randomised to repeated PSA testing or no invitation, with **prostate cancer mortality as the primary outcome**. Long-term follow-up **confirms a sustained reduction in prostate cancer deaths with an improved harm–benefit ratio**, and the authors conclude that **future screening should adopt risk-based approaches to minimise overdiagnosis while maintaining benefit**. At 23 years the **relative reduction in prostate cancer–specific mortality was 13% (RR 0.87, 95% CI 0.80–0.95), an absolute risk reduction of 0.22%**.

2. The Rotterdam arm gives the most quotable numbers. At a median follow-up of **21 years**, the rate ratio for prostate cancer–specific mortality was **0.73 (95% CI 0.61–0.88)** favouring screening, with a **number needed to invite of 246** and a **number needed to diagnose of 14** to prevent one prostate cancer death (*European Urology, 2023*).

3. The updated systematic review, including the UK CAP trial, gives a more sober estimate. The latest review added the **UK CAP trial (2002–2021, 15 years of follow-up)** and up to 23 years of ERSPC data; in sensitivity analyses based mainly on ERSPC, **PSA screening was associated with a 13% relative reduction in prostate cancer mortality (RR 0.87, moderate-certainty evidence)**, with ERSPC judged to have the lowest risk of bias. Repeated PSA screening reduces **prostate cancer–specific mortality and metastatic disease but has no effect on all-cause mortality**, and the benefit **increases with longer follow-up**.

> **The single most important new number for a geriatrics answer**
>
> **Overdiagnosis rises steeply with age.** Using 15-year follow-up from the CAP randomised trial and English competing-mortality rates, men diagnosed at age **50 had a 16% chance** that the cancer would not have been detected within 15 years; this rose to **32% at age 70 and 58% at age 80**. **In other words, more than half of screen-detected prostate cancers in an 80-year-old are overdiagnosis.**

4. Treatment choice matters less than was assumed — the ProtecT 15-year results. In ProtecT (*NEJM*, 2023), men with localised, PSA-detected prostate cancer randomised to **active monitoring, radical prostatectomy or radiotherapy had essentially identical prostate cancer mortality at 15 years (around 3% in all three arms)**, though metastasis and progression were commoner with monitoring. **This substantially weakens the argument that early detection must be followed by radical treatment**, and strengthens the case for active surveillance in older men.

5. The screening pathway is being redesigned around MRI, not PSA alone. The **ProScreen trial** (*JAMA*, 2024) uses a stepwise pathway — **PSA ≥3 ng/mL → four-kallikrein panel → mpMRI if score ≥7.5% → biopsy if PI-RADS ≥3**, with screening intervals of 2, 4 or 6 years depending on the PSA level; mortality analyses are planned at 10 and 15 years. The randomised **PROSA trial** (*European Urology*, 2025) found an **MRI-first pathway detected clinically significant cancer in 4.6% versus 1.8% with a PSA-based strategy** — a 2.5-fold increase — **without increasing detection of low-grade (Grade Group 1) disease**. A contrast-free MRI-first strategy therefore **improves detection of clinically significant prostate cancer without increasing overdiagnosis or biopsy rates**. This builds on **PRECISION (2018)**, in which **28% of men randomised to MRI-targeted biopsy avoided biopsy altogether while 12% more clinically significant cancers were detected** than with standard TRUS biopsy.

6. A definitive answer is being sought. The **TRANSFORM trial**, announced May 2024 and backed by a **£42 million grant**, is one of the largest screening studies ever, involving **over a quarter of a million men followed for more than 10 years**, aiming to increase detection of life-threatening cancer while reducing unnecessary biopsies and overtreatment. It will invite men aged **50–75, or 45–75 for Black men**, and test four approaches including **12-minute fast MRI, genetic testing and PSA**, with early results **not expected before 2028**.

7. Where policy currently stands. Most countries recommend against population-based prostate screening despite the ERSPC results, because harms may outweigh benefits; the **UK National Screening Committee voted against PSA-alone screening in 2020**. The **USPSTF position is unchanged since 2018**: **shared decision-making for men 55–69 (grade C)** and **against screening in men 70 and older (grade D)**. **India has no national prostate screening programme**; testing is entirely opportunistic and privately funded.

C. Current Practice Update

Aspect	Textbook Position	Current Evidence (2023–2026)
Does PSA screening save lives?	"No major organisation supports routine testing"	**Yes, modestly** — ERSPC at 23 years: **13% relative, 0.22% absolute** reduction in prostate cancer mortality; Rotterdam at 21 years RR 0.73, **NNI 246, NND 14**. **No all-cause mortality benefit.**

Age cut-off	USPSTF against ≥70 (Pathy's) or ≥75 (Essentials)	**Unchanged** — USPSTF 2018: SDM 55–69 (C), against ≥70 (D)
Overdiagnosis	Stated qualitatively	**Quantified by age**: ~16% at 50, **32% at 70, 58% at 80** (CAP trial analysis)
Consequence of detection	Surgery or radiation with serious side effects	**ProtecT at 15 years: ~3% prostate cancer mortality whether monitored, operated or irradiated** — active surveillance is a legitimate first choice
The test itself	PSA alone, then systematic biopsy	**Risk-stratified pathways** — PSA → kallikrein panel → **MRI** → targeted biopsy (ProScreen); **MRI-first** doubles detection of significant cancer without more overdiagnosis (PROSA 2025)
Policy direction	Against routine screening	Moving from "screen or not" to **"how to screen better"** — organised, risk-adapted, MRI-based programmes; **TRANSFORM results due 2028+**
Practical rule in geriatrics	Life expectancy ≥10 years; discuss pros and cons	**Same, but sharper**: ask whether the man would accept biopsy and treatment; above 75–80 the majority of detected cancers are overdiagnosis

Examiner's Tip

Do not answer "yes" or "no" — answer with the **shape of the evidence**: "*PSA screening does reduce prostate cancer mortality — 13% relatively but only 0.22% absolutely at 23 years in ERSPC, with 246 men needing to be invited and 14 needing to be diagnosed to prevent one death — but it produces no all-cause mortality benefit, and the proportion of detected cancers that represent overdiagnosis rises from 16% at age 50 to 58% at age 80.*"
Then deliver the geriatric conclusion: "*That is why the USPSTF supports shared decision-making at 55–69 and recommends against screening at 70 and above — and why in a man over 75 the correct question is not his age but whether he would accept a biopsy and its consequences.*"

Extra Points to Remember

Four trials, four messages: ERSPC — screening works modestly; PLCO — contaminated, invalid; CAP — quantifies overdiagnosis by age; ProtecT — treatment choice barely changes mortality at 15 years.
The direction of travel is away from "PSA yes/no" and toward **risk-stratified, MRI-first, organised pathways** (ProScreen, PROSA, TRANSFORM).
Never forget the confounders of a raised PSA in an older man: BPH (one in three men over 70), prostatitis, UTI, retention, catheterisation, DRE, ejaculation — and remember to **double the value in men on 5-alpha-reductase inhibitors**.

Appendix. Rapid Revision Grid

Key numbers, trials, cut-offs and Indian data compiled from all seven answers — for last-night revision.

1. Immunization — Numbers to Quote

Item	Figure
Influenza deaths	>90% occur in adults ≥65
Influenza vaccine	↓ influenza risk ~50%; ↓ influenza mortality ~70%; only ~40% effective vs clinical illness in nursing homes
Herd protection	Vaccinating >80% of nursing-home residents prevents outbreaks
Nichol 2007 (Essentials)	HR 0.75 for pneumonia/influenza hospitalisation; HR 0.52 for death
CAPITA	PCV13 ↓ vaccine-type pneumococcal pneumonia by 45%
Zoster vaccines	Zostavax 30% protective; Shingrix ~90% protective
Post-herpetic neuralgia	>40% of those over 60; zoster in up to 50% by age 85
Tetanus	>50% of cases occur in people ≥60
AReSVi-006 (RSV)	82.6% season 1; 94.6% with comorbidity; 62.9% cumulative over 3 seasons; 48% in season 3
DANFLU-2	332,438 adults ≥65; primary endpoint **neutral**; cardiorespiratory hospitalisation 2.25% vs 2.38%
Zoster & dementia (Wales)	~20% reduction in new dementia diagnoses over 7 years; uptake jump 0.01% → 47.2%
Zoster & cardiovascular	23% lower risk of cardiovascular events
PCV21	Covers ~85% of IPD in ≥50s vs ~59% for PCV20; **lacks serotype 4**
PCV20 real-world VE	IPD 25.6% overall (35.4% at 65–74; 24.0% at 75–84; 16.6% at ≥85); all-cause pneumonia 15.2%

2. Immunization — Age Thresholds (Current)

Vaccine	Current recommendation
Influenza	Annual, all ages ≥50 (API 2026); HD-IIV3 / RIV3 / aIIV3 preferred at ≥65
Pneumococcal	Single dose PCV20 or PCV21 from age **50** (ACIP, Oct 2024)
Herpes zoster (RZV)	2 doses, all adults ≥50; immunocompromised ≥18
RSV	Single dose — universal ≥75; risk-based 60–74; extended risk-based 50–59 (Apr 2025)
Td / Tdap	Booster every 10 years; ≥1 lifetime Tdap
COVID-19	Shared clinical decision-making (2025–26)

3. Travel Vaccination — Key Facts

Item	Figure / rule
Pre-travel consult	4–6 weeks before departure
YF certificate	Valid 10 days after vaccination; now **valid for life**; India — 6 days' quarantine if absent
YF and age ≥60	**Precaution, not contraindication**
YEL-AND	0.8/100,000 doses overall; **2.2/100,000 at ≥60**
YEL-AVD	Multi-organ failure, ~48% mortality; 6× higher in the elderly
Serious AEs in elderly	3× higher overall; 5× higher above 70
Hajj / Umrah	Meningococcal ACWY required, ≥10 days before arrival, valid 3–5 years
Rabies PrEP	2 doses — day 0 and day 7
Japanese encephalitis	Stay ≥1 month in rural endemic area; age itself is a risk factor for severe JE
Hepatitis A in India	Check anti-HAV IgG first — most older Indians already immune

4. Cancer Screening — Age Cut-offs

Cancer	Pathy's / textbook	Current (USPSTF)
Breast	Mammography 1–2 yearly: Robust to 80, Frail to 75, Demented to 70; "effective to 84"	Biennial **40–74 (B)**; **≥75 insufficient (I)**

Cervical	Stop at 65 if adequate prior screening	Primary HPV q5y, **30–65**; self-collection endorsed; stop at 65
Colorectal	Start 50 (USPSTF/NCCN) or 45 (ACS); 76–85 individual; >85 against	**45–75 (A/B)**; **76–85 selective (C)**; **>85 (D)**
Lung	LDCT 55–74/77/80; ≥30 pack-years	LDCT **50–80**; **≥20 pack-years**; quit within 15 years
Prostate	ACS/NCCN if life expectancy ≥10 y; USPSTF against ≥70	SDM **55–69 (C)**; **against ≥70 (D)**
Ovarian	Against routine screening (D)	Unchanged (D)

5. Cancer Screening — Trials and Numbers

Trial / source	Result
NordICC (NEJM 2022)	10 y: CRC 0.98% vs 1.20% = **18% ↓ incidence**; **no mortality benefit**; uptake 42%; per-protocol 31% / 50%
NordICC (Lancet 2026)	13 y: incidence reduced, **mortality still not**; 30% of endoscopists below ADR of 25%
ECLIPSE / Shield	83% sensitivity for CRC; 90% specificity; **only 13.2% for advanced adenoma**; FDA approved 29 July 2024
FOBT (Pathy's)	Sensitivity 82%
MASAI (Lancet 2026)	105,934 women; **+29% detection, −44% workload, −12% interval cancers**; sensitivity 80.5% vs 73.8% at specificity 98.5%
Mammography ≥70 meta-analysis	28% ↓ breast cancer mortality (RR 0.72); **overdiagnosis ~19%**
ERSPC (NEJM 2025)	162,236 men aged 55–69; 23 y: **13% relative, 0.22% absolute** ↓ prostate cancer mortality
ERSPC Rotterdam (2023)	21 y: RR 0.73; **NNI 246, NND 14**
CAP overdiagnosis analysis	Overdiagnosis **16% at 50 → 32% at 70 → 58% at 80**
ProtecT (NEJM 2023)	15 y: ~3% prostate cancer mortality in **all three arms** (monitoring, surgery, radiotherapy)
PROSA (Eur Urol 2025)	MRI-first detected csPCa **4.6% vs 1.8%**, no rise in GG1 disease
PRECISION (2018)	28% avoided biopsy; 12% more clinically significant cancers detected
TRANSFORM	>250,000 men; £42 million; results not before 2028
LDCT	↓ lung cancer mortality ~20% vs chest X-ray

6. Indian Data — Must-Quote Figures

Item	Figure
LASI Wave 1 (n = 31,464, ≥60)	DT **2.75%**; typhoid 1.84%; hepatitis B 1.82%; influenza **1.59%**; pneumococcal **0.74%**
Adult vaccine coverage overall	**<3%** for most vaccines in older adults
Influenza uptake ≥45 y	≤2% (IJMR rapid review, 2024)
Hepatitis B in healthcare workers	Only 55–64% (Delhi, Mumbai)
Invasive pneumococcal disease	CFR **20–25% at ≥65** vs 5–10% in younger adults
Herpes zoster lifetime risk	~30%, rising to ~50% by age ≥85
Cancer burden	Breast + cervix + oral ≈ **34% of all cancers in India**
NP-NCD screening	Oral, breast, cervical — free at AAMs, **ages 30–65 only**; VIA-based
Cervical screening coverage	10.18 crore of 25.42 crore eligible women screened (July 2025); but **<2%** of women 30–49 screened at NFHS 2019–21
National Cancer Grid	VIA 1–3× lifetime (low resource); HPV q10y (enhanced); HPV q5y at 25–65 (high resource)
ICMR breast screening	Clinical breast examination every **1–3 years**
Programme gaps	Adult vaccines outside **UIP and NPHCE**; **no CRC or prostate screening programme**; **no cancer screening at all above 65**

The Five Sentences That Carry This Whole Topic

1. In geriatrics, prevention is governed by *life expectancy versus lag time to benefit*, not by chronological age.
2. Immunosenescence means older adults need higher-antigen, adjuvanted or recombinant vaccines — and paradoxically the

frailest gain most from preventing hospitalisation while responding least immunologically.
3. The global immunization framework has moved from age-based schedules to *life-course, risk-profile* immunization; age thresholds for pneumococcal, zoster and RSV have all fallen to 50.
4. Cancer screening in the old is limited less by the test's accuracy — which often improves with age — than by *competing mortality and overdiagnosis*.
5. In India the limiting factor is not the guideline but the programme: adult vaccines lie outside the UIP and NPHCE, and organised cancer screening stops at 65.

MD Geriatrics ××× Cardiology Answer Bank

MD Geriatric Medicine — Cardiology Answer Bank

15-Mark University Answer Format

Primary source: *Pathy's Principles and Practice of Geriatric Medicine*, 6th edition (2022) and *Essentials of Clinical Geriatrics*, 7th edition. Updates drawn from guidelines and peer-reviewed literature 2023 onward.

Units are Indian/conventional throughout.

Q1. Management of ST-Elevation Myocardial Infarction and Recent Advances

15–20 High-Yield Points

DEFINITION AND DIAGNOSIS

1. **STEMI** is an acute coronary syndrome caused by **complete thrombotic occlusion** of a coronary artery following **plaque rupture or erosion** — a **Type 1 myocardial infarction**.
2. Diagnosis rests on three things: **clinical presentation**, **ST-segment elevation on ECG**, and a **rise in high-sensitivity cardiac troponin**.
3. In older people the presentation is often **atypical** — dyspnoea, confusion, lethargy, epigastric pain, shoulder or back pain, or altered mental status rather than classic chest pain.
4. Only a **minority of older people** with ischaemic heart disease complain of typical cardiac chest pain, so a high index of suspicion is essential.

PRINCIPLE OF REPERFUSION

5. **Total ischaemic time** is the key concept — the greatest benefit from reperfusion occurs within the **first 2–3 hours** after symptom onset.
6. Time targets: **first medical contact to diagnosis <10 minutes**; **first medical contact to balloon (FMCTB) <90 minutes** in a PCI centre; **PCI within 120 minutes** of diagnosis if transfer is needed.
7. **Primary PCI has replaced fibrinolysis** as the preferred reperfusion strategy, provided it can be done immediately by experienced operators in a centre with **24×7 catheterisation laboratory activation**.
8. If PCI is **not possible within 120 minutes**, give **fibrinolysis** immediately, then transfer to a PCI centre. Perform **coronary angiography at 3–24 hours** if lysis succeeds, and **rescue PCI** if it fails.
9. Older patients have an **increased risk of intracranial haemorrhage** with fibrinolytics, so **PCI has clearly superior outcomes** in this age group (TRIANA trial).

ADJUNCTIVE DRUG THERAPY

10. **Dual antiplatelet therapy (DAPT)** — aspirin 75–100 mg daily plus a **P2Y12 inhibitor** — is given to all patients, along with **parenteral anticoagulation**.
11. **Prasugrel** is **not recommended in patients ≥75 years** unless ischaemic risk is very high, because major and fatal bleeding are increased without added benefit.
12. **Ticagrelor** can be used in older patients; its bleeding risk is similar to clopidogrel.
13. **Early high-dose statin therapy** improves outcome. **Beta blockers** and **ACE inhibitors/ARBs** give prognostic benefit after ACS.
14. Add a **proton pump inhibitor** in older patients on DAPT, especially with prior gastrointestinal bleeding.
15. **Unfractionated heparin dose must be adjusted for renal function** in older people to reduce bleeding.

PROCEDURAL AND ELDERLY-SPECIFIC CONSIDERATIONS

16. The **trans-radial approach** is now recommended in all age groups — it reduces access-site bleeding, which is the commonest non-cardiac complication of PCI.
17. **Bleeding is poorly tolerated in the elderly** because of reduced LV function, vascular stiffness, and endothelial dysfunction; peri-procedural bleeding predicts death, reinfarction, and stroke.
18. **Emergency CABG** is rarely needed — reserved for **failed PCI** or **mechanical complications** (papillary muscle rupture, ventricular septal rupture, free wall rupture).
19. An **implantable cardioverter-defibrillator (ICD)** is advised after MI if there is **ventricular fibrillation, sustained VT, or LVEF ≤30%**.
20. **Treatment should never be withheld on the basis of age alone**; instead use **comprehensive geriatric assessment** — frailty, cognition, life expectancy, and patient preference — to guide intensity of care.
21. **Cardiac rehabilitation** improves survival in older coronary patients and should be offered routinely.

Current Practice Update

Textbook position (Pathy's 6th ed.)	Current evidence (2023–2026)
Primary PCI within 120 min; fibrinolysis if not available	Unchanged and reinforced by the **2025 ACC/AHA/ACEP/NAEMSP/SCAI ACS guideline** — the first major US update since 2013
Culprit-lesion PCI, with staged treatment of other vessels	**Complete revascularisation is now Class 1**, and non-culprit PCI may be done **at the index procedure or later** (except in cardiogenic shock)
Clopidogrel, prasugrel, or ticagrelor as P2Y12 options	**Ticagrelor or prasugrel preferred over clopidogrel** after PCI; prasugrel still avoided at ≥75 years
DAPT duration guided by stent type	**Default DAPT ≥12 months** if not high bleeding risk, then **de-escalation to P2Y12 monotherapy** is an accepted option
Intra-aortic balloon pump for shock	**Microaxial flow pump** may be considered in selected cardiogenic shock; prompt revascularisation remains Class 1

Latest Updates (2023–Present)

- The **2025 ACC/AHA/ACEP/NAEMSP/SCAI ACS Guideline** (*Circulation* 2025; *JACC* 2025) consolidates STEMI and NSTEMI into a single document and retires the 2013 STEMI and 2014 NSTE-ACS guidelines.
- **Complete revascularisation** for multivessel disease has been **upgraded from Class 2a to Class 1**, and can now be performed at the index procedure rather than only as a staged procedure (*2025 ACC/AHA ACS Guideline*).
- **Potent P2Y12 inhibitors (ticagrelor or prasugrel) are explicitly preferred over clopidogrel** in patients undergoing PCI, on the basis of superior reduction in major adverse cardiovascular events (*2025 ACC/AHA ACS Guideline*).
- The **2023 ESC ACS Guideline** (*European Heart Journal* 2023) similarly unified the ACS spectrum and recommends the **radial approach as default** and **immediate invasive strategy** for very-high-risk NSTE-ACS.
- **Cardiogenic shock**: prompt revascularisation remains Class 1; **microaxial flow pump support** may be considered in selected patients based on individual risk–benefit (*2025 ACC/AHA ACS Guideline*).
- **Lipid management post-STEMI** has become more aggressive, with early combination therapy (high-intensity statin ± ezetimibe) endorsed to reach LDL-C targets rapidly.

Examiner's Tip — Mark-Earning Lines

- "**Total ischaemic time** determines myocardial salvage; the greatest benefit is within the first 2–3 hours."
- "**FMCTB <90 minutes** in a PCI centre; **PCI within 120 minutes** of diagnosis if transfer is required."
- "**Primary PCI is superior to fibrinolysis in the elderly**, chiefly because of the higher risk of intracranial haemorrhage with lytics (TRIANA)."
- "**Prasugrel is avoided in patients ≥75 years** because of excess major and fatal bleeding."
- "**Radial access** reduces bleeding, and bleeding in the elderly independently predicts death, reinfarction, and stroke."
- "The **2025 ACC/AHA ACS guideline** made **complete revascularisation Class 1** and preferred **ticagrelor/prasugrel over clopidogrel**."
- "**Age alone is never a contraindication** — use comprehensive geriatric assessment to individualise."

Q2. Drug-Eluting Stents — Advantages and Disadvantages over Bare-Metal Stents

15–20 High-Yield Points

BACKGROUND AND MECHANISM

1. **Bare-metal stents (BMS)** are plain metal scaffolds. They abolish elastic recoil but do nothing to prevent **neointimal hyperplasia**, so restenosis rates are high.
2. **Drug-eluting stents (DES)** have a metal platform coated with a **polymer carrying an antiproliferative drug** (sirolimus, everolimus, zotarolimus, biolimus, or paclitaxel).
3. The eluted drug **inhibits smooth muscle proliferation and migration**, thereby suppressing neointimal hyperplasia and restenosis.
4. **Generations of DES**: first generation (thick struts, durable polymer, sirolimus/paclitaxel), second generation (thinner cobalt-chromium struts, biocompatible polymer, everolimus/zotarolimus), and third generation (bioabsorbable polymer or polymer-free).

ADVANTAGES OF DES OVER BMS

5. **Superior reduction in restenosis** — this is the single greatest advantage and the reason DES have superseded BMS.
6. **Marked reduction in target vessel revascularisation** and therefore fewer repeat procedures.
7. **Reduced incidence of myocardial infarction** in subsequent years with third-generation drug-eluting and drug-coated stents.
8. Repeat interventions are **particularly hazardous in older people** (contrast nephropathy, bleeding, vascular injury), so avoiding them is a major geriatric gain.
9. The **SENIOR trial** (patients ≥75 years) showed DES plus **short DAPT** gave a **29% reduction** in the composite of all-cause death, MI, stroke, and revascularisation at one year compared with BMS.
10. Because of this, **the available evidence no longer supports the use of BMS in elderly patients**.

DISADVANTAGES OF DES

11. **Delayed endothelialisation** of the stent struts leaves metal exposed to blood for longer, creating a risk of **late and very late stent thrombosis**.
12. This mandates a **longer duration of DAPT**, traditionally **at least 6 months after DES** versus **1 month after BMS**.
13. Longer DAPT means **more bleeding**, which older patients with multimorbidity tolerate poorly.
14. Older patients frequently need **DAPT interruption** — for example, to allow safe major non-cardiac surgery — and premature interruption after DES carries thrombotic risk.
15. **Polymer hypersensitivity and chronic inflammation** contribute to **neoatherosclerosis** within the stent.
16. DES are **more expensive** than BMS — a real consideration in the Indian public-sector setting.

CURRENT POSITION ON DAPT DURATION

17. In older people, **bleeding is more common than recurrent thrombotic events** after revascularisation, so shorter DAPT is often the safer choice.
18. A short DAPT strategy of **1 month for stable ischaemic heart disease** and **6 months for ACS** with a DES is safe and effective in patients ≥75 years.
19. Emerging data show **1–3 months of DAPT followed by single antiplatelet monotherapy** is possible with newer DES.
20. **Lifelong single antiplatelet therapy** (usually aspirin) follows completion of DAPT in all patients.

Current Practice Update

Textbook position (Pathy's 6th ed.)	Current evidence (2023–2026)
DES require prolonged DAPT (≥6 months), a concern in the elderly	**1–3 months DAPT then P2Y12 monotherapy** is now well established for contemporary DES, even in high-bleeding-risk patients
BMS still an option when short DAPT is needed	**BMS are effectively obsolete**; the 2025 ACC/AHA ACS guideline no longer positions BMS as a strategy to shorten DAPT
Aspirin as the long-term single agent after DAPT	Growing evidence favours **P2Y12 inhibitor (clopidogrel) monotherapy over aspirin** for long-term single antiplatelet therapy
Concern about late stent thrombosis with DES	Very late stent thrombosis is now **rare with second/third-generation DES**; **neoatherosclerosis** is recognised as the dominant late failure mechanism

Latest Updates (2023–Present)

- The **2025 ACC/AHA ACS Guideline** sets **DAPT for at least 12 months** as the default in ACS patients who are not at high bleeding risk, but formally endorses **de-escalation to P2Y12 inhibitor monotherapy after 1–3 months** in patients at high bleeding risk.
- Randomised evidence and meta-analyses through 2024–2025 confirm that **abbreviated DAPT with contemporary DES does not increase ischaemic events** while significantly reducing major bleeding — the strategy of choice in frail older adults.
- **Clopidogrel monotherapy is increasingly preferred over aspirin monotherapy** for chronic single antiplatelet therapy after PCI, based on trials such as HOST-EXAM and its extended follow-up.
- The **2023 ESC ACS Guideline** recommends DES over BMS in all patients regardless of bleeding risk, with DAPT duration then tailored to bleeding risk rather than stent type.
- **Polymer-free and bioabsorbable-polymer DES** and **drug-coated balloons** are expanding the options for patients in whom prolonged DAPT is undesirable.

Examiner's Tip — Mark-Earning Lines

- "DES suppress **neointimal hyperplasia** via an antiproliferative drug on a polymer coating."
- "The chief advantage is **reduced restenosis and target vessel revascularisation**; the chief disadvantage is **delayed endothelialisation** requiring longer DAPT."
- "The **SENIOR trial** showed a **29% reduction** in death/MI/stroke/revascularisation with DES plus short DAPT in patients ≥75 years."
- "**The evidence no longer supports the use of BMS in elderly patients.**"
- "**Short DAPT (1 month stable IHD, 6 months ACS)** with a DES is safe in older people; current practice extends this to **1–3 months followed by monotherapy**."
- "In older people, **bleeding risk exceeds recurrent thrombotic risk** after revascularisation."

Q3. Etiopathogenesis and Management of In-Stent Restenosis

> *Note: In-stent restenosis is discussed only briefly in Pathy's (in the context of DES superiority over BMS). The detailed mechanisms and management below are therefore drawn from current peer-reviewed cardiology literature and guidelines, as permitted.*

15–20 High-Yield Points

DEFINITION

1. **In-stent restenosis (ISR)** is **≥50% diameter stenosis** within the stent or its **5 mm proximal or distal margins** on follow-up angiography.
2. **Clinical ISR** means angiographic restenosis accompanied by **symptoms or objective ischaemia**, and is the clinically relevant entity.
3. ISR occurs in roughly **5–10% of DES implants** and up to **20–30% of BMS implants**.

ETIOPATHOGENESIS

4. **Neointimal hyperplasia** is the classical mechanism — vessel injury during stenting causes **endothelial denudation**, platelet deposition, and inflammation.
5. Released **growth factors and cytokines** drive **smooth muscle cell proliferation and migration** from the media into the intima, with **extracellular matrix deposition**.
6. **Neoatherosclerosis** is the second and increasingly dominant mechanism, especially with DES — new atheromatous plaque with lipid-laden foam cells forms *inside* the stent and may **rupture**, causing late ACS.
7. **Mechanical factors**: stent under-expansion, stent fracture, geographical miss (lesion not fully covered), and stent gap.
8. **Biological factors**: **polymer hypersensitivity**, drug resistance, and persistent chronic inflammation.
9. **Patient factors**: **diabetes mellitus**, chronic kidney disease, and ongoing smoking.
10. **Lesion factors**: long lesions, small vessel diameter (<2.5 mm), bifurcation lesions, ostial lesions, chronic total occlusions, and heavy calcification.
11. **BMS restenosis** is dominated by **neointimal hyperplasia** and presents **early (6–12 months)**; **DES restenosis** is more often due to **neoatherosclerosis** and presents **late (beyond 1–2 years)**.

CLINICAL PRESENTATION

12. Most present as **recurrent stable angina**; about **10–20% present as an acute coronary syndrome**, particularly when neoatherosclerotic plaque ruptures.
13. In older people the presentation may again be **atypical** — exertional dyspnoea, fatigue, or reduced exercise tolerance.

DIAGNOSIS

14. **Coronary angiography** confirms the diagnosis and defines the **Mehran classification** — Type I focal, Type II diffuse intra-stent, Type III proliferative extending beyond the stent, Type IV occlusive.
15. **Intravascular imaging is mandatory** — **IVUS** best assesses stent under-expansion and vessel size; **OCT** best characterises neointimal morphology and detects neoatherosclerosis.
16. Imaging is what distinguishes **mechanical failure** (needs high-pressure dilatation) from **biological failure** (needs an antiproliferative strategy).

MANAGEMENT

17. **Lesion preparation first** — non-compliant balloon at high pressure, **cutting or scoring balloon**, or **intravascular lithotripsy/rotational atherectomy** for underexpanded or calcified segments.
18. **Drug-coated balloon (DCB) angioplasty** is a first-line option — it delivers paclitaxel or sirolimus without adding another metal layer, which is a major advantage in a vessel that already has one or more stents.
19. **Repeat DES implantation** is the alternative first-line option, preferred when there is significant dissection, elastic recoil, or a suboptimal DCB result.
20. **Plain balloon angioplasty alone is inadequate** and has unacceptably high recurrence.
21. **CABG** is considered for recurrent, multi-layered, or diffuse ISR, especially with left main or multivessel involvement — but the risk–benefit must be weighed against frailty in older patients.
22. **Aggressive secondary prevention** is essential: high-intensity statin, glycaemic control, blood pressure control, smoking cessation, and appropriate antiplatelet therapy.

Current Practice Update

Traditional teaching	Current evidence (2023–2026)
ISR is essentially a problem of neointimal hyperplasia	**Neoatherosclerosis** is now recognised as the dominant mechanism of late DES failure and explains ACS presentations
Repeat DES is the default treatment	**DCB and repeat DES are equivalent first-line options**; DCB is preferred to avoid additional metal layers
DCB was available only in Europe	**FDA approval of the AGENT paclitaxel-coated balloon (2024)** made DCB available in the USA and consolidated it as standard therapy
Angiography alone guides ISR treatment	**Intravascular imaging (IVUS/OCT) is now considered essential** to identify the mechanism before choosing therapy

Latest Updates (2023–Present)

- The **AGENT IDE randomised trial** (*JAMA* 2024) showed the paclitaxel-coated balloon reduced the primary composite endpoint to **18% versus 29%** with uncoated balloon (HR 0.59), with significantly less target lesion revascularisation. **Multi-layer ISR was present in 43%** of patients.
- Benefit was **sustained at 2 years** (CRT 2025) and **3 years** (CRT 2026), establishing DCB durability in ISR.
- The **AGENT DCB received FDA approval in 2024** — the first coronary drug-coated balloon approved in the United States, formally endorsing the "leave nothing behind" strategy.
- Network and mixed-treatment meta-analyses (*Circulation: Cardiovascular Interventions* 2025) confirm **DES and DCB are broadly comparable** for ISR, with **both clearly superior to plain balloon angioplasty**.
- The **ISAR-DESIRE 5 trial** is testing an imaging-guided strategy — DCB versus repeat DES **chosen according to neointimal morphology on OCT** — reflecting the shift toward mechanism-directed therapy.
- **Sirolimus-coated balloons** are emerging as an alternative to paclitaxel, with theoretical advantages in drug retention and vessel healing.

Examiner's Tip — Mark-Earning Lines

- "**ISR is ≥50% stenosis within the stent or 5 mm of its margins.**"
- "Two mechanisms: **neointimal hyperplasia** (early, BMS-dominant) and **neoatherosclerosis** (late, DES-dominant, causes ACS)."
- "**Mehran classification** — Type I focal to Type IV occlusive."
- "**Intravascular imaging distinguishes mechanical from biological failure** and directs treatment."
- "First-line treatment is **lesion preparation followed by DCB or repeat DES**; **plain balloon angioplasty alone is inadequate**."
- "The **AGENT IDE trial (JAMA 2024)** established the paclitaxel-coated balloon for ISR, with FDA approval in 2024."

Q4. Newer Cardiac Biomarkers in Myocardial Injury and Their Interpretation

15–20 High-Yield Points

THE CENTRAL BIOMARKER

1. **High-sensitivity cardiac troponin (hs-cTn) I and T** is the **gold standard** biomarker of myocardial injury and the cornerstone of ACS diagnosis.
2. **Myocardial injury** is defined by a troponin value **above the 99th percentile upper reference limit**. It becomes **acute** when there is a **rise and/or fall** on serial testing.
3. **Myocardial infarction** requires evidence of injury **plus clinical evidence of ischaemia** — symptoms, ECG change, imaging evidence, or thrombus on angiography.
4. **Troponin elevation is not synonymous with myocardial infarction** — this is the single most important interpretive principle.

CLASSIFICATION OF MYOCARDIAL INFARCTION

5. **Type 1 MI** — plaque rupture or erosion with coronary thrombosis. This is atherothrombotic.
6. **Type 2 MI** — **oxygen supply–demand mismatch** without plaque rupture; the myocardium is starved rather than blocked.
7. Common causes of Type 2 MI in older people are **sepsis, hypotension, tachyarrhythmias, anaemia, hypoxaemia, and organ dysfunction**.
8. Older people are **far more likely to have raised hs-troponin from Type 2 MI** than younger individuals, because of demand-supply mismatch causing non-atherosclerotic myonecrosis.
9. **Type 2 MI carries a poor prognosis** — mortality up to **63% at 3 years** — but the deaths are driven by **comorbidities rather than myocardial damage**, and **PCI is unlikely to help**.
10. **Types 3, 4 and 5** cover sudden cardiac death before biomarker sampling, PCI-related MI, and CABG-related MI respectively.

CHRONIC AND NON-ISCHAEMIC ELEVATION

11. **Chronic troponin elevation** without a rise-and-fall pattern occurs in **chronic kidney disease, heart failure, left ventricular hypertrophy, amyloidosis, and advanced age itself**.
12. Other non-ischaemic causes: **myocarditis, pulmonary embolism, stroke and subarachnoid haemorrhage, cardiac contusion, cardioversion, ablation, and chemotherapy-related cardiotoxicity**.
13. In old age, elevated troponin combined with **non-specific symptoms and age-related baseline ECG changes** (Q waves, bundle branch block) can lead to a **false diagnosis of ACS**.

NATRIURETIC PEPTIDES

14. **BNP and NT-proBNP** are markers of **myocardial wall stress**, not injury, and are the primary rule-out test for heart failure.
15. ESC thresholds for further investigation: **BNP >35 pg/ml** and **NT-proBNP >125 pg/ml** in the non-acute setting. **Normal levels make heart failure unlikely.**
16. They are **equally predictive in HFrEF and HFpEF** and carry independent prognostic information.
17. Levels **rise with age and with renal impairment**, and **fall with obesity** — all must be factored into interpretation.

NEWER AND ADJUNCTIVE MARKERS

18. **Cardiac myosin-binding protein C (cMyC)** is the most promising competitor to troponin — it is released earlier and is currently the leading candidate for very early rule-out.
19. **Copeptin**, the C-terminal fragment of provasopressin, is a marker of **endogenous stress**; combined with troponin it allows a **dual-marker single-draw rule-out**, but it lacks cardiac specificity.
20. **GDF-15, sST2, galectin-3, and hs-CRP** are **prognostic rather than diagnostic** — they reflect oxidative stress, fibrosis, and inflammation, and predict mortality and hospitalisation.
21. **GDF-15 and hs-cTnT are components of the ABC bleeding risk score** in atrial fibrillation, illustrating the biomarker-based risk-scoring approach.
22. Adding **NT-proBNP, hs-cTnT, and hs-CRP** to the pooled cohort equation improved **4-year cardiovascular risk prediction** in adults aged 69–88 years (ARIC study).

Current Practice Update

Textbook position (Pathy's 6th ed.)	Current evidence (2023–2026)
Serial troponin at 0 and 3–6 hours	**ESC 0/1-hour algorithm** is now the preferred rapid rule-in/rule-out pathway, with **0/2-hour** as an alternative
Copeptin useful as an adjunct	**2023 ESC ACS Guideline does not recommend copeptin or cMyC routinely** unless hs-cTn or rapid protocols are unavailable
Type 2 MI recognised but management unclear	Type 2 MI is now managed by **treating the precipitant**; routine invasive strategy is discouraged
Novel markers (sST2, galectin-3, GDF-15) considered emerging diagnostics	Now firmly positioned as **prognostic/risk-stratification tools only** — they lack cardiac specificity for diagnosis

Latest Updates (2023–Present)

- The **2023 ESC ACS Guideline** endorses the **hs-cTn 0/1-hour algorithm** (or 0/2-hour) in preference to serial testing based on the 99th percentile, and permits **immediate rule-out when hs-cTn is below the limit of detection** in patients presenting more than 3 hours after symptom onset.
- The **2025 ACC/AHA ACS Guideline** confirms **high-sensitivity troponin as the preferred biomarker** for evaluating suspected ACS.
- A large single-centre study of **1,765 patients** (*Journal of Clinical Medicine* 2025) compared **cMyBP-C, copeptin, GDF-15, BMP10, FABP3 and FGF23** against the ESC algorithms and found **cMyC to be the only viable competitor to troponin**; the others added prognostic but not diagnostic value.
- A **2025 review of myocardial injury biomarkers** (*Expert Review of Cardiovascular Therapy* 2025) concluded that copeptin, galectin-3 and sST2 **lack cardiac specificity**, limiting their diagnostic utility, while **cMyC remains the most credible alternative** to cTn.
- **Point-of-care hs-cTn assays** are being validated for use in emergency departments and pre-hospital settings, which is particularly relevant to Indian district hospital practice.
- **Sex-specific and age-specific 99th percentile thresholds** are increasingly recommended to reduce over-diagnosis in older women and under-diagnosis in older men.

Examiner's Tip — Mark-Earning Lines

- "**Myocardial injury** = troponin above the **99th percentile URL**; **acute injury** = a **rise and/or fall**; **infarction** = injury **plus** evidence of ischaemia."
- "**Troponin elevation is not synonymous with myocardial infarction.**"
- "**Type 2 MI** results from **supply–demand mismatch** — sepsis, hypotension, tachyarrhythmia, anaemia — and is far commoner in the elderly."
- "**Type 2 MI mortality reaches 63% at 3 years**, driven by comorbidity, and **PCI is unlikely to be effective**."
- "**BNP >35 pg/ml** and **NT-proBNP >125 pg/ml** prompt echocardiography; normal levels make heart failure unlikely."
- "**Natriuretic peptides rise with age and renal impairment and fall with obesity.**"
- "The **ESC 0/1-hour hs-cTn algorithm** is the current standard rapid diagnostic pathway."
- "**GDF-15, sST2 and galectin-3 are prognostic, not diagnostic.**"

Q5. AHA Guidelines for Stroke Prevention in Atrial Fibrillation

15–20 High-Yield Points

BURDEN AND RATIONALE

1. **Atrial fibrillation (AF)** is the commonest sustained arrhythmia in older people and is a major cause of **cardioembolic stroke**, heart failure, and death.
2. Stroke in AF arises mainly from thrombus in the **left atrial appendage (LAA)** — hence the anatomical basis of all preventive strategies.
3. The **absolute stroke reduction from oral anticoagulation is greatest in the elderly**, because baseline stroke risk rises steeply with age and outweighs the bleeding risk.

RISK STRATIFICATION

4. **All patients with AF must be routinely scored** for stroke risk — the recommended tool is the **CHA2DS2-VASc score**.
5. Anticoagulation is a **Class I recommendation** at **CHA2DS2-VASc ≥2 in men** and **≥3 in women**. It **should be considered** at a score of **1 in men** and **2 in women**.
6. **All patients over 75 with AF should therefore be anticoagulated** unless there is a major contraindication.
7. **Bleeding risk must be assessed in parallel** using **HAS-BLED, ORBIT, ATRIA, HEMORR2HAGES**, or the biomarker-based **ABC bleeding score**.
8. A high bleeding score is **not a reason to withhold anticoagulation** — it identifies **modifiable risk factors** (uncontrolled BP, labile INR, alcohol, concomitant NSAIDs/antiplatelets) that should be corrected.

CHOICE OF AGENT

9. In **non-valvular AF, DOACs are recommended over warfarin** (Class I) in patients without contraindications.
10. **Warfarin is mandatory** in **moderate-to-severe mitral stenosis** and in **mechanical prosthetic heart valves** (Class I) — DOACs are contraindicated and less effective in these groups.
11. Warfarin efficacy depends on **time in therapeutic range (TTR)** with a target **INR 2–3**; the higher the TTR, the lower the risk of both stroke and bleeding.
12. **Antiplatelet therapy is inferior** — a meta-analysis of nine trials showed **VKA superior to aspirin with a 39% relative risk reduction**, and **ACTIVE-W** showed anticoagulation superior to aspirin–clopidogrel (RRR 40%).

KEY TRIAL EVIDENCE

13. **BAFTA** (age ≥75): warfarin (INR 2–3) reduced stroke, systemic embolism, and intracranial bleeding by **52%** versus aspirin 75 mg, with **no difference in major haemorrhage**.
14. **RE-LY**: dabigatran **150 mg BD reduced stroke/systemic embolism by 35%**; **110 mg BD** was non-inferior with **20% less bleeding**.
15. **ARISTOTLE**: apixaban 5 mg BD reduced stroke/systemic embolism by **21%**, major bleeding by **31%**, and death by **11%**.
16. **ENGAGE AF-TIMI 48**: edoxaban 60 mg OD had similar efficacy with **20% less major bleeding**. **ROCKET-AF**: rivaroxaban 20 mg OD had similar efficacy with less intracranial and life-threatening bleeding.
17. **AVERROES**: apixaban was superior to aspirin in patients unsuitable for warfarin, with an acceptable safety profile.
18. In patients **>75 years, DOACs as a class were superior to warfarin in both efficacy and safety**. **Apixaban had the lowest risk of major bleeding**, and apixaban, edoxaban, and dabigatran had **lower rates of intracranial haemorrhage** than rivaroxaban or warfarin.

NON-PHARMACOLOGICAL OPTIONS AND SPECIAL SITUATIONS

19. **Percutaneous LAA occlusion (WATCHMAN)** is an option for patients with high stroke risk and a **non-reversible contraindication to long-term anticoagulation** — for example prior intracranial haemorrhage on OAC, or recurrent embolic stroke despite adequate anticoagulation.
20. **Surgical LAA excision or exclusion** should be considered in AF patients undergoing cardiac surgery; concomitant surgical AF ablation reduces stroke and has a **Class I indication**.
21. After **cardioembolic stroke**, delay starting anticoagulation for **about 2 weeks** because early benefit is offset by haemorrhagic transformation — but start immediately after **TIA or minor stroke**.
22. **Falls risk and dementia are frequently used as inappropriate reasons to withhold anticoagulation**; only end-stage dementia clearly tips the balance against treatment.

Current Practice Update

Textbook position (Pathy's 6th ed.)	Current evidence (2023–2026)
Anticoagulate on the basis of CHA2DS2-VASc score alone	The **2023 ACC/AHA/ACCP/HRS AF Guideline** bases the decision on **annual thromboembolic risk (≥2%/year = Class 1)**, and permits **ATRIA or GARFIELD-AF** as alternative scores
Female sex is a risk factor scoring 1 point	Female sex is now a **risk modifier**, not an independent risk factor; the 2024 ESC guideline uses **CHA2DS2-VA** (sex removed)
LAA occlusion for those unable to take OAC	LAA occlusion given a **higher class of recommendation**; **OPTION trial (2024)** supports LAAO after ablation in selected patients
Device-detected AF handled case by case	**Prescriptive recommendations** now exist based on **AHRE duration** (<5 min, 5 min–24 h, ≥24 h) from NOAH-AFNET 6 and ARTESiA

Latest Updates (2023–Present)

- The **2023 ACC/AHA/ACCP/HRS AF Guideline** (*Circulation* 2023; *JACC* 2023) shifted from a score-threshold approach to **anticoagulation based on estimated annual stroke risk**: **≥2% per year is a Class 1 indication**, equivalent to CHA2DS2-VASc ≥2 in men and ≥3 in women.
- The same guideline introduced **AF stages** (including a pre-detection stage), emphasised **aggressive risk-factor modification**, and favoured **early rhythm control**.
- **Device-detected AF**: for an atrial high-rate episode **≥24 hours with CHA2DS2-VASc ≥2**, initiating anticoagulation is reasonable; for episodes of **5 minutes to 24 hours with CHA2DS2-VASc ≥3**, it may be reasonable; for episodes **<5 minutes**, anticoagulation should **not** be given.
- The **2024 ESC AF Guideline** (*European Heart Journal* 2024) adopted **CHA2DS2-VA**, dropping the sex category, to simplify decision-making and avoid sex-based treatment disparity.
- **Percutaneous LAA occlusion** received a stronger recommendation, and the **OPTION trial** (presented AHA 2024) supported LAAO as an alternative to DOAC after catheter ablation in selected patients.
- **Factor XI inhibitors** are the major emerging class — **AZALEA-TIMI 71** (*NEJM* 2023) showed abelacimab reduced major or clinically relevant non-major bleeding by **62–69% versus rivaroxaban**, but the phase 3 **OCEANIC-AF** trial of asundexian was **stopped early for inferiority to apixaban**. **LILAC-TIMI 76** (abelacimab versus placebo in patients unsuitable for OAC) and **LIBREXIA-AF** (milvexian versus apixaban) will report from late 2026.

Examiner's Tip — Mark-Earning Lines

- "**Thrombus forms in the left atrial appendage** — the anatomical basis for all preventive strategies."
- "**Anticoagulate at CHA2DS2-VASc ≥2 in men, ≥3 in women (Class I)** — so effectively **all AF patients over 75**."
- "**High bleeding score is not a reason to withhold anticoagulation** — it flags modifiable risk factors."
- "**DOACs are preferred over warfarin in non-valvular AF**; **warfarin is mandatory in moderate-severe mitral stenosis and mechanical valves**."
- "**BAFTA: 52% relative risk reduction with warfarin over aspirin in patients ≥75.**"
- "**Apixaban has the lowest major bleeding risk** among DOACs in the over-75 population."
- "The **2023 ACC/AHA AF guideline** moved to **risk-based (≥2%/year) rather than score-threshold** anticoagulation; the **2024 ESC guideline adopted CHA2DS2-VA**."

Q6. CHA2DS2-VASc Score — Use and Clinical Significance

15–20 High-Yield Points

PURPOSE

1. **CHA2DS2-VASc** is a **clinical risk-stratification score** that estimates the **annual risk of ischaemic stroke and systemic embolism** in patients with **non-valvular atrial fibrillation**.
2. It was developed to refine the earlier **CHADS2 score**, which classified too many patients as "low risk" and missed those who would benefit from anticoagulation.
3. Its purpose is **decision-making** — it balances the reduction in stroke risk against the increased risk of bleeding from anticoagulation.

COMPONENTS (MAXIMUM SCORE 9)

4. **C — Congestive cardiac failure** (or LV dysfunction): **1 point**
5. **H — Hypertension**: **1 point**
6. **A2 — Age ≥75 years**: **2 points**
7. **D — Diabetes mellitus**: **1 point**
8. **S2 — Stroke / TIA / thromboembolism**: **2 points**
9. **V — Vascular disease** (prior MI, peripheral arterial disease, or aortic plaque): **1 point**
10. **A — Age 65–74 years**: **1 point**
11. **Sc — Sex category (female)**: **1 point**
12. The **maximum score is 9**, not 10, because **age can score only 0, 1 or 2** — it cannot count in both age bands.

CLINICAL APPLICATION

13. **Score ≥2 in men or ≥3 in women** — oral anticoagulation is **recommended (Class I)**.
14. **Score of 1 in men or 2 in women** — anticoagulation **should be considered**, after shared decision-making.
15. **Score 0 in men or 1 in women (female sex alone) — no antithrombotic therapy** is required.
16. **Age ≥75 alone gives 2 points**, so **every older person with AF qualifies for anticoagulation** unless contraindicated. This is the single most important geriatric implication of the score.
17. The score should be **reassessed at least annually**, because risk factors accumulate dynamically with ageing.

WIDER USES AND LIMITATIONS

18. The score is also used in **device-detected atrial high-rate episodes**, to guide **LAA occlusion** eligibility, and to inform **peri-procedural anticoagulation** decisions.
19. **Limitation** — it does **not include bleeding risk**, so it must always be paired with a bleeding score such as **HAS-BLED, ORBIT, ATRIA or ABC**.
20. **Limitation** — it **omits renal impairment, frailty, anaemia, cognitive status, and AF burden**, all highly relevant in geriatric practice.
21. **Limitation** — it has **modest discriminatory power (c-statistic ~0.6)** and was validated in populations under-representing the very old.
22. **Limitation** — because **age contributes so heavily**, the score has **poor discrimination within the elderly population**, where almost everyone scores ≥2.

Current Practice Update

Textbook position (Pathy's 6th ed.)	Current evidence (2023–2026)
CHA2DS2-VASc is the single recommended score	The **2023 ACC/AHA AF Guideline** endorses **CHA2DS2-VASc, ATRIA, or GARFIELD-AF**, allowing flexibility
Decision based on the score threshold	Decision now based on **estimated annual event rate — ≥2% per year is Class 1**
Female sex scores 1 point as a risk factor	Female sex is now a **risk modifier**; the **2024 ESC guideline uses CHA2DS2-VA**, dropping the sex category entirely
Score used at diagnosis	Score should be **reassessed at least annually**, as risk is dynamic

Latest Updates (2023–Present)

- The **2023 ACC/AHA/ACCP/HRS AF Guideline** allows use of **other validated risk scores (ATRIA, GARFIELD-AF)** alongside CHA2DS2-VASc, particularly for **patients at intermediate risk** where the anticoagulation decision is

genuinely uncertain.

- The guideline introduced the concept of **stroke risk modifiers** — AF burden, blood pressure control, and other dynamic factors — for patients with an annual risk below 2%.
- The **2024 ESC AF Guideline** formally adopted **CHA2DS2-VA (maximum 8 points)**, removing sex category, on the evidence that **women's excess stroke risk is not evident until other risk factors are present**.
- Studies show that applying the lower ACC/AHA hypertension thresholds causes **"up-scoring"** of CHA2DS2-VASc, reclassifying many patients into the anticoagulation-eligible category — a recognised limitation of a score built on binary comorbidity definitions (*JAMA Cardiology* 2023).
- There is an ongoing move toward **biomarker-enhanced scores** such as the **ABC stroke score** (age, biomarkers — NT-proBNP and hs-cTnT — and clinical history), which outperform CHA2DS2-VASc in trial cohorts but have shown **limited advantage in real-world elderly populations**.

Examiner's Tip — Mark-Earning Lines

- "Congestive failure 1, Hypertension 1, **Age ≥75 = 2**, Diabetes 1, **Stroke/TIA = 2**, Vascular disease 1, Age 65–74 = 1, Sex (female) 1."
- "**Maximum score is 9, not 10** — because age scores either 1 or 2, never both."
- "**Anticoagulate at ≥2 in men, ≥3 in women (Class I).**"
- "**Age ≥75 alone scores 2** — so every older patient with AF qualifies for anticoagulation."
- "The score **assesses stroke risk only** and must always be paired with a **bleeding risk score**."
- "It **omits renal function, frailty, anaemia and AF burden**, and has **poor discrimination within the elderly**."
- "The **2024 ESC guideline moved to CHA2DS2-VA**, removing sex category."

Q7. Lifestyle Modification to Reduce Cardiovascular Risk

15–20 High-Yield Points

GENERAL PRINCIPLE

1. **Compliance with healthy lifestyle recommendations reduced mortality and the prevalence of chronic disease** in a cohort of people aged 65–98 years — the benefits of prevention apply **regardless of age or number of risk factors**.
2. Standard risk calculators such as the **pooled cohort equation are not validated beyond age 79**, so lifestyle advice should be given universally rather than being made conditional on a calculated risk score.

SMOKING CESSATION

3. **Smoking is a major cardiovascular risk factor** associated with increased mortality, and **cessation is beneficial even for those who quit late in life**.
4. Smoking also increases the risk of heart failure — relative risk **1.75 in current smokers** and **1.16 in former smokers** — and the risk falls with years of abstinence.
5. A **structured smoking cessation programme** should be strongly recommended to every older smoker.

PHYSICAL ACTIVITY

6. **All movement counts.** In 5,861 older women (mean age 78.5 years), the highest quartile of **light physical activity** was associated with a **42% reduction** in myocardial infarction or coronary death.
7. In the **Cardiovascular Health Study** (4,207 subjects, mean age 72.5), greater physical activity was inversely associated with IHD, stroke, and total cardiovascular disease **even in those aged ≥75**.
8. **Walking pace and walking distance** were each independently associated with lower risk — important because walking is the commonest form of activity in older people.
9. In a Finnish study of 2,456 people aged 65–74, **leisure-time physical activity reduced total and cardiovascular mortality in a dose-dependent manner**, independent of baseline risk factors.
10. In 2,153 older men (mean age 75), **mortality risk was 48% lower in the most fit compared with the least fit** individuals.
11. **Three months of exercise-based lifestyle intervention** lowers blood pressure by approximately **5 mmHg systolic and 3 mmHg diastolic** in older individuals.
12. Exercise prescription should include **aerobic, resistance, balance, and flexibility** components — resistance training is essential to counter sarcopenia.

DIET

13. Meta-analyses of observational studies show an **overall protective effect of the Mediterranean diet** on cardiovascular disease.
14. **Supplementing the Mediterranean diet with extra-virgin olive oil or nuts produced a 30% reduction in major cardiovascular events** in people aged 55–80 (PREDIMED).
15. Higher circulating **omega-3 polyunsaturated fatty acids from seafood** were associated with a lower risk of unhealthy ageing — dietary fish intake in older people should be increased.
16. The **Mediterranean diet is preferred over a conventional low-fat, high-carbohydrate diet**, because low-fat diets lower HDL-C as much as LDL-C.
17. **Low sodium intake (<2 g/day)** reduces systolic BP by **3.47 mmHg** and diastolic BP by **1.81 mmHg**. High sodium intake is associated with increased risk of **stroke (RR 1.24), stroke mortality (1.63), and coronary heart disease mortality (1.32)**.

WEIGHT, ALCOHOL AND OTHER MEASURES

18. **Maintenance of ideal body weight** helps reduce BP, but **restrictive diets that impair quality of life are inappropriate in older people** — elevated weight short of frank obesity is associated with longer life than being underweight.
19. **Low alcohol consumption** is advised; heavy drinking causes alcoholic cardiomyopathy and heart failure.
20. **Sodium restriction to about 1500 mg/day** is prudent in heart failure, mainly for blood pressure control.
21. **Cardiac rehabilitation** — a structured, supervised lifestyle programme — improves survival in older coronary patients and reduces depression.
22. In older people a **sedentary lifestyle showed the strongest association with incident heart failure**, while the associations of smoking and hypertension attenuated with age.

Current Practice Update

Textbook position (Pathy's 6th ed.)	Current evidence (2023–2026)
Lifestyle described as individual risk-factor measures	**AHA "Life's Essential 8"** provides a single composite cardiovascular health score covering diet, activity, nicotine, sleep, weight, lipids, glucose and BP
Nicotine exposure means cigarette smoking	Now includes **e-cigarettes, vaping and second-hand smoke**
Sleep not listed as a cardiovascular risk factor	**Sleep duration is now a formal component** of cardiovascular health scoring
Pooled cohort equation for risk estimation	**PREVENT equations** (2023) have replaced pooled cohort equations, incorporating **renal function, statin use and social drivers of health**
Weight loss achieved by lifestyle alone	**GLP-1 receptor agonists** now have proven cardiovascular outcome benefit and are an adjunct in selected patients

Latest Updates (2023–Present)

- The **AHA "Life's Essential 8" framework** (an update of Life's Simple 7) adds **sleep health** as an eighth metric. A **10-point increase in the LE8 score significantly reduces all-cause and cardiovascular mortality** (*Journal of Cardiology* 2025).
- The **PREVENT risk equations** were adopted by the **2025 AHA/ACC Hypertension Guideline**, replacing the pooled cohort equations and improving risk estimation by including **renal function, statin use, and social drivers of health**.
- The **2025 AHA/ACC Hypertension Guideline** recommends lifestyle counselling for all hypertensive patients: **≥5% weight loss if overweight, DASH diet, sodium reduction, alcohol reduction or abstinence, and increased physical activity**, with drug therapy added if BP remains ≥130/80 mmHg after a **3–6 month lifestyle trial** in low-risk stage 1 hypertension (PREVENT <7.5%).
- The **SELECT trial** (*NEJM* 2023) showed **semaglutide 2.4 mg weekly reduced major adverse cardiovascular events** in overweight or obese patients with established cardiovascular disease and no diabetes; FDA approval for MACE reduction followed in 2024. Mean age was 61 years, so applicability to the very old still requires caution.
- The **2025 AHA/ACC Hypertension Guideline** explicitly mentions **GLP-1 receptor agonists** as a possible addition for selected patients with hypertension and overweight or obesity.
- Physical activity guidance has moved from a fixed threshold to a **dose-response model** — **any increase above baseline confers benefit**, which is a far more achievable message for frail older adults.

Examiner's Tip — Mark-Earning Lines

- "**Smoking cessation is beneficial even for those who quit late in life.**"
- "**All movement counts** — light physical activity gave a **42% reduction** in MI or coronary death in older women."
- "**PREDIMED**: Mediterranean diet with olive oil or nuts gave a **30% reduction** in major cardiovascular events."
- "**Sodium <2 g/day lowers SBP by ~3.5 mmHg**; high intake raises stroke risk (RR 1.24)."
- "**Restrictive diets that impair quality of life are inappropriate in older people.**"
- "**Sedentary lifestyle shows the strongest association with incident heart failure in old age.**"
- "**Life's Essential 8** adds **sleep** to the traditional seven metrics; the **PREVENT equations** now replace the pooled cohort equations."

Q8. Causes of Secondary Hypertension — Evaluation and Treatment

15–20 High-Yield Points

DEFINITION AND EPIDEMIOLOGY

1. **Secondary hypertension** is high blood pressure due to an **identifiable and often correctable underlying cause**.
2. It is **uncommon in older adults overall**, but should be actively considered in **treatment-resistant hypertension** and in those with **diastolic pressures >115 mmHg**.
3. **Atherosclerotic renovascular hypertension and primary hyperaldosteronism occur more frequently in older persons** than in the young.
4. **Pheochromocytoma is uncommon in older adults** and particularly unusual after age 75.

CAUSES AND THEIR CLINICAL MARKERS

5. **Renovascular disease** — abdominal bruit, a **rise in serum creatinine of >30% after starting an ACE inhibitor or ARB**, and **asymmetrical kidney size differing by >1.5 cm** on ultrasound.
6. **Renoparenchymal disease** — often asymptomatic; raised urea and creatinine, **small kidneys**, and abnormal urinalysis.
7. **Conn's syndrome (primary hyperaldosteronism)** — **elevated serum aldosterone with resistant hypokalaemia**.
8. **Cushing's syndrome** — elevated serum cortisol, **cushingoid facies**, persistent hypokalaemia.
9. **Pheochromocytoma** — **elevated urinary metanephrines**, episodic sympathetic overactivity (headache, palpitation, sweating).
10. **Hypothyroidism** — constipation, bradycardia, **high TSH with low thyroxine**. **Hyperthyroidism** — diarrhoea, tachycardia, **low TSH with high thyroxine**.
11. **Hyperparathyroidism** — **high serum calcium and PTH**, constipation. Increasingly diagnosed with automated calcium testing, particularly in postmenopausal women; treating it may ameliorate the blood pressure.
12. **Drugs** — **NSAIDs and corticosteroids** are the commonest culprits; also sodium-containing antacids, antidepressants, nicotine, and oral contraceptives.
13. **Obstructive sleep apnoea** — daytime sleepiness and nocturnal snoring.
14. **Excess alcohol** — established by careful history taking.
15. **Oestrogen therapy** in the postmenopausal woman may cause hypertension; this can be tested by **withdrawing oestrogen for several months** and observing the blood pressure response.

MIMICS THAT MUST BE EXCLUDED FIRST

16. **Pseudohypertension** — falsely elevated readings due to **non-collapsible sclerotic arteries**. Suspect it in apparently resistant hypertension **with no end-organ damage**. Detected by the **Osler manoeuvre** (radial artery remains palpable when the cuff is inflated above systolic pressure), which suggests a **10–15 mmHg error**; confirmed by **intra-arterial monitoring**.
17. **White-coat hypertension** — high office but normal home BP. **Masked hypertension** — normal office but high home BP. Both need **ambulatory or home BP monitoring**.
18. **Non-adherence and inadequate diuretic dosing** are the commonest reasons for apparent resistance and must be excluded before labelling a patient resistant.

EVALUATION

19. **Resistant hypertension** is BP uncontrolled despite **three or more antihypertensive agents including a diuretic**.
20. Initial laboratory evaluation: **urinalysis, complete blood count, serum electrolytes and calcium, estimated GFR, fasting glucose, lipids, and a 12-lead ECG**.
21. Second-line testing is directed by clinical suspicion — **aldosterone-renin ratio, urinary metanephrines, TSH, renal Doppler/CT angiography, overnight dexamethasone suppression test, polysomnography**.

TREATMENT

22. **Treat the cause where possible** — adrenalectomy for aldosterone-producing adenoma or pheochromocytoma, parathyroidectomy for hyperparathyroidism, thyroid hormone replacement, CPAP for sleep apnoea, and withdrawal of the offending drug.
23. **Spironolactone or eplerenone** is the drug of choice in primary aldosteronism and in resistant hypertension generally. **Eplerenone avoids gynaecomastia** but both require careful **potassium and renal function monitoring** in older patients.
24. Renal artery **revascularisation is reserved** for flash pulmonary oedema, rapidly deteriorating renal function, or truly refractory hypertension — medical therapy is otherwise preferred.

25. **Targets should be individualised by functional status** rather than chronological age — approximately **<130/80 mmHg in the independent older person, <140/90 in the partially dependent, and <150/90 in the dependent or frail**.

Current Practice Update

Textbook position (Pathy's / Essentials)	Current evidence (2023–2026)
Screen for primary aldosteronism only in resistant hypertension **with hypokalaemia**	**2025 AHA/ACC Guideline: screen all patients with resistant hypertension irrespective of potassium**, since PA commonly exists without hypokalaemia
Antihypertensives usually withheld before aldosterone-renin ratio testing	**Continue all antihypertensives except MRAs** during screening, to avoid delays and barriers
Screening confined to specific clinical settings	Screening **expanded to stage 2 and resistant hypertension**; the **2025 Endocrine Society guideline** goes further and recommends screening **all hypertensive patients**
Renal denervation not established	**Renal denervation is now an adjunctive option** for selected patients with resistant hypertension
Pooled cohort equations for risk assessment	**PREVENT risk calculator** now used

Latest Updates (2023–Present)

- The **2025 AHA/ACC Hypertension Guideline** — the first major update since 2017 — recommends **screening for primary aldosteronism in all adults with resistant hypertension regardless of whether hypokalaemia is present** (Class 1, LOE B-NR), because PA frequently occurs with normal potassium.
- The same guideline recommends **continuing most antihypertensive medications (all except MRAs) during aldosterone-renin ratio testing**, to remove a major practical barrier to diagnosis.
- Screening indications also include **unexplained hypokalaemia, obstructive sleep apnoea, incidental adrenal mass, family history of early-onset hypertension, and stroke before age 40** (*2025 AHA/ACC Hypertension Guideline*).
- **Renal denervation** has been introduced as an **adjunctive option in selected patients with resistant hypertension** (*2025 AHA/ACC Hypertension Guideline*).
- The **2025 Endocrine Society primary aldosteronism guideline** and the **2024 ESC hypertension guideline** both move toward **broader, near-universal PA screening**, reflecting recognition that PA is substantially under-diagnosed.
- The guideline recommends **initiating two agents at once, preferably as a single-pill combination**, and notes that **fewer cardiovascular events and deaths occur with single-pill combinations** than with equivalent multi-pill regimens.

Examiner's Tip — Mark-Earning Lines

- "**Renovascular disease and primary hyperaldosteronism are the commonest secondary causes in the elderly**; pheochromocytoma is unusual after 75."
- "Suspect renovascular disease if creatinine **rises >30% after starting an ACE inhibitor/ARB** or kidneys differ by **>1.5 cm**."
- "**Pseudohypertension** — resistant hypertension **without end-organ damage**; test with the **Osler manoeuvre**; error is **10–15 mmHg**."
- "**Resistant hypertension = uncontrolled despite three agents including a diuretic.**"
- "**Exclude non-adherence, inadequate diuretic dose, NSAIDs and white-coat effect before labelling resistance.**"
- "The **2025 AHA/ACC guideline screens all resistant hypertensives for primary aldosteronism regardless of potassium**, and **continues all drugs except MRAs** during testing."
- "**Targets should be individualised by functional status, not chronological age.**"

Q9. Complete Heart Block

15–20 High-Yield Points

DEFINITION AND ELECTROPHYSIOLOGY

1. **Complete (third-degree) heart block** is a **total failure of conduction between the atria and the ventricles**, resulting in **regular atrial activity with an independent ventricular escape rhythm** — atrioventricular dissociation.
2. The escape rhythm is **usually ventricular in origin**, with a **wide QRS complex** and a rate of about **30–40 beats per minute**.
3. A **nodal or junctional escape** with a narrow QRS implies the anatomical level of block is **higher — within the AV node or bundle of His**.
4. **The lower the origin of the escape rhythm, the less specialised and therefore the less reliable the conduction tissue** — this determines both the rate and the risk of asystole.

AETIOLOGY IN OLDER PEOPLE

5. **Idiopathic age-related fibrosis** of the conduction system (Lev's and Lenègre's disease) is the commonest cause in the elderly.
6. **Ischaemic heart disease** — acute inferior MI (usually reversible, nodal level) or anterior MI (infranodal, poor prognosis).
7. **Drugs** — beta blockers, non-dihydropyridine calcium channel blockers (verapamil, diltiazem), digoxin, and antiarrhythmics. **Concomitant use of diltiazem or verapamil with beta blockers increases the risk of bradycardia, heart block and asystole, particularly in older people.**
8. **Infiltrative disease** — cardiac sarcoidosis, amyloidosis.
9. **Neuromuscular disease — myotonic dystrophy type 1**, **Kearns-Sayre syndrome**, lamin A/C mutations.
10. **Iatrogenic** — following cardiac surgery, TAVI, or catheter ablation of the AV junction.
11. **Infective** — infective endocarditis with aortic root abscess, Lyme carditis; also hyperkalaemia and hypothyroidism as reversible causes.

CLINICAL FEATURES

12. **Syncope (Stokes-Adams attack)**, presyncope, dizziness, fatigue, exertional breathlessness, and worsening heart failure.
13. Examination: **slow regular pulse**, **cannon 'a' waves** in the JVP, **variable intensity of the first heart sound**, and a wide pulse pressure.
14. In frail older patients the presentation may be **falls, confusion, or functional decline** rather than classical syncope.

INDICATIONS FOR PERMANENT PACING

15. **Pacing should be undertaken in acquired complete heart block not attributable to reversible or physiologic causes, regardless of symptoms** — **Class I recommendation**.
16. **Complete heart block due to neuromuscular disease with conduction disorders** (myotonic dystrophy type 1, Kearns-Sayre syndrome) — **Class I**.
17. **Symptomatic complete heart block arising from clinically necessary guideline-directed therapy** (for example a beta blocker that cannot be stopped) — **Class I**.
18. **Infiltrative cardiomyopathy such as cardiac sarcoidosis with Mobitz II block** — **Class IIa**.

CHOICE OF PACING MODE

19. **DDD pacing is recommended over AAI or VVI** in those requiring permanent pacing for AV block (**Class I**), because it preserves AV synchrony.
20. **Single-chamber ventricular (VVI) pacing is effective** where frequent ventricular pacing is not expected, or where **significant comorbidities are likely to determine outcome** and limit the benefit of dual-chamber pacing (**Class I**).
21. **Active patients over 70 benefit from DDD pacing in terms of quality of life**; **UKPACE** (n=2021, mean age 80) found **no difference in all-cause mortality, AF or heart failure** between single- and dual-chamber pacing for AV block.
22. **Physiological pacing (CRT or His bundle pacing) should be considered** when **LVEF <50%** and **>40% ventricular pacing** is expected (**Class IIa**). **His bundle pacing may be considered** for AV nodal-level block (**Class IIb**).
23. **BLOCK-HF** showed CRT reduced the combined endpoint of urgent heart failure admission, death, or ≥15% increase in LV volume index, with about a **30% reduction in heart failure hospitalisation**, though no mortality benefit.
24. **In permanent or persistent AF where rhythm control is not planned, an atrial lead should not be implanted**

(Class III).

COMPLICATIONS AND GERIATRIC CONSIDERATIONS

25. Complications: **pneumothorax** (1–2% subclavian, <0.1% cephalic/axillary), bleeding, **myocardial perforation and tamponade (<0.2%)**, lead dislodgement, infection (<1%), and late device erosion or lead fracture. Early complications occur in about **7% within two weeks**; late complications in up to **10%**.
26. **Increasing age was not associated with a higher risk of complications** (FOLLOWPACE) — female sex and low BMI were.
27. In cachectic patients with thin skin or skeletal deformity from osteoarthritis or osteoporosis, the device may need to be placed **submuscularly** to avoid erosion.
28. **All patients should undergo transthoracic echocardiography before implantation** to identify pre-existing LV dysfunction and guide mode selection.

Current Practice Update

Textbook position (Pathy's 6th ed.)	Current evidence (2023–2026)
Right ventricular apical pacing is the default	**Conduction system pacing — particularly left bundle branch area pacing (LBBAP)** — has largely superseded His bundle pacing and is increasingly the preferred physiological alternative
His bundle pacing as the physiological option	**His bundle pacing is limited** by low success rate, high thresholds, sensing problems and inability to correct distal block; **LBBAP overcomes most of these**
CRT for those with LVEF <50% and high pacing burden	**2025 ESC/EHRA consensus on conduction system pacing** still **favours biventricular pacing over CSP for standard CRT indications**, but supports CSP for bradycardia pacing with expected high pacing burden
Pacing-induced cardiomyopathy managed by upgrade to CRT	Increasing use of **primary LBBAP to prevent** pacing-induced cardiomyopathy in patients who will be pacing-dependent
Transvenous systems only	**Leadless pacemakers** are now an option in selected patients with limited venous access or high infection risk

Latest Updates (2023–Present)

- The **2025 ESC/EHRA consensus document on conduction system pacing** provides the first dedicated guidance on **His bundle pacing and left bundle branch area pacing**, and **favours biventricular pacing over CSP for traditional CRT indications** while endorsing CSP for bradycardia indications with an anticipated high ventricular pacing burden (*EP Europace* 2025).
- **LBBAP produces a significantly narrower paced QRS** than biventricular pacing (about **129 ms versus 157 ms**), with **lower pacing thresholds and shorter procedure and fluoroscopy times**, and comparable improvement in LVEF and NYHA class.
- **Pacing-induced cardiomyopathy** is now a well-recognised complication of chronic right ventricular apical pacing, and is a major driver of the shift toward conduction system pacing in patients with complete heart block and preserved LV function (*Circulation* 2024–2025).
- **Leadless LBBAP** using left endocardial pacing systems has been demonstrated in early case series (*HeartRhythm Case Reports* 2025), pointing toward completely lead-free physiological pacing.
- Meta-analyses through 2025–2026 continue to show **CSP benefit is clearest in patients with LBBB morphology** and less consistent in non-LBBB intraventricular conduction delay.
- In older patients, the decision remains individualised — **VVI pacing is still entirely appropriate** where comorbidity dominates prognosis, and current guidelines explicitly preserve this option.

Examiner's Tip — Mark-Earning Lines

- "**Complete heart block = AV dissociation with an independent escape rhythm**, ventricular escape at **30–40 bpm with wide QRS**."
- "**The lower the escape focus, the less reliable the conduction tissue.**"
- "**Pace all acquired complete heart block not due to reversible or physiologic causes, regardless of symptoms — Class I.**"
- "**DDD is preferred over VVI (Class I)**, but **VVI is effective where comorbidities determine outcome**."
- "**UKPACE found no mortality difference** between single- and dual-chamber pacing in the elderly with AV block."
- "**Consider CRT or His bundle pacing when LVEF <50% and >40% pacing is expected (Class IIa).**"

- “**Increasing age is not a risk factor for pacemaker complications** — female sex and low BMI are.”
- “**Left bundle branch area pacing** has now largely superseded His bundle pacing as the practical form of conduction system pacing.”

Q10. Heart Failure — Etiopathology, Clinical Presentation, Stages and Management

15–20 High-Yield Points

DEFINITION AND EPIDEMIOLOGY

1. **Heart failure (HF)** is a clinical syndrome in which structural or functional cardiac abnormality causes **reduced cardiac output and/or elevated intracardiac pressures**, producing typical symptoms and signs.
2. Prevalence rises steeply with age — HF is present in **13–14% of people over 80 years**.
3. Nearly **one-third of HF patients have chronic kidney disease**, and **more than 50% of HF patients have atrial fibrillation** (Framingham data).

ETIOPATHOLOGY

4. **Causes**: hypertensive heart disease, coronary artery disease, cardiomyopathies, valvular heart disease, arrhythmias, pericardial disease, myocarditis, drugs (alcohol, NSAIDs, chemotherapy, biologics), nutritional causes (**thiamine deficiency, anaemia**), and primary right heart failure from pulmonary disease.
5. **Hypertension increases the risk of HF two- to threefold** and is the commonest underlying cause in older people; treating it significantly reduces incident HF.
6. **Pathophysiology** rests on **neurohormonal activation** — sympathetic nervous system and renin-angiotensin-aldosterone system activation initially compensate but ultimately drive **adverse remodelling, fibrosis and apoptosis**.
7. Contributing age-related changes: **increased ventricular and arterial stiffness, impaired diastolic relaxation, reduced beta-adrenergic responsiveness, and mitochondrial dysfunction**.
8. **Precipitants** of decompensation: acute coronary events, **atrial fibrillation with rapid ventricular rate**, medication non-adherence, infection, excess fluid or sodium intake, anaemia, renal impairment, thyroid disease, and drugs such as NSAIDs and glucocorticoids.

CLASSIFICATION AND STAGES

9. **Left- versus right-sided**, **low-output versus high-output**, and **systolic versus diastolic** heart failure.
10. **NYHA functional classification**: **I** — no limitation; **II** — slight limitation, symptoms on ordinary activity; **III** — marked limitation, symptoms on less-than-ordinary activity; **IV** — symptoms at rest or on any activity.
11. **ESC classification by ejection fraction**: **HFrEF <40%**, **HFmrEF 41–49%**, **HFpEF ≥50%**. HFpEF and HFmrEF additionally require **elevated natriuretic peptides plus structural abnormality or diastolic dysfunction**.
12. **ACC/AHA stages** describe progression: **Stage A** at risk without structural disease; **Stage B** structural disease without symptoms; **Stage C** structural disease with current or prior symptoms; **Stage D** refractory HF requiring advanced interventions.

CLINICAL PRESENTATION

13. Symptoms — exertional dyspnoea, orthopnoea, paroxysmal nocturnal dyspnoea, fatigue, reduced exercise tolerance, ankle swelling.
14. Signs — raised JVP, displaced apex, third heart sound, basal crackles, hepatomegaly, ascites, pleural effusion, peripheral oedema.
15. **One-third of older people with HF display none of these signs**, and comorbidities confuse the picture; individual symptoms and signs have **low sensitivity**, but their **combination** supports the diagnosis.
16. In older people presentation is often atypical — **confusion, falls, functional decline, or unexplained weight loss**.

DIAGNOSIS

17. The **ESC algorithm** combines symptoms, signs, prevalent heart conditions and an abnormal ECG; the presence of any of these prompts **natriuretic peptide measurement**.
18. **BNP >35 pg/ml or NT-proBNP >125 pg/ml** should trigger echocardiography. **Normal levels make HF unlikely.** Levels are equally predictive in HFrEF and HFpEF and carry prognostic value.
19. **Echocardiography** is the key confirmatory test; **chest X-ray** helps exclude COPD and pulmonary fibrosis.

MANAGEMENT — NON-PHARMACOLOGICAL

20. **Smoking cessation** (RR of HF is 1.75 in current smokers), **alcohol counselling**, **sodium restriction to about 1500 mg/day**, fluid restriction where there is dilutional hyponatraemia, patient education, medication review, vaccination, multidisciplinary input, and structured follow-up.
21. **Exercise training reduces mortality** (HR 0.65) and counteracts the deconditioning spiral; it should be offered to older HF patients.

MANAGEMENT — PHARMACOLOGICAL (HFREF)

22. **All patients** should receive an **ACE inhibitor or ARB regardless of NYHA class**, plus **optimal diuretic therapy to maintain euvolaemia**.
23. **Symptomatic patients** should also receive a **beta blocker** (carvedilol, bisoprolol, nebivolol, or modified-release metoprolol — benefit demonstrated up to age 80) and a **mineralocorticoid receptor antagonist** (spironolactone 12.5–50 mg reduced morbidity and mortality by **30%** in NYHA III–IV; eplerenone benefits mild symptoms).
24. **ARNI (sacubitril/valsartan)** reduced cardiovascular death and HF admission (HR 0.80) versus enalapril; **do not combine with an ACE inhibitor** and **wait 36 hours after the last ACE inhibitor dose**.
25. **Ivabradine** is added if the patient remains symptomatic in **sinus rhythm with heart rate >70/min** (HR 0.82 for CV death and HF hospitalisation).
26. **Digoxin** reduces hospitalisation but not mortality (DIG trial); in those over 70 use **0.125 mg daily**, targeting a plasma level of **0.5–0.9 ng/ml**, with monitoring of potassium and renal function.
27. **Hydralazine-isosorbide dinitrate** is used in self-declared Black patients and as an alternative when ACE inhibitors/ARBs are contraindicated.
28. **"Start low and go slow"** is the governing principle in older people, with monitoring of BP, creatinine and potassium after each dose change.

DEVICE THERAPY AND PROGNOSIS

29. **CRT** if QRS >130 ms; **ICD** for secondary prevention after cardiac arrest or ventricular arrhythmia, and for primary prevention in NYHA II–III HFrEF with ischaemic or dilated cardiomyopathy and expected survival >12 months. Mortality benefit is seen across all age groups including >75, though the effect attenuates with age.
30. **HFpEF trials have been largely negative** — CHARM-Preserved, I-PRESERVE, PEP-CHF, SENIORS, DIG-ancillary, TOPCAT and PARAGON-HF showed **no mortality benefit**, with some reduction in hospitalisation.
31. **Prognosis remains poor** despite therapy; **multidisciplinary team involvement and advance care planning, including palliative care, are of paramount importance**.

Current Practice Update

Textbook position (Pathy's 6th ed.)	Current evidence (2023–2026)
Sequential therapy: ACE inhibitor/ARB → beta blocker → MRA → consider ARNI	**Four pillars started early and together**: **ARNI/ACEi, beta blocker, MRA, and SGLT2 inhibitor**, with rapid up-titration
SGLT2 inhibitors not included	**SGLT2 inhibitors are recommended across the entire ejection fraction spectrum** (HFrEF, HFmrEF, HFpEF)
HFpEF has no disease-modifying therapy (all trials negative)	**SGLT2 inhibitors and finerenone now have proven benefit in HFpEF/HFmrEF**
Slow, cautious up-titration after discharge	**STRONG-HF "high-intensity care"** — rapid up-titration with frequent review after acute HF improves outcomes
Spironolactone/eplerenone as the only MRAs	**Finerenone**, a non-steroidal MRA, is FDA-approved (2025) for **HF with LVEF ≥40%**
ESC classification: HFmrEF = "mid-range"	Now termed **"mildly reduced"** ejection fraction

Latest Updates (2023–Present)

- The **2023 ESC Focused Update** of the HF guidelines recommends **SGLT2 inhibitors across the whole LVEF spectrum**, and recommends **SGLT2 inhibitors and finerenone for prevention of HF in diabetic chronic kidney disease**.
- **FINEARTS-HF** (*NEJM* 2024; presented ESC 2024) enrolled **6,001 patients with LVEF ≥40%** (mean age **72 years**, 48% with eGFR <60 ml/min) and showed **finerenone reduced worsening HF events and cardiovascular death**. The FDA approved finerenone for **HF with LVEF ≥40% in 2025**.
- Benefit of finerenone was **independent of NYHA class, KDIGO risk category, atrial fibrillation, diabetes status, and concomitant SGLT2 inhibitor use** (*Circulation* 2025; *JACC* 2025).
- An **individual patient-level meta-analysis of MRAs in heart failure** (*Lancet* 2024) confirmed benefit across the ejection fraction spectrum, with greater effect in HFrEF.
- **STRONG-HF** established that **rapid up-titration of guideline-directed medical therapy with close follow-up ("high-intensity care") after an acute HF episode improves outcomes** — a strategy that must be applied thoughtfully in frail older adults.
- **Intravenous iron** (ferric carboxymaltose/derisomaltose) improves symptoms, functional capacity and quality of life in HF with iron deficiency, and reduces HF hospitalisation.
- The first **guideline addressing cardiovascular-kidney-metabolic (CKM) syndrome** was published in 2026, formalising the integrated management of the HF–CKD–diabetes–obesity cluster that dominates geriatric practice.

Examiner’s Tip — Mark-Earning Lines

- “**HFrEF <40%, HFmrEF 41–49%, HFpEF ≥50%** — the latter two need **raised natriuretic peptides plus structural or diastolic abnormality**.”
- “**One-third of older people with heart failure display none of the classical signs.**”
- “**BNP >35 pg/ml or NT-proBNP >125 pg/ml** prompts echocardiography; **normal levels make HF unlikely**.”
- “**Spironolactone reduced morbidity and mortality by 30% in NYHA III–IV**; **ARNI gave HR 0.80** versus enalapril.”
- “**Digoxin 0.125 mg daily in those over 70, target level 0.5–0.9 ng/ml.**”
- “**Start low and go slow**, monitoring BP, creatinine and potassium after each dose change.”
- “**Four pillars — ARNI/ACEi, beta blocker, MRA, SGLT2 inhibitor — started early and together.**”
- “**FINEARTS-HF established finerenone for HF with LVEF ≥40%**, with FDA approval in 2025.”

Q11. Management of Systolic Heart Failure in an Elderly Patient with CKD (GFR 40 ml/min)

15–20 High-Yield Points

FRAMING THE PROBLEM

1. This is **HFrEF with stage 3b chronic kidney disease** — the classic **cardiorenal** situation in geriatric practice.
2. **CKD is present in nearly one-third of heart failure patients**; renal function declines with age and creatinine clearance is often below 60 ml/min in older HF patients.
3. Renal impairment in HF arises from **reduced renal perfusion, renal vasoconstriction, renal venous congestion**, and **over-diuresis**; older people are also **less able to excrete excess sodium and water**.
4. Importantly, **a GFR of 40 ml/min does not contraindicate disease-modifying therapy** — it mandates lower starting doses, slower titration, and closer monitoring.

ASSESSMENT FIRST

5. Confirm the diagnosis with **echocardiography** and **natriuretic peptides**, remembering that **NT-proBNP is elevated by renal impairment itself** and must be interpreted accordingly.
6. Perform a **comprehensive geriatric assessment** — frailty, cognition, functional status, polypharmacy, nutrition, and patient preference.
7. **Exclude reversible contributors**: renal artery stenosis, obstructive uropathy, NSAIDs, hypovolaemia, and contrast exposure.
8. Establish **baseline creatinine, eGFR, potassium and blood pressure** before starting or changing therapy.

DISEASE-MODIFYING THERAPY

9. **ACE inhibitor or ARB** — start at low dose (for example **enalapril 2.5 mg twice daily**) and up-titrate slowly. Even **low doses provide benefit** if the maximum dose cannot be reached.
10. Accept a **rise in creatinine of up to 30% and a fall in eGFR** after initiation — this is haemodynamic, not nephrotoxic, and is not a reason to stop. Beyond 30%, halve the dose and investigate.
11. **Do not combine an ACE inhibitor with an ARB** — the combination failed to reduce mortality and carries significant risk of **renal impairment, hyperkalaemia and hypotension**.
12. **Beta blocker** — carvedilol, bisoprolol, nebivolol, or modified-release metoprolol. Beta blockers are **not renally limited** and are often the best-tolerated pillar in CKD. **Start low, go slow.**
13. **Mineralocorticoid receptor antagonist** — spironolactone or eplerenone. Use with **great caution at GFR 40**: start **12.5 mg daily**, check **potassium and creatinine at 1 week, 4 weeks, then periodically**. Withhold if **potassium >5.0 mEq/l**.
14. **ARNI (sacubitril/valsartan)** — may replace the ACE inhibitor/ARB; note **hyperkalaemia, hypotension and angioedema** as adverse effects, and observe the **36-hour washout** from any ACE inhibitor.
15. **Diuretics** — loop diuretics (furosemide, bumetanide, torsemide) are the mainstay for congestion. **Higher doses are needed in CKD** because tubular delivery of drug is reduced. **Diuretics are less effective in the elderly and increase the risk of electrolyte abnormality.**
16. In diuretic resistance, use **intravenous loop diuretic** or **sequential nephron blockade** by adding a thiazide (metolazone) — but this greatly increases the risk of hypokalaemia, hyponatraemia and AKI, and requires close inpatient monitoring.
17. **Ivabradine** if still symptomatic in sinus rhythm with heart rate >70/min — it has **no renal dose adjustment**, making it useful here.
18. **Digoxin** — reduce to **0.125 mg daily or alternate days**; **renal impairment markedly increases toxicity risk**. Monitor potassium and levels (target 0.5–0.9 ng/ml).
19. **Hydralazine-isosorbide dinitrate** is a useful alternative when RAAS blockade cannot be tolerated because of hyperkalaemia or worsening renal function.

COMORBIDITY MANAGEMENT

20. Treat **anaemia** — common in this group from CKD, poor haematinic intake, and antiplatelet/anticoagulant use. **Intravenous iron is safe and effective** and improves symptoms, functional capacity and quality of life.
21. Manage **atrial fibrillation** with **beta blockers** (which reduce mortality in AF with HF) and/or digoxin; **rhythm control has not proven superior to rate control**.
22. **Avoid NSAIDs absolutely** — they worsen heart failure, cause AKI, and interfere with ACE inhibitors.
23. **Restrict sodium to about 1500 mg/day** and restrict fluid where there is dilutional hyponatraemia. Advise **daily weights**.

24. Review the **entire drug list** at every visit; polypharmacy and drug–drug interactions are the dominant iatrogenic hazards in this population.

MONITORING AND ESCALATION

25. Check **renal function, electrolytes and blood pressure after every dose change** and periodically thereafter. **Falls and postural hypotension** are the key clinical toxicities to look for.
26. **Device therapy** — CRT if QRS >130 ms; ICD only if expected survival exceeds 12 months, weighing comorbidity, frailty, and the ethical issue of eventual deactivation.
27. **Multidisciplinary follow-up with nephrology input** and, where trajectory is poor, early **advance care planning and palliative care**.

Current Practice Update

Textbook position (Pathy's 6th ed.)	Current evidence (2023–2026)
Triple therapy (ACEi/ARB + beta blocker + MRA), ARNI as an alternative	**Four pillars including an SGLT2 inhibitor**, started early and together
SGLT2 inhibitors not mentioned	**SGLT2 inhibitors (dapagliflozin, empagliflozin) are now a cornerstone** — they slow CKD progression *and* reduce HF events, and can be started down to **eGFR 20–25 ml/min/1.73 m²**
Renal impairment as a reason for caution with RAAS blockade	The **initial eGFR dip with SGLT2 inhibitors and RAAS blockade is expected and haemodynamic**, and does not warrant discontinuation
Spironolactone or eplerenone as the MRA options	**Finerenone** offers cardiorenal benefit with **lower rates of hyperkalaemia and AKI** than spironolactone in real-world comparisons
Slow, cautious up-titration	**STRONG-HF high-intensity care** — faster up-titration with close monitoring improves outcomes

Latest Updates (2023–Present)

- The **2023 ESC Focused Update** recommends **SGLT2 inhibitors across the full ejection fraction spectrum**, and specifically for **prevention of heart failure in diabetic chronic kidney disease**, alongside finerenone.
- **DAPA-CKD and EMPA-KIDNEY** established renal protection with SGLT2 inhibitors independent of diabetes, and **DAPA-HF and EMPEROR-Reduced** established HF benefit — making the SGLT2 inhibitor the single most useful addition for the HFrEF-plus-CKD patient.
- **FINEARTS-HF** subgroup analyses showed finerenone's benefit was **consistent across KDIGO risk categories** and in patients already receiving an SGLT2 inhibitor, supporting **complementary mechanisms** (*Circulation* 2025; *JACC* 2025).
- The **CONFIDENCE trial** (*NEJM* 2025) showed **finerenone plus empagliflozin reduced albuminuria more than either agent alone** in CKD with type 2 diabetes, supporting genuine combination cardiorenal therapy.
- Real-world comparative data show **finerenone associated with lower rates of hospitalisation, mortality, AKI and hyperkalaemia than spironolactone** — relevant where hyperkalaemia is the rate-limiting problem.
- **Potassium binders (patiromer, sodium zirconium cyclosilicate)** now allow RAAS blockade and MRA therapy to be **maintained rather than withdrawn** in patients with recurrent hyperkalaemia.
- The **2026 CKM syndrome guideline** provides the first integrated framework for managing the overlapping cardiovascular, kidney and metabolic disease burden typical of this patient.

Examiner's Tip — Mark-Earning Lines

- "**CKD is present in nearly one-third of heart failure patients**; renal impairment arises from **reduced perfusion, venous congestion and over-diuresis**."
- "**A GFR of 40 ml/min does not contraindicate disease-modifying therapy** — it mandates lower doses and closer monitoring."
- "**Accept a creatinine rise of up to 30%** after starting RAAS blockade — it is haemodynamic, not nephrotoxic."
- "**Never combine an ACE inhibitor with an ARB** — no mortality benefit, significant renal and hyperkalaemia risk."
- "**Start spironolactone at 12.5 mg; withhold if potassium >5.0 mEq/l**; recheck at 1 and 4 weeks."
- "**Higher loop diuretic doses are needed in CKD** because tubular drug delivery is reduced."
- "**Digoxin 0.125 mg daily or alternate days**; renal impairment markedly increases toxicity."
- "**Avoid NSAIDs absolutely.**"
- "**SGLT2 inhibitors are now the single most valuable addition** — they treat both the heart failure and the CKD, and

can be started down to eGFR 20–25 ml/min/1.73 m^2.”

Q12. Statins in the Elderly

15–20 High-Yield Points

MECHANISM AND EFFECT

1. **Statins are HMG-CoA reductase inhibitors** — they block the rate-limiting step of hepatic cholesterol synthesis, upregulate LDL receptors, and lower LDL-C by **20–55%**.
2. **Pleiotropic effects** matter as much as lipid lowering: statins **stabilise vulnerable atherosclerotic plaque** by reducing lipid content, minimising inflammation, and improving endothelial function.
3. **Three out of four acute myocardial infarcts** are thought to result from rupture or erosion of such vulnerable plaque.
4. **High-intensity statin therapy reduces LDL-C by 50–55%**; moderate intensity by about 30–49%.

SECONDARY PREVENTION — STRONG EVIDENCE

5. A meta-analysis of **28 randomised controlled trials** showed **significant benefit of statins for secondary prevention in all age groups, including patients over 75** (Cholesterol Treatment Trialists' Collaboration, *Lancet* 2019).
6. **Relative risk reduction of major vascular events per 1 mmol/l LDL-C reduction in those over 75 was 26%** — at least as good as in younger patients.
7. **Key trials in the elderly**: **PROSPER** (age 70–82, pravastatin 40 mg, RR 0.85 for cardiovascular events); **HPS** (up to age 80, simvastatin 40 mg, RR 0.76 for major vascular events, RR 0.87 for all-cause death); **LIPS** (fluvastatin 80 mg after first PCI, RR 0.78 for MACE).
8. **ACC/AHA guidance** — in patients over 75 with clinical ASCVD it is **reasonable to initiate moderate- or high-intensity statin therapy** after considering risk reduction, adverse effects, drug interactions, frailty and patient preference.
9. **Moderate-intensity statin may be preferable** in older adults because of higher adverse event rates, lower adherence, and higher discontinuation with high-intensity therapy.
10. **Secondary prevention treatment thresholds**: ACC/AHA and Canadian guidelines use **LDL-C >70 mg/dl** or **non-HDL-C >100 mg/dl**; ESC/EAS set an "ideal" target of **LDL-C <55 mg/dl** and **non-HDL-C <85 mg/dl**.

PRIMARY PREVENTION — WEAK AND CONFLICTING EVIDENCE

11. **Clinical trial data demonstrating statin benefit for primary prevention are not available for adults older than 75.**
12. In the primary prevention subgroup of **PROSPER (56% of participants), pravastatin showed no significant reduction** in coronary death, non-fatal MI, or stroke over 3.2 years.
13. **ALLHAT-LLT post hoc analysis** found **no benefit of pravastatin for primary prevention in those aged 75 and over**, with a **non-significant trend toward increased all-cause mortality**.
14. In a well-characterised cohort aged 75 and over, **LDL-C ≥130 mg/dl was not associated with increased 5-year ASCVD event rates**.
15. In the meta-analysis of statins by age, **there was no significant benefit among patients older than 70 for primary prevention**, with a trend toward smaller proportional risk reduction with increasing age.
16. **ACC/AHA states it "may be reasonable"** to start moderate-intensity statin in adults ≥75 with LDL-C between **70 and 189 mg/dl** — but equally, **it may be reasonable not to**. **ESC/EAS** says it "may be considered" if high risk, such as diabetes.
17. **EWTOPIA 75** (open-label, ezetimibe, entry LDL-C ≥140 mg/dl) is the main primary-prevention trial in this age group and carries considerable weight in meta-analyses.
18. Retrospective data show statins for primary prevention in those aged **75–84 with type 2 diabetes reduced cardiovascular events by 24% and all-cause mortality by 16%**.

ADVERSE EFFECTS AND PRACTICAL ISSUES

19. Adverse effects: **muscle aches, rhabdomyolysis, deranged liver function**, possible increase in type 2 diabetes, and concerns about cognitive dysfunction.
20. **Adherence is poor** — only about **one in four older people is still taking a statin two years after prescription**, and about **20% show "primary non-adherence"**, never starting at all.
21. **Time to benefit for statins in primary prevention is at least two years** — so **life expectancy, lag time to benefit, and the therapeutic horizon** must be weighed against competing mortality and polypharmacy.
22. The decision in adults 75 or older should be made through a **tailored, shared decision-making approach** balancing benefit, harm, and patient values.

Current Practice Update

Textbook position (Pathy's 6th ed.)	Current evidence (2023–2026)
Secondary prevention strongly supported; primary prevention uncertain over 75	**Unchanged** — the evidence gap persists; **STAREE** was designed to fill it and its results are awaited
Statin monotherapy is the mainstay	**Early combination therapy (statin + ezetimibe)** is increasingly favoured to reach targets faster and reduce statin dose-related adverse effects
Deprescribing not addressed	**Structured deprescribing** in advanced frailty and limited life expectancy is now an accepted and recommended practice
Age-based thresholds	Guidelines have shifted decisively toward **individualised, shared decision-making rather than age cut-offs**

Latest Updates (2023–Present)

- **STAREE (Statins in Reducing Events in the Elderly)** is the definitive trial for this question — **9,971 community-dwelling Australians aged ≥70 (mean age 74.7 years, 40% aged ≥75, 52% women, mean LDL-C 126 mg/dl)** randomised to **atorvastatin 40 mg or placebo**, with **co-primary endpoints of disability-free survival and major cardiovascular events** (*JAHA* 2024). Follow-up concluded in December 2025 and **results are awaited**; nested substudies (**STAREE-Mind** for brain MRI, **STAREE-HEART** for cardiac ageing) will report alongside.
- **PREVENTABLE** is the parallel US trial of atorvastatin 40 mg in adults ≥75, with dementia and disability endpoints — together with STAREE it will settle the primary prevention question.
- The **2026 ADA Standards of Care** state that it **"may be reasonable" to initiate moderate-intensity statin therapy in those aged over 75 with diabetes** not already on a statin, reflecting continued equipoise.
- A large **target trial emulation study** (*PLOS Medicine* 2026) in older adults with type 2 diabetes supported cardiovascular benefit of statins for primary prevention in this subgroup, aligning with guidelines that favour **individualised, shared decision-making over age-based thresholds**.
- **SCOPE-70** is comparing **low- versus moderate-intensity rosuvastatin** for primary prevention in those aged ≥70 — addressing the practical question of dose rather than whether to treat.
- Contemporary consensus increasingly frames the question as **"time to benefit versus life expectancy"** rather than "old versus young", and endorses **deprescribing statins in advanced frailty, terminal illness, or life expectancy under one year**.

Examiner's Tip — Mark-Earning Lines

- "Statins **stabilise vulnerable plaque** — three out of four MIs arise from plaque rupture or erosion."
- "**Secondary prevention benefit is proven in all age groups including over 75** — RRR of **26% per 1 mmol/l LDL-C reduction**."
- "**Primary prevention trial data are not available for adults older than 75**; PROSPER's primary prevention arm and ALLHAT-LLT were both negative."
- "**PROSPER RR 0.85; HPS RR 0.76 for major vascular events.**"
- "**Moderate-intensity statin is often preferable in older adults** because of adverse effects and poor adherence."
- "**Only one in four older patients is still on a statin at two years.**"
- "**Time to benefit in primary prevention is at least two years** — weigh this against life expectancy and lag time."
- "**STAREE and PREVENTABLE** are the trials that will settle the primary prevention question; STAREE results are awaited."

Q13. Ezetimibe — Indications, Mode of Action, Metabolism, Excretion, Side Effects and Dosage

> *Note: Pathy's covers ezetimibe's place in lipid management and the key trial evidence, but not its detailed pharmacokinetics. The pharmacology below is supplemented from standard pharmacological and regulatory sources.*

15–20 High-Yield Points

MODE OF ACTION

1. Ezetimibe is a **selective inhibitor of intestinal cholesterol absorption** — it is **not** a statin and does not inhibit HMG-CoA reductase.
2. It acts on the **Niemann-Pick C1-Like 1 (NPC1L1) transporter** on the brush border of jejunal enterocytes, blocking uptake of both **dietary and biliary cholesterol** and plant sterols.
3. Reduced cholesterol delivery to the liver **depletes hepatic cholesterol stores**, which **upregulates LDL receptors** and increases clearance of LDL from the blood.
4. It **does not affect absorption of triglycerides, fatty acids, bile acids, or fat-soluble vitamins A, D, E and K**.
5. This **complementary mechanism** to statins — blocking absorption while the statin blocks synthesis — is the basis of combination therapy.

EFFECT ON LIPIDS

6. **Monotherapy lowers LDL-C by about 18–20%**, with minimal effect on HDL-C and triglycerides.
7. **Added to a statin, ezetimibe produces a further LDL-C reduction of about 20%** — equivalent to roughly three doublings of the statin dose.
8. This makes it the **preferred first add-on** when a statin alone does not achieve target, or when high-intensity statin is not tolerated.

INDICATIONS

9. **Adjunct to statin therapy** in primary hypercholesterolaemia when LDL-C targets are not reached on maximally tolerated statin.
10. **Monotherapy** in patients who are **statin-intolerant** or in whom statins are contraindicated.
11. **Secondary prevention of ASCVD** — ACC/AHA guidance: in patients on maximal statin with **LDL-C ≥70 mg/dl**, adding ezetimibe **is reasonable** in very-high-risk ASCVD (Class IIa) and **may be reasonable** in others (Class IIb).
12. **Before adding a PCSK9 inhibitor** — guidelines specify that **ezetimibe should be added to maximal statin therapy before a PCSK9 inhibitor is considered (Class I)**.
13. **Homozygous familial hypercholesterolaemia**, as an adjunct to statin ± LDL apheresis.
14. **Homozygous sitosterolaemia (phytosterolaemia)** — a specific licensed indication, because ezetimibe also blocks plant sterol absorption.
15. **Mixed dyslipidaemia**, in combination with fenofibrate.

METABOLISM AND EXCRETION

16. **Absorption**: rapidly absorbed after oral dosing; can be taken **with or without food**. Time to peak concentration is about **4–12 hours** for the active metabolite.
17. **Metabolism**: undergoes extensive **glucuronidation in the small intestinal wall and liver** by **UGT enzymes**, forming **ezetimibe-glucuronide**, which is **pharmacologically active** and accounts for most of the drug effect. **Cytochrome P450 is not significantly involved**, which is why ezetimibe has **few drug interactions** — a real advantage in polypharmacy.
18. **Enterohepatic recirculation** of the glucuronide delivers drug repeatedly to its site of action and gives a **long half-life of about 22 hours**.
19. **Excretion**: approximately **78% in faeces** (mainly as unchanged ezetimibe) and **about 11% in urine** (mainly as the glucuronide).
20. **No dose adjustment is required** for mild hepatic impairment, renal impairment, or in the elderly. It should be **avoided in moderate to severe hepatic impairment** (Child-Pugh ≥7), where drug exposure rises several-fold.

DOSAGE

21. **10 mg once daily**, orally, with or without food, at any time of day. **There is no dose titration** — 10 mg is the only dose.
22. **Fixed-dose combinations** are widely available (ezetimibe with simvastatin, atorvastatin, or rosuvastatin) and improve adherence.

23. If given with a **bile acid sequestrant**, administer ezetimibe **at least 2 hours before or 4 hours after** the sequestrant, because binding resins reduce its absorption.

SIDE EFFECTS AND CAUTIONS

24. Generally **very well tolerated**. Common: **diarrhoea, abdominal pain, flatulence, fatigue, arthralgia, upper respiratory tract infection**.
25. **Myalgia and myopathy** occur, and the **risk of rhabdomyolysis increases when combined with a statin** — though the incremental risk is small.
26. **Elevated transaminases**, particularly in combination with a statin; check liver function before starting combination therapy.
27. Rare: **hypersensitivity reactions including angioedema and rash**, **hepatitis**, **cholelithiasis and cholecystitis**, **pancreatitis**, and **thrombocytopenia**.
28. **Interactions**: **ciclosporin** raises ezetimibe levels (and ezetimibe raises ciclosporin levels) — monitor levels; **fibrates** increase biliary cholesterol and the risk of gallstones; **cholestyramine** reduces efficacy.

KEY OUTCOME EVIDENCE

29. **IMPROVE-IT** — in post-ACS patients on **simvastatin 40 mg**, adding **ezetimibe 10 mg** gave a further **22% decrease in LDL-C**, a **6.4% relative and 2% absolute reduction** in the primary composite endpoint (cardiovascular death, MI, unstable angina, coronary revascularisation, stroke) over six years. This was the **first trial to prove that a non-statin LDL-lowering drug reduces events**, validating the "LDL hypothesis".
30. **EWTOPIA 75** — an open-label trial of **ezetimibe monotherapy in Japanese patients aged 75 and over** with LDL-C ≥140 mg/dl, showing reduction in cardiovascular events. It carries considerable weight in meta-analyses of lipid lowering in the very old.
31. **PRECISE-IVUS** — ezetimibe plus atorvastatin produced greater **coronary plaque regression** on intravascular ultrasound than atorvastatin alone.

Current Practice Update

Textbook position (Pathy's 6th ed.)	Current evidence (2023–2026)
Ezetimibe added when statin alone fails to reach target	**Upfront combination (statin + ezetimibe) from the outset** is increasingly recommended, especially in high-risk ACS patients
Statin dose maximised first, then ezetimibe added	Evidence favours **moderate-intensity statin plus ezetimibe over high-intensity statin monotherapy** — similar LDL-C reduction with **fewer muscle-related adverse effects and better adherence**
Ezetimibe as the sole non-statin add-on	Now one of several: **bempedoic acid, PCSK9 inhibitors, and inclisiran** have all entered practice
Limited data in the very old	**EWTOPIA 75** provides direct randomised evidence in patients aged ≥75

Latest Updates (2023–Present)

- **Upfront combination lipid-lowering therapy** (moderate-intensity statin plus ezetimibe from the start) is now favoured in high-risk patients over sequential dose escalation, on evidence that it achieves targets faster with **fewer statin-associated muscle symptoms** — highly relevant to older adults, in whom myalgia is a major cause of discontinuation.
- The **2023 ESC ACS Guideline** endorses early, intensive lipid lowering after ACS, with **ezetimibe added early** rather than after a prolonged period of statin monotherapy.
- **Bempedoic acid** (an ATP-citrate lyase inhibitor, acting upstream of HMG-CoA reductase and **not activated in skeletal muscle**) is now a further option for statin-intolerant patients, with cardiovascular outcome benefit demonstrated in **CLEAR Outcomes** (*NEJM* 2023). It is often combined with ezetimibe.
- **Inclisiran**, a twice-yearly siRNA targeting PCSK9, offers a **6-monthly dosing schedule** that is attractive for adherence in older adults with polypharmacy.
- Ezetimibe's position as a **mandatory step before PCSK9 inhibitor initiation** is retained in current ACC/AHA guidance, largely on cost-effectiveness grounds.
- **Generic ezetimibe** is inexpensive and widely available in India, making it the most cost-effective non-statin LDL-lowering agent for routine practice.

Examiner's Tip — Mark-Earning Lines

- "Ezetimibe **inhibits the NPC1L1 transporter** on jejunal enterocytes, blocking absorption of dietary and biliary

cholesterol."

- "It **does not affect absorption of triglycerides, fatty acids, bile acids, or fat-soluble vitamins**."
- "**Monotherapy lowers LDL-C by ~18–20%; added to a statin, a further ~20%** — equivalent to three doublings of the statin dose."
- "**Glucuronidated in the intestinal wall and liver; the glucuronide is the active metabolite. CYP450 is not involved**, hence few interactions."
- "**Enterohepatic recirculation gives a half-life of about 22 hours; 78% faecal and 11% urinary excretion.**"
- "**Dose is 10 mg once daily — there is no titration.** No adjustment needed for renal impairment or old age; avoid in moderate-severe hepatic impairment."
- "**IMPROVE-IT was the first trial to prove a non-statin LDL-lowering drug reduces cardiovascular events** — 22% further LDL-C reduction, 6.4% relative risk reduction."
- "**EWTOPIA 75** provides randomised evidence for ezetimibe in patients aged 75 and over."

Q14. Low Molecular Weight Heparins — Types, Indications, Benefits and Side-Effect Profile

15–20 High-Yield Points

NATURE AND MECHANISM

1. **Low molecular weight heparins (LMWHs)** are produced by **chemical or enzymatic depolymerisation of unfractionated heparin (UFH)**, giving fragments of mean molecular weight **4,000–6,000 daltons** (versus 15,000 for UFH).
2. Like UFH, LMWH acts by **potentiating antithrombin**. However, the shorter chains cannot bridge antithrombin to thrombin.
3. Consequently **LMWH has a much greater effect on factor Xa than on thrombin (IIa)** — the **anti-Xa : anti-IIa ratio is about 3:1 to 4:1**, compared with **1:1 for UFH**.
4. Because of this, **LMWH cannot be monitored by the APTT** — it requires an **anti-factor Xa assay** where monitoring is needed.

TYPES

5. **Enoxaparin** — the most widely used; anti-Xa:anti-IIa ratio ~3.8:1.
6. **Dalteparin** — ratio ~2.7:1; the preferred agent in cancer-associated thrombosis in many protocols.
7. **Tinzaparin** — ratio ~1.9:1; the **largest mean molecular weight**, and therefore **least dependent on renal clearance**, which makes it useful in renal impairment.
8. **Nadroparin, reviparin, parnaparin, bemiparin** are other agents in use.
9. **Fondaparinux** is a **synthetic pentasaccharide** — a pure **indirect factor Xa inhibitor** with **no anti-IIa activity**. It is not strictly an LMWH but is used in the same clinical settings and is the agent of choice in **heparin-induced thrombocytopenia**.

ADVANTAGES OVER UNFRACTIONATED HEPARIN

10. **Longer half-life** (about 4 hours versus 1 hour), permitting **once- or twice-daily** dosing.
11. **Increased bioavailability** after subcutaneous injection (>90% versus about 30% for UFH).
12. **More predictable pharmacokinetics**, because LMWH binds far less to plasma proteins, endothelium and macrophages.
13. Therefore it can be given by **subcutaneous injection without routine monitoring, even at treatment doses**.
14. UFH's binding to multiple plasma proteins explains its **unpredictable kinetics, especially in the elderly**, where variability in these determinants is greater.
15. **UFH carries a higher bleeding risk in older adults** — patients over 72 treated with UFH for VTE had bleeding in **14.1% versus 7.1%**, and major bleeding in **11.1% versus 3.1%**, compared with younger patients.
16. Standard, non-weight-adjusted heparin doses in older patients produce **higher heparin levels and a tendency to higher APTT**.
17. **Unless there is an absolute contraindication, LMWH has generally replaced UFH and should be preferred** because of its predictable pharmacokinetics, efficacy and safety.
18. **Lower incidence of heparin-induced thrombocytopenia and of heparin-induced osteoporosis** than UFH.
19. **LMWH has a survival advantage in patients with malignancy and thrombosis** and has traditionally been the anticoagulant of choice in this group.

INDICATIONS

20. **Prophylaxis of venous thromboembolism** in medical and surgical inpatients, and after major orthopaedic surgery.
21. **Treatment of established DVT and pulmonary embolism**.
22. **Acute coronary syndromes** — unstable angina and NSTEMI, and as adjunct to fibrinolysis in STEMI.
23. **Bridging anticoagulation** around surgery or procedures in patients on warfarin.
24. **Cancer-associated thrombosis**, **pregnancy-associated VTE**, and **extracorporeal circuits** (haemodialysis).
25. **LMWH and UFH remain the preferred anticoagulants where surgery or clinical instability is involved** — cancer, sepsis, inability to take oral medication, or malabsorption.

SIDE EFFECTS AND CAUTIONS

26. **Bleeding** is the principal adverse effect.
27. **Renal clearance** — LMWHs are **predominantly eliminated by renal excretion**, so kidney impairment leads to **prolonged half-life and accumulation**. Dose should be reduced or the dosing interval extended; **current data are insufficient to establish definitive LMWH dose adjustment in older patients with renal impairment**.

28. **Heparin-induced thrombocytopenia (HIT)** — immune-mediated, causing **both thrombocytopenia and paradoxical arterial and venous thrombosis**, with mortality around **30% if unrecognised**. It is **less common with LMWH than UFH but can occur with either**. Treatment is **immediate withdrawal of all heparin** and substitution with **argatroban** (acute) or **fondaparinux** (subsequent treatment/prophylaxis). **Warfarin can worsen acute HIT** and must be withheld or reversed with vitamin K.
29. Other effects: **hyperkalaemia** (aldosterone suppression), **elevated transaminases**, **injection-site haematoma and skin necrosis**, **osteoporosis with prolonged use**, and **spinal or epidural haematoma** with neuraxial anaesthesia.
30. **Reversal**: **protamine sulfate neutralises only about 60% of LMWH's anti-Xa activity** (it fully reverses UFH). In overdose, LMWH will prolong the APTT, and bleeding may require protamine.

Current Practice Update

Textbook position (Pathy's 6th ed.)	Current evidence (2023–2026)
LMWH is the anticoagulant of choice in cancer-associated thrombosis	**DOACs (apixaban, edoxaban, rivaroxaban) are now first-line for most cancer-associated VTE**, with LMWH retained for gastrointestinal/genitourinary malignancy and high bleeding risk
LMWH used as a bridge to warfarin for acute VTE	**DOACs are first-line for acute VTE** without cancer or severe renal insufficiency; LMWH is now a bridge only for dabigatran and edoxaban
No specific reversal agent	**Andexanet alfa** is available for factor Xa inhibitor reversal (though not licensed for LMWH); protamine remains partial for LMWH
Anti-Xa monitoring reserved for special cases	Anti-Xa monitoring increasingly recommended in **obesity, severe renal impairment, pregnancy and the very elderly**

Latest Updates (2023–Present)

- **DOACs have displaced LMWH as first-line therapy for most acute VTE**, including cancer-associated thrombosis, on the basis of equivalent efficacy with greater convenience — though **LMWH is still preferred in luminal gastrointestinal and genitourinary malignancy**, where DOACs increase bleeding.
- LMWH retains an essential role in **acute coronary syndromes, peri-procedural bridging, pregnancy, severe renal or hepatic impairment where DOACs are unsuitable, and patients unable to take oral medication**.
- Comparative reviews through 2025 (systematic review of enoxaparin versus dalteparin in arthroplasty) found **no consistent difference in efficacy or safety between individual LMWHs**, so choice is driven largely by **cost, availability and dosing convenience** — an important practical point in Indian practice.
- **Anti-Xa monitoring** is increasingly recommended in older patients with **eGFR <30 ml/min**, extremes of body weight, and pregnancy, where the usual assumption of predictable kinetics breaks down.
- **Fondaparinux and argatroban** remain the standard alternatives in confirmed or suspected HIT; **DOACs are being used increasingly for HIT** in the sub-acute phase.
- Emerging **factor XI/XIa inhibitors** (abelacimab, milvexian, osocimab) aim to provide "haemostasis-sparing anticoagulation"; **osocimab was superior to enoxaparin** for DVT prevention after knee replacement in the FOXTROT study, though phase 3 orthopaedic trials have not started.

Examiner's Tip — Mark-Earning Lines

- "LMWH acts through **antithrombin** but has a **greater effect on factor Xa than on thrombin**, ratio about **3–4:1 versus 1:1 for UFH**."
- "**LMWH cannot be monitored by APTT — it requires an anti-factor Xa assay.**"
- "Advantages: **longer half-life, greater bioavailability, more predictable pharmacokinetics**, allowing once-daily subcutaneous dosing without monitoring."
- "**UFH bleeding rates in the elderly: 14.1% versus 7.1%; major bleeding 11.1% versus 3.1%.**"
- "**Unless absolutely contraindicated, LMWH has replaced UFH and should be preferred.**"
- "**LMWH is predominantly renally excreted** — reduce dose or extend interval in renal impairment; **tinzaparin is least renally dependent**."
- "**HIT causes both thrombocytopenia and thrombosis, mortality ~30% if unrecognised.** Stop all heparin; use **argatroban acutely, fondaparinux subsequently**; **warfarin can worsen acute HIT**."
- "**Protamine reverses only about 60% of LMWH anti-Xa activity.**"

Q15. Role of Newer Anticoagulants

15–20 High-Yield Points

THE CLASSES

1. **Direct oral anticoagulants (DOACs)** comprise a **direct thrombin (factor IIa) inhibitor — dabigatran** — and **direct factor Xa inhibitors — apixaban, rivaroxaban, and edoxaban**.
2. They act **directly on their target enzyme**, unlike warfarin (which depletes vitamin K–dependent factors II, VII, IX, X) or heparin (which acts indirectly via antithrombin).

ADVANTAGES OVER WARFARIN

3. **No routine coagulation monitoring** and no INR testing.
4. **Rapid onset and offset of action**, so no bridging is required.
5. **Predictable dose response**, with far fewer **food and drug interactions** — warfarin is affected by vitamin K–rich foods, alcohol, antibiotics, amiodarone, and many other drugs.
6. **Wider therapeutic window** and no need for the frequent dose adjustment that ageing, illness, and renal decline force upon warfarin therapy.
7. Warfarin dose requirements **fall with age** — for every 10-year increase in age there is a **15% increase in the risk of an INR ≥5**.
8. A **meta-analysis of 12 studies (77,011 patients)** showed **significantly more effective stroke prevention with a 50% reduction in intracranial bleeding** with DOACs compared with warfarin.
9. **DOACs have therefore become the oral anticoagulant of first choice for the majority of patients.**

INDICATIONS

10. **Stroke prevention in non-valvular atrial fibrillation** — Class I recommendation, preferred over warfarin.
11. **Treatment of acute DVT and pulmonary embolism**, and **prevention of recurrence** — often without hospital admission.
12. **Prevention of VTE after elective hip or knee replacement**, including extended out-of-hospital prophylaxis.
13. **Secondary prophylaxis of VTE** at reduced dose.
14. **DOACs are the first choice of anticoagulant for acute VTE in patients without cancer or severe renal insufficiency**, mainly because of the lower intracranial bleeding risk versus VKAs.

DOSING IN VTE

15. **Dabigatran 150 mg twice daily** — requires **at least 5 days of initial LMWH/UFH/fondaparinux**.
16. **Rivaroxaban 15 mg twice daily for 21 days, then 20 mg once daily** — no lead-in parenteral therapy. Extended treatment: consider **10 mg once daily**.
17. **Apixaban 10 mg twice daily for 7 days, then 5 mg twice daily** — no lead-in. Extended treatment: consider **2.5 mg twice daily**.
18. **Edoxaban 60 mg once daily** after **at least 5 days** of parenteral anticoagulation; reduce to **30 mg once daily** if **creatinine clearance 30–50 ml/min, body weight ≤60 kg, or on strong P-glycoprotein inhibitors**.

EVIDENCE IN THE ELDERLY

19. In patients **>75, DOACs as a class were superior to warfarin in both efficacy and safety**. **Apixaban had the lowest risk of major bleeding**, and **apixaban, edoxaban and dabigatran had lower rates of intracranial haemorrhage** than rivaroxaban or warfarin.
20. Patients aged **≥75 treated with DOACs for VTE had a significantly lower risk of major bleeding than those on VKAs (relative risk 0.49)**.
21. **ARISTOPHANES** and the **PREFER in AF** registries confirmed the benefit in the very old, showing lower major bleeding with DOACs even in patients with **low body mass index or aged ≥85**.
22. **Rivaroxaban and edoxaban are once-daily** agents, which may aid adherence. **Dabigatran is not suitable for dosette boxes**, an important practical point in older people using pill organisers.

LIMITATIONS AND CONTRAINDICATIONS

23. **DOACs are only equivalent to an INR of 2–3**, so **warfarin remains mandatory** where a higher target is needed: **mechanical prosthetic heart valves**, **moderate-severe mitral stenosis**, recurrent thrombosis despite anticoagulation, and some patients with **antiphospholipid syndrome**.
24. **All DOACs are renally excreted to some extent** and undergo some hepatic metabolism — dose reduction is guided by **age, weight, renal function and concomitant drugs**.
25. In **low body weight** patients there is a risk of over-anticoagulation.

26. Because the **half-life is short**, **missed doses create periods of under-anticoagulation and increased stroke risk** — adherence matters more, not less, than with warfarin.

REVERSAL

27. **Idarucizumab** is a specific monoclonal antibody fragment reversing dabigatran. **Andexanet alfa** is a modified recombinant factor Xa decoy reversing apixaban and rivaroxaban.
28. **Prothrombin complex concentrate (PCC)** is used where specific agents are unavailable — the usual situation in much of India.

Current Practice Update

Textbook position (Pathy's 6th ed.)	Current evidence (2023–2026)
DOACs not first choice for cancer-associated thrombosis	**DOACs are now first-line for most cancer-associated VTE**; LMWH retained for gastrointestinal/genitourinary tumours
DOAC dosing based on labelled criteria	**Inappropriate dose reduction ("under-dosing") in the elderly is now recognised as a major and harmful problem** — it increases stroke without reducing bleeding
Four DOACs represent the newest class	**Factor XI/XIa inhibitors** are the true emerging class, promising "haemostasis-sparing anticoagulation"
LAA occlusion only when anticoagulation contraindicated	LAA occlusion has a **stronger recommendation** and is being tested as an alternative in broader populations

Latest Updates (2023–Present)

- **Factor XI/XIa inhibitors** are the major development. The rationale is **dissociating haemostasis from thrombosis** — factor XI contributes to pathological thrombus formation but is largely dispensable for normal haemostasis.
- **AZALEA-TIMI 71** (*NEJM* 2023): **abelacimab**, a monoclonal antibody given **monthly by subcutaneous injection**, reduced **major or clinically relevant non-major bleeding by 62–69%** versus rivaroxaban in AF, and the trial was **stopped early** for this overwhelming safety benefit. However, ischaemic strokes were **numerically more frequent** with abelacimab, and the trial was **not powered for efficacy**.
- **OCEANIC-AF**, the phase 3 trial of the oral FXIa inhibitor **asundexian** versus apixaban, was **stopped early for inferiority in efficacy** — a critical cautionary result showing that superior safety does not guarantee adequate efficacy.
- **LILAC-TIMI 76** is randomising about **1,900 AF patients deemed unsuitable for anticoagulation** (aged 65–74 with CHA2DS2-VASc ≥4, or ≥75 with CHA2DS2-VASc ≥3) to **abelacimab versus placebo**, with completion expected in late 2026. **LIBREXIA-AF** is testing **milvexian versus apixaban in 15,000 patients**, reporting from 2026.
- **OCEANIC-STROKE** (asundexian versus placebo added to antiplatelet therapy after non-cardioembolic stroke) and **LIBREXIA-STROKE / LIBREXIA-ACS** are testing factor XI inhibition as a **safer platform for dual pathway inhibition**.
- These agents **remain investigational**; no factor XI inhibitor is yet approved, and their eventual clinical role will be determined by the phase 3 results.
- For geriatric practice the immediate implication is different: the priority remains **correct DOAC dosing** and **avoiding both under-treatment and inappropriate dose reduction**, which the SAGE-AF study showed is common and associated with geriatric conditions.

Examiner's Tip — Mark-Earning Lines

- "**Dabigatran is a direct thrombin inhibitor; apixaban, rivaroxaban and edoxaban are direct factor Xa inhibitors.**"
- "**DOACs gave a 50% reduction in intracranial bleeding** versus warfarin in a meta-analysis of 77,011 patients."
- "**In patients over 75, DOACs are superior to warfarin in both efficacy and safety**; **apixaban has the lowest major bleeding risk**."
- "**DOACs are equivalent only to an INR of 2–3**, so **warfarin is mandatory for mechanical valves and moderate-severe mitral stenosis**."
- "**Edoxaban 60 mg is reduced to 30 mg if CrCl 30–50 ml/min, weight ≤60 kg, or on a strong P-glycoprotein inhibitor.**"
- "**Short half-life means missed doses cause under-anticoagulation** — adherence matters more, not less."
- "**Idarucizumab reverses dabigatran; andexanet alfa reverses apixaban and rivaroxaban.**"
- "**Factor XI inhibitors promise haemostasis-sparing anticoagulation** — AZALEA-TIMI 71 showed 62–69% less bleeding, but **OCEANIC-AF was stopped for inferiority**, so efficacy remains unproven."

Q16. Evidence-Based VTE Prophylaxis Protocol After Elective Total Hip / Knee Replacement

15–20 High-Yield Points

RATIONALE

1. **Major lower limb arthroplasty is among the highest-risk settings for venous thromboembolism**, because it combines all three elements of **Virchow's triad** — venous stasis from immobilisation, endothelial injury from surgical trauma, and a post-operative hypercoagulable state.
2. **Venous thrombosis is primarily a disease of old age**, and hip, knee and pelvic surgery are the classic predisposing factors, with obesity and malignancy as accessory factors.
3. In up to **50% of cases of venous thrombosis an additional underlying genetic predisposition becomes manifest** under these circumstances.
4. Without prophylaxis, DVT rates after arthroplasty historically reached **40–60%**; **thromboprophylaxis of surgical and medical patients at medium and high risk is now firmly established practice**.
5. Risk is **highest in the first 2 weeks** but remains elevated for up to **3 months**, which is why extended prophylaxis matters.

RISK ASSESSMENT

6. **Assess every patient pre-operatively** for VTE risk (prior VTE, cancer, thrombophilia, obesity, immobility, hormone therapy) and **bleeding risk** (active bleeding, thrombocytopenia, severe renal or hepatic impairment, recent intracranial haemorrhage).
7. **Older age, frailty, and renal impairment** shift the balance and demand dose adjustment rather than omission.

MECHANICAL PROPHYLAXIS

8. **Intermittent pneumatic compression devices (IPC)** should be applied from admission and continued while the patient is in bed.
9. **IPC alone** is used when the bleeding risk is unacceptable or the patient refuses pharmacological prophylaxis.
10. **Graduated compression stockings** may be added, but are contraindicated in peripheral arterial disease and severe neuropathy.
11. **Early mobilisation and adequate hydration** are essential adjuncts and are the cheapest interventions available.

PHARMACOLOGICAL OPTIONS

12. All of the following are supported as prophylactic agents after arthroplasty: **LMWH; fondaparinux; dabigatran, apixaban or rivaroxaban (for THA/TKA but not hip fracture surgery); low-dose unfractionated heparin; adjusted-dose vitamin K antagonist; or aspirin.**
13. **LMWH (enoxaparin 40 mg once daily subcutaneously)** remains the reference standard against which all other agents are compared.
14. **DOACs are used at reduced thromboprophylactic doses** and have been used specifically for **extended out-of-hospital thromboprophylaxis in high-risk orthopaedic surgery such as hip and knee replacement**.
15. **Fondaparinux 2.5 mg once daily** is an alternative; avoid if creatinine clearance is below 30 ml/min.
16. **Aspirin** is an option, but see the current evidence below — it is **not equivalent to LMWH**.

DURATION

17. **Treatment should be for at least 10 to 14 days and ideally up to 35 days.**
18. In practice: **up to 35 days after total hip replacement** and about **14 days after total knee replacement**, reflecting the different risk profiles of the two operations.

TIMING AND NEURAXIAL ANAESTHESIA

19. Start LMWH **12 hours before or 12 hours after surgery**, or at least **6–8 hours post-operatively** once haemostasis is secure.
20. With **neuraxial (spinal or epidural) anaesthesia**, observe strict intervals to avoid **spinal haematoma**: at least **12 hours after a prophylactic LMWH dose** before needle placement or catheter removal, and at least **4 hours after catheter removal** before the next dose.
21. The current standard of care in hip fracture is to **stabilise comorbid conditions, repair the hip (ideally under spinal anaesthesia), and encourage early ambulation with aggressive rehabilitation**, with prophylactic anticoagulation throughout.

GERIATRIC MODIFICATIONS

22. **Reduce the dose or extend the dosing interval** of LMWH in renal impairment; **tinzaparin** is least renally

dependent.

23. DOAC prophylactic doses require **renal-function-based adjustment**, and DOACs are **not recommended for hip fracture surgery** (as opposed to elective arthroplasty).
24. **Do not routinely screen asymptomatic patients with ultrasound** before discharge — it does not improve outcomes.
25. **Educate the patient and family** about the symptoms of DVT and PE before discharge, and ensure a clear plan for who supervises injections at home.

Current Practice Update

Textbook position (Essentials of Clinical Geriatrics / ACCP 2012)	Current evidence (2023–2026)
Aspirin listed as an equivalent option (Grade 1B) alongside LMWH and DOACs	**CRISTAL trial showed aspirin is inferior to enoxaparin** — symptomatic VTE **3.27% versus 1.76%** at 90 days
Choice between agents largely a matter of preference	Evidence now supports **anticoagulant prophylaxis over aspirin** for most patients, with aspirin reserved for genuinely low-risk patients or where anticoagulation is unsuitable
Duration “at least 10–14 days, ideally up to 35 days”	**Unchanged** — remains the standard, with 35 days for THA and about 14 days for TKA
Individual LMWHs assumed interchangeable	**Confirmed** — no consistent efficacy or safety difference between enoxaparin and dalteparin; choice driven by cost and availability

Latest Updates (2023–Present)

- The **CRISTAL trial** (*JAMA* 2022, secondary analysis *PLOS One* 2024) — a cluster-randomised, crossover, registry-nested trial across **31 Australian hospitals** — compared **aspirin 100 mg daily** with **enoxaparin 40 mg daily**, given for **35 days after hip and 14 days after knee arthroplasty**. In the full cohort of **11,728 patients**, symptomatic VTE within 90 days occurred in **3.27% with aspirin versus 1.76% with enoxaparin** (treatment difference 1.85%, p = 0.004), **favouring enoxaparin**.
- **Joint-related reoperation within 90 days was also lower with enoxaparin** (2.26% versus 2.47%, p = 0.03), and there was **no difference in major bleeding** — undermining the main argument for aspirin.
- This result directly challenges the widespread practice, reported in a 2021 surgeon survey, of **95% of practitioners using aspirin plus mechanical prophylaxis** as their default strategy.
- A **2026 systematic review and meta-analysis** of 8 studies and **117,367 patients** (*Frontiers in Medicine* 2026) again examined aspirin versus enoxaparin after THA, TKA and hip fracture surgery, confirming the continuing clinical importance of this question.
- Patients with a **history of prior VTE** are a group in whom aspirin appears clearly inadequate and anticoagulant prophylaxis should be used (*Journal of Arthroplasty* 2025).
- A **2025 systematic review comparing enoxaparin with dalteparin** in arthroplasty found **limited direct comparative evidence and no consistent difference**, supporting selection on the basis of **cost, availability and dosing convenience**.
- **Factor XI inhibitors** are being explored in this setting — **osocimab was superior to enoxaparin** for DVT prevention after knee replacement in the phase 2 **FOXTROT** study, and **milvexian** was studied in **AXIOMATIC-TKR**; phase 3 orthopaedic trials have not yet begun.

Examiner’s Tip — Mark-Earning Lines

- “Arthroplasty combines all three elements of **Virchow’s triad**.”
- “**Assess VTE risk and bleeding risk in every patient pre-operatively.**”
- “Options: **LMWH, fondaparinux, dabigatran/apixaban/rivaroxaban (for arthroplasty, not hip fracture), low-dose UFH, adjusted-dose VKA, aspirin, or IPC if bleeding risk is high.**”
- “**Duration at least 10–14 days, ideally up to 35 days — 35 days for hip, about 14 days for knee.**”
- “**Neuraxial anaesthesia: 12 hours after a prophylactic LMWH dose before needle placement or catheter removal; 4 hours after removal before the next dose.**”
- “**Enoxaparin 40 mg once daily is the reference standard.**”
- “**The CRISTAL trial showed aspirin is inferior to enoxaparin — symptomatic VTE 3.27% versus 1.76% at 90 days, with no difference in major bleeding.**”
- “**Do not routinely screen asymptomatic patients with ultrasound before discharge.**”

End of document.

MD Geriatric Medicine — High-Yield Examination Answers

Set 1: Assessment, Function, Cognition, Capacity and Geriatric Syndromes

Primary source: Pathy's Principles and Practice of Geriatric Medicine, 6th edition; supported by Kane's Essentials of Clinical Geriatrics and current international and Indian guidelines.

QUESTION 1

Comprehensive Geriatric Assessment — What It Is and Its Usefulness in Clinical Practice

A. 20 High-Yield Points

Definition and Concept

1. **Comprehensive geriatric assessment (CGA)**, also called **multidimensional geriatric assessment**, is a **diagnostic process, usually interdisciplinary**, that determines an older person's **medical, psychosocial, functional and environmental** resources and problems.
2. Its stated objective is to **develop an overall plan for treatment and long-term follow-up**. An assessment **without a linked care plan is NOT a CGA** — a favourite examination point.
3. It **differs from a standard medical evaluation** in three ways: it targets older people with complex problems, it **emphasises functional status and quality of life**, and it uses **interdisciplinary teams with quantitative scales**.
4. A purely psychosocial or environmental home visit **without a medical evaluation** also does not qualify as CGA, because multidimensionality is incomplete.
5. **Historical origin: Dr Marjory Warren**, London infirmary, **late 1930s**. Through evaluation, mobilisation and rehabilitation she got long-bedfast patients out of bed and home, and advocated **assessment and rehabilitation before any long-term institutionalisation**.

Rationale — Why in the Elderly? (five textbook reasons)

6. **Multi-morbidity and complexity** — systematic assessment helps set priorities among multiple conditions.
7. **Unrecognised problems** — many problems are never reported, often because they are **falsely thought to be non-modifiable effects of ageing**.
8. **Chronic conditions** — a diagnosis without information on **functional relevance** is of limited therapeutic value.
9. **Social and environmental interaction** — home layout and social network determine whether a person can stay at home.
10. **Functional status** — the main goal of geriatric medicine is to **prevent or delay functional decline**; benefit is greatest when intervention occurs **early** in the path from robustness to frailty to disability.

Components — What Is Assessed

11. CGA begins with a **functional status "review of systems"** built on three measures: **frailty, basic ADLs, and IADLs**.
12. **Principle of hierarchy / parsimony:** if a patient is **severely ADL-dependent, do not assess frailty**; if the patient is **robust or pre-frail, do not assess basic ADLs**.
13. Frailty is measured by two models — the **phenotype model** (Fried phenotype, Frailty Trait Scale, FRAIL index) and the **cumulative deficit model** (Frailty Index, Clinical Frailty Scale).
14. **Dimensions and tools (Table 96.1):** BADL — **Katz, Barthel, Lawton PSMS**; IADL — **Lawton, OARS**; Social — **Lubben Social Network Scale**; Affective — **Geriatric Depression Scale, Zung**; Cognitive — **MMSE (Folstein), Kahn MSQ**; Gait and balance — **Tinetti, Get-Up-and-Go**; Nutrition — **MNA**; Senses — **whispered voice test, Snellen**; Oral — **GOHAI**.
15. Two screens are **mandatory** in any older person with functional disability: **cognitive impairment and depression**.

Process in Office and Clinic Practice

16. **Step 1 — set priorities:** work first on the problem that **most bothers the patient**, or the problem on which resolution of others depends (**alcoholism, depression**).
17. **Step 2 — task and symptom analysis:** define the exact nature of the impairment and correlate it with **new drugs or dose changes (including over-the-counter), change in living conditions, and intercurrent acute illness**. Then examination, laboratory tests, therapist and social-work input, and a **long-term follow-up / case-management plan**.

Settings and Team

18. **Settings:** home (preventive visits), office and outpatient clinic, **day hospital**, **acute care for the elderly (ACE) unit**, post-acute rehabilitation unit, nursing home (**Resident Assessment Instrument**), and hospital-at-home programmes. Inpatient and day-hospital settings give intensity and speed for the frail; outpatient settings are cheaper.
19. **Team (minimum):** physician, nurse, social worker, physiotherapist, occupational therapist; **optimally** dietician, pharmacist, ethicist, home-care specialist. The **interdisciplinary team conference is critical** — present in most successful trials; teams mature through **forming, storming, norming, performing**.

Evidence of Usefulness — The Exam Core

20. **Proven benefits:** CGA **improves function and survival while reducing healthcare utilisation and costs**. Supporting data:

- **Improved diagnostic accuracy — 1 to more than 4 new diagnoses per patient**.
- **1993 Stuck/Rubenstein meta-analysis (28 trials, ~10,000 patients):** mortality down **22%** (hospital-based, 12 months) and **14%** (all programmes); living at home up **47%** (hospital-based) and **26%** (all); readmissions down **12%**; cognitive improvement up **47%**; physical function up **72%** on hospital units.
- **2011 Cochrane meta-analysis:** inpatient CGA increases the likelihood of being **alive and in one's own home**, with potential cost reduction.
- **Better prescribing** — CGA reduces unnecessary and inappropriate medication use.
- **Targeting matters:** best results when patients are at **high risk of deterioration yet retain rehabilitation potential** — exclude those **"too well or too ill."**
- **Programmes with hands-on care and/or long-term follow-up** outperform purely consultative programmes.
- **Weak areas:** outpatient CGA showed **no mortality benefit** in one meta-analysis; **preventive home visits are not recommended routinely** (benefit only with CGA-based extended follow-up in relatively well elders; **number needed to visit about 40** to prevent one nursing-home admission).
- **Oncology (SIOG task force):** CGA detects many problems missed by regular assessment and is associated with **fewer treatment side effects**.

B. Latest Updates (2023–Present)

1. German DGG S3 Guideline on CGA in Hospital (2024) — the first S3-level CGA guideline. Twelve evidence-based and eight consensus-based recommendations. Strongest evidence in **acute geriatric, oncology and orthopaedic/trauma wards**; weaker in emergency departments and general surgery. Core elements: **minimum CGA duration of 15 minutes** and **six core dimensions — self-help ability, mobility, cognitive function/delirium, affect, nutrition and social situation**; the **G8 questionnaire** is recommended for screening mainly in oncology.

2. Italian National CGA Guideline (2024) — 25 scientific societies plus the National Institute of Health, GRADE methodology. Sixteen recommendations from **117 studies** across primary care, clinics, emergency departments, hospital wards and nursing homes. CGA **reduces hospitalisation, mortality, institutionalisation and delirium risk**, improves prescribing appropriateness, and **maintains functional activities**. The **Multidimensional Prognostic Index (MPI)** is specifically recommended to predict negative outcomes.

3. Japanese CGA-Based Healthcare Guidelines 2024. The main objective of CGA is stated to be **personalised care through holistic management plans and targeted interventions — not merely status evaluation or prognostication**.

4. ASCO Geriatric Oncology Guideline Update (2023). Geriatric assessment should be used in **all patients over 65 with cancer**; all older adults with GA-identified deficits should receive **GA-guided management**, and the **Practical Geriatric Assessment (PGA)** is the recommended tool. Evidence base: **GAP70+** and **GAIN** trials.

5. ASCO Global (Resource-Stratified) Geriatric Assessment Guideline (2025) — adapts the 2023 guideline for **resource-limited settings** using ADAPTE methodology. Directly relevant to Indian practice.

6. Cochrane 2022 — Community CGA. Twenty-one studies, 7,893 participants, 10 countries, evaluating domiciliary CGA and CGA in general practice, with outcomes of death, nursing-home admission, unplanned hospital admission and emergency visits.

7. Meta-analysis of CGA in acute hospitalisation (2025) — CGA **may improve short-term survival and reduce readmission rates** after acute hospitalisation.

8. Perioperative CGA is now mainstream. The **2023 World Society of Emergency Surgery guidelines** advocate frailty assessment and multidisciplinary geriatric-surgical management in older trauma patients; the UK **NELA Best Practice Tariff (April 2023)** mandates perioperative team involvement in frailer laparotomy patients. CGA outperforms traditional preoperative risk models in predicting **postoperative delirium, infection and prolonged stay**.

9. Indian context. Evidence is predominantly from high-income countries and hospitalised patients; research on CGA in primary care and community settings in low- and middle-income countries is lacking. Under **NPHCE**, community services are delivered by **ASHA workers** with clinics at PHCs, but implementation is inconsistent. Short culturally adapted tools such as **EHSAS** are being validated for rural India because full CGA is impractical with the available workforce.

C. Current Practice Update

Aspect	Pathy's 6th ed. (2022)	Current position (2023–2026)
Standardisation	Deliberately broad; "no single optimal blueprint"	**Minimum standards defined** — German S3: at least 15 minutes, six mandatory core domains
Prognostic tool	Many scales listed; none preferred	**MPI specifically recommended** (Italian guideline)
Oncology tool	SIOG could not recommend any specific tool	**PGA specifically recommended** (ASCO 2023)
Oncology strength	CGA "possibly" improves survival	**GA mandatory at 65 and above**; GA-guided management proven to cut severe chemotoxicity
Outpatient CGA	"Less impressive"; no mortality benefit	Italian guideline **supports** primary-care CGA for reducing hospitalisation and institutionalisation
Preventive home visits	Cannot be recommended as routine practice	**Largely unchanged** — Cochrane 2022 still shows uncertain benefit
Perioperative CGA	Not covered	Now **embedded in surgical pathways** (NELA BPT, WSES 2023, POPS model)
Emergency department	Not addressed	Recognised as important but **evidence still weak** (German S3)

D. Exam One-Liners

- **CGA without a care plan and follow-up is not a CGA.**
- **Marjory Warren, 1930s London** — mother of CGA and of British geriatrics.
- **Function is the common language of geriatrics; assessment is its core method.**
- **Hierarchy rule:** ADL-dependent, skip frailty testing; robust or pre-frail, skip basic ADL testing.
- **Targeting rule:** benefit is greatest in those **"not too well, not too ill"** — high risk with rehabilitation potential.
- **Hands-on care plus long-term follow-up** separates effective from ineffective programmes.
- **1993 meta-analysis:** mortality down 22%, living at home up 47%, physical function up 72% on hospital units.
- **Two mandatory screens** in functional disability: **cognition and depression**.
- **German S3 six core domains:** self-care, mobility, cognition/delirium, affect, nutrition, social situation.
- **ASCO 2023:** every cancer patient 65 and above gets a geriatric assessment, using the **PGA**.
- **Italian 2024:** use the **MPI** for prognosis.
- **India:** CGA delivery constrained by workforce — hence short screening tools within **NPHCE**.

QUESTION 2

Tools of Functional Assessment

A. 20 High-Yield Points

Framework

1. **Functional assessment** measures **what a person actually does in daily life**, not merely what diseases they have. Tools exist to make this measurement **standardised, reproducible and comparable across settings**.
2. **Purpose of using standardised tools:** they allow **information exchange between acute, rehabilitation and community settings**, permit **measurement of effectiveness**, foster **comparison of treatments**, and **minimise repeated collection of identical information** by different team members.
3. **Three levels of measurement (ICF, 2001): impairment** (body function or structure), **activity limitation** (difficulty executing a task), and **participation restriction** (problems in involvement in life situations). Different tools address different levels.
4. **Three functional core measures** in CGA: **frailty, basic ADLs, and IADLs**. The **hierarchy rule** determines which to use — severely ADL-dependent patients are not tested for frailty; robust patients are not tested for basic ADLs.
5. **Two methods of data capture: report** (by the patient or a proxy — tells you what they do under real living conditions) and **performance testing** (tells you what they can do under standard conditions). **Discrepancies between the two are common and informative.**

Basic ADL Tools

6. **Katz Index (1963)** — six items: **bathing, dressing, toileting, transferring, continence, feeding**. Graded independent/dependent, letters A to G. The classic **hierarchy of ADL loss**.
7. **Barthel Index (1965; modified 1988)** — ten items: feeding, bathing, grooming, dressing, bowels, bladder, toilet use, transfer, mobility, stairs. Scored **0–100** (or 0–20). It is an **observation of what the patient does**, and is the **RCP/BGS-endorsed ADL scale**, but has a **ceiling effect in ambulatory patients**.
8. **Lawton Physical Self-Maintenance Scale (PSMS, 1969)** — six items: **toileting, feeding, dressing, grooming, physical ambulation, bathing**. Each graded on **five levels**, dichotomised to a total of **0–6** (6 = independent). Published in the same paper as the Lawton IADL scale.

IADL Tools

9. **Lawton IADL scale (1969)** — eight items: **telephone, shopping, food preparation, housekeeping, laundry, transport, medications, finances**. IADLs need "instruments," and are the **first functions to be lost**.
10. **OARS Multidimensional Functional Assessment Questionnaire** — assesses IADLs alongside social and economic resources; widely used in surveys.
11. **Clinical rule:** if difficulty is reported, always ask **"how does she manage?"** — is the task neglected, or is someone else doing it?

Mobility, Gait and Balance Tools (high-yield numbers)

12. **Gait speed** over a 4-metre track: **below 0.6 m/s is abnormal**; **0.6–0.8 m/s is high-risk**. Each **0.1 m/s fall in gait speed equals a 7% rise in fall risk**.
13. **Timed Get-Up-and-Go (Mathias, Nayak and Isaacs, 1986): more than 12 seconds indicates high fall risk** (sensitivity 77%, positive predictive value 93% in the community).
14. **Tinetti Performance-Oriented Mobility Assessment (1986):** 14 tasks, **score below 24 out of 28 indicates fall risk**; takes 10–15 minutes.
15. **Short Physical Performance Battery (SPPB):** combines gait speed, **30-second chair stand** and the **4-stage balance test** (each position held **10 seconds**). **Berg Balance Scale:** 14 items, 0–56 (0–20 wheelchair-bound, 21–40 walks with assistance, 41–56 independent).

Composite, Global and Domain-Specific Tools

16. **Functional Independence Measure (FIM)** — scores **18 functions** across self-care, sphincter control, mobility, locomotion, communication and social cognition; the standard rehabilitation outcome measure.
17. **RCP/BGS endorsed scales (Table 98.3):** ADL — **Barthel Index**; vision, hearing and communication — **Lambeth Disability Screening Questionnaire**; memory — **Abbreviated Mental Test (10 items)**; depression — **Geriatric Depression Scale (30-item, 15-item short form)**; morale — **Philadelphia Geriatric Center Morale Scale**.
18. **Other domain tools (Table 96.1):** nutrition — **Mini Nutritional Assessment (MNA)**, SNAQ; special senses — **whispered voice test, Hearing Handicap Inventory, Snellen chart**; oral health — **GOHAI**; social network — **Lubben Social Network Scale**; cognition — **MMSE**; mood — **GDS, Zung**.
19. **Frailty tools: Fried phenotype** (weight loss, exhaustion, weakness, slow gait, low activity); **FRAIL scale** (Fatigue,

Resistance, Aerobic, Illnesses more than 5, Loss of weight 5%) — answerable in 15–30 seconds; **SOF index** (weight loss, cannot rise from chair 5 times without arms, reduced energy); **Clinical Frailty Scale** (9 points, seconds to apply); **Frailty Index** (30 or more deficits, continuous 0–1).

20. **Screening questions usable at the bedside:** "*Do you need help at home?*" (function) · "*Do you have trouble walking or lose your balance? Have you had a fall? Are you afraid of falling?*" (mobility) · "*Are you sad?*" (mood) · "*Have you lost weight?*" (nutrition) · "*Are you on seven or more medicines?*" (polypharmacy) · "*Do you wet yourself?*" (continence).

B. Master Comparison Table

Domain	Screening tool	Assessment tool	Key threshold
Frailty	FRAIL scale, Clinical Frailty Scale	Fried phenotype, Frailty Index	3 or more of 5 = frail
Basic ADL	—	**Katz, Barthel, Lawton PSMS**	Barthel below 60 = dependent
IADL	—	**Lawton IADL, OARS**	Loss precedes ADL loss
Gait / mobility	Gait speed, TUG	Tinetti POMA, SPPB, Berg	Gait below 0.8 m/s; TUG above 12 s; Tinetti below 24
Cognition	Mini-Cog, AD8	MMSE, MoCA, SLUMS	MMSE below 24
Mood	PHQ-2	GDS-15/30, PHQ-9, CSDD	GDS-15 of 5 or more
Nutrition	MNA-SF, SNAQ	MNA full, BMI, weight loss	MNA-SF 11 or less
Vision	Snellen	Formal ophthalmic review	Below 6/18
Hearing	Whispered voice test	Audiometry, HHIE	Fails at 2 feet
Social	Lubben Social Network Scale	OARS social section	LSNS-6 below 12
Rehabilitation outcome	—	**FIM**	18 items

C. Latest Updates (2023–Present)

1. WHO ICOPE — from deficit to capacity. The ICOPE 5-step pathway (screen, assess, intervene, follow up, community engagement) screens **six domains — mobility, cognition, vitality/nutrition, vision, hearing and psychological well-being** — either by a professional or by self-assessment through the **ICOPE Monitor** app. Step-2 tools are named explicitly: **SPPB for mobility; Mini-Cog, MoCA, MMSE or GPCOG for cognition; MNA, DETERMINE, MUST or SNAQ65+ for nutrition; PHQ-9 for mood; pure tone and speech audiometry for hearing**.

2. Minimum data sets are now mandated. The **German S3 guideline (2024)** requires **six core dimensions in at least 15 minutes**; the **Italian guideline (2024)** recommends the **Multidimensional Prognostic Index (MPI)** for prognosis; **ASCO 2023** mandates the **Practical Geriatric Assessment (PGA)** in oncology. The trend is **domain-mandated but tool-agnostic**.

3. Gait speed is being repositioned as the single best functional marker. A 2025 Age and Ageing study of about 3,600 older adults found that among all components of frailty and sarcopenia, **low physical performance (slow gait speed) was the best single predictor of incident basic and instrumental ADL disability**; sarcopenia as a whole construct was not associated with increased disability risk. **EWGSOP2 defines low physical performance as gait speed of 0.8 m/s or less.**

4. Automated and digital tools. The **electronic frailty index (eFI2)** is replacing the original eFI in English primary care from 2025, allowing **population-level functional risk stratification from routine records**. mHealth and wearable-based functional assessment gives **objective, quantitative data** in place of subjective observer-rated scales, but a 2024 systematic review found **high risk of bias and minimal integration into daily practice**.

5. Diagnostic accuracy data for screening instruments. In emergency-department settings, seven screens pooled across 1,663 participants gave **sensitivity 0.85 and specificity 0.77** against full CGA; **PRISMA-7 was the most sensitive (0.84–0.98)** and the **Clinical Frailty Scale the most specific**, both with AUROC around 0.90.

6. Indian use. LASI applies Katz-type ADL and Lawton-type IADL items; grip strength was the **most discriminant frailty component in Indian men (79.1%)** and physical activity in women (81.6%). **AWGS (not EWGSOP2) cut-offs** should be used for sarcopenia-related functional measures in Indians, and **BIA rather than DXA** is the practical mass-measurement modality.

D. Current Practice Update

Aspect	Pathy's 6th ed. (2022)	Current position (2023–2026)
Choice of tool	Broad menu; "no consensus on the best scale"	**Domain mandated, tool still flexible**, but specific tools named per setting
Gait speed cut-off	Below 0.6 m/s abnormal; 0.6–0.8 high-risk	**0.8 m/s or less** operative threshold (EWGSOP2/AWGS); best single predictor of disability
Who administers	Trained professional or therapist	**Self-assessment and digital screening validated**; community health workers in LMICs
Data source	Interview and bedside observation	**Automated extraction from electronic records** (eFI2); wearables and sensors
Framing	Deficit-based (ADL/IADL loss)	Complemented by **capacity-based** framing (WHO intrinsic capacity)
Sarcopenia link	Not integrated	Functional tools now feed directly into **sarcopenia case-finding** (SARC-F, grip strength, gait speed)

E. Exam One-Liners

- **Katz and Barthel measure ADL; Lawton measures IADL; Tinetti and TUG measure mobility; MNA nutrition; GDS mood; MMSE cognition; Lubben social.**
- **Barthel is what the patient does, not what they can do** — and has a **ceiling effect** in ambulant patients.
- **FIM has 18 items** across six areas and is the rehabilitation standard.
- **TUG above 12 seconds; Tinetti below 24 of 28; gait speed below 0.6 m/s (Pathy) or 0.8 m/s (EWGSOP2).**
- **0.1 m/s slower gait equals 7% higher fall risk.**
- **PSMS has grooming and ambulation; Katz has continence and transferring** — the classic distinguishing point.
- **Report tells you what they do; performance tells you what they can do** — measure both.
- **WHO ICOPE six domains:** mobility, cognition, vitality, vision, hearing, psychological.
- **A tool is only useful if its result changes the care plan.**

QUESTION 3

Functional Assessment in the Elderly

A. 20 High-Yield Points

Why Function Matters

1. **Functional assessment** means measuring **what the person actually does in daily life**. In geriatrics, **function is the common language** and assessment is the core method.
2. In an older person, **function predicts outcome better than diagnosis**. There is **no direct relationship between disease and functioning** — the same illness produces very different disability in different people, so function must be measured separately.
3. Functional decline results from **medical, functional, psychological, social and environmental** risk factors acting together. Hence assessment must be **multidimensional**.
4. A **baseline functional measure is essential** so that progress can be monitored and the effect of treatment or rehabilitation judged. **Reassess at least weekly during rehabilitation.**
5. Three levels are assessed: **impairment** (the deficit) → **activity restriction** (ADL/IADL loss) → **participation restriction** (loss of social role). Participation restriction is **unique to the individual** and depends largely on the ability to adapt.
6. Mobility must always be tested — **about half of people aged 85 and over report difficulty walking**, and gait disorders threaten independence and cause falls.

The Core Measures

7. The functional "review of systems" rests on three measures: **frailty, basic ADLs, and IADLs**.
8. **Principle of hierarchy:** if the patient is **severely ADL-dependent, do not test frailty**; if **robust or pre-frail, do not test basic ADLs**. Match the depth of assessment to the patient and the setting.
9. **Basic ADLs (personal care):** bathing, dressing, toileting, transferring, continence, feeding, grooming — **Katz, Barthel, Lawton PSMS**.
10. **IADLs (tasks needing instruments):** cooking, shopping, housework, laundry, telephone, transport, money, medicines — **Lawton IADL, OARS**.
11. **IADLs are lost first, ADLs later**, so IADL loss is the earlier warning sign. Always ask **"how does she manage?"**
12. **Always record premorbid function as well as present function**, especially after acute illness, a fall or hospital admission.

Domains to Cover

13. **Mobility, gait and balance:** gait speed (**below 0.6 m/s abnormal; 0.6–0.8 high-risk**), **TUG above 12 seconds**, **Tinetti below 24 of 28**, 30-second chair stand, 4-stage balance test, SPPB, Berg Balance Scale.
14. **Two screens are mandatory** in any older person with functional disability: **cognition** (MMSE) and **depression** (GDS or Zung).
15. **Other essential domains:** nutrition (**MNA**, SNAQ, weight loss), **special senses** (whispered voice test, Snellen), **oral health** (GOHAI), **continence**, and **social network and support** (**Lubben Social Network Scale**).
16. **Also assess:** pain, polypharmacy (Beers/STOPP-START), continence, sleep, alcohol (CAGE, SMAST-G), caregiver capacity, financial resources, and **home environment**.

How to Do It in Practice

17. Use **both report and performance**. Reports tell you what the person does **under real living conditions**; performance tests tell you what they can do **under standard conditions**.
18. **Grade each task** as: independent without difficulty / independent but with difficulty or an aid / needs human help / unable even with help. Distinguishing "independent with difficulty" adds prognostic value.
19. **Perform a task analysis** of the specific problem. *Example:* "difficulty dressing" may be due to choosing clothes, remembering to finish, buttoning, reaching overhead, or bending for shoes — pointing respectively to **cognitive impairment, poor finger mobility, or shoulder, back or hip disease**. Each demands a different treatment.
20. **Correlate any new functional loss** with **new drugs or dose changes (including over-the-counter)**, **changes in living conditions**, and **intercurrent acute illness (even mild)**. Remember the **cascade** — impaired gait leads to depression and withdrawal, and **immobility of any cause produces deconditioning that persists after the cause is removed**. Finally, **link assessment to a plan**: rehabilitation steps of **assessment, goal setting, therapy, aids and adaptations, education, psychological support, evaluation and follow-up**.

B. Practical Bedside Scheme

Step 1 — History Premorbid and current ADL/IADL status · exact task that fails · duration and rate of decline · pain · falls · continence · drugs (including over-the-counter) · nutrition and weight change · mood and motivation · social support and caregiver capacity · housing and environment.

Step 2 — Examination Postural blood pressure · vision and hearing screening · **observation of gait** · musculoskeletal (tone, power, joint range of motion, feet) · neurological (focal signs, sensation, proprioception) · **skin over pressure areas** · cognition (MMSE/MoCA) and mood (GDS).

Step 3 — Performance testing Gait speed over 4 metres · Timed Up-and-Go · 30-second chair stand · 4-stage balance · grip strength · observed transfer and stair climbing.

Step 4 — Baseline investigations Complete blood count, blood chemistry, **serum calcium (mg/dL), sodium (mEq/L), creatinine (mg/dL)**, thyroid function, vitamin B12, **25-hydroxy vitamin D**, haemoglobin (g/dL), fasting glucose or HbA1c, urinalysis.

Step 5 — Formulate and act Identify **reversible causes** → treat disease and pain → **deprescribe** → refer to physiotherapy, occupational therapy, dietician and social work → prescribe aids and adaptations → set goals with the patient → arrange **follow-up and case management**.

C. Latest Updates (2023–Present)

1. WHO ICOPE and intrinsic capacity. The five-step pathway screens **six domains — mobility, cognition, nutrition/vitality, vision, hearing and psychological well-being** — and can be done by a professional or by self-assessment through the **ICOPE Monitor** app. Reference centiles for intrinsic capacity and real-world screening data were published in *Nature Aging* (2023, 2024).

2. German DGG S3 Guideline (2024) defines minimum requirements — **six core dimensions in at least 15 minutes** — the first guideline to set a floor standard for functional assessment in hospital.

3. Age-Friendly Health Systems "4Ms." What Matters, Medication, Mentation and Mobility. In an eight-hospital 2024 evaluation of 3,103 admissions, patients receiving all 4Ms had **fewer 30-day emergency visits (16% vs 22%)**; **Mobility generated the highest "act-on" rate (83%)** of the four.

4. Gait speed as the single best marker. A 2025 longitudinal study found **slow gait speed the best single predictor of incident ADL and IADL disability**, outperforming both frailty and sarcopenia as constructs.

5. Italian CGA guideline (2024) — across 117 studies, CGA **maintains functional activities** and reduces hospitalisation, mortality, institutionalisation and delirium.

6. Oncology. ASCO 2023 requires geriatric assessment including **physical function** in all patients over 65 on systemic cancer therapy, using the **Practical Geriatric Assessment**.

7. Indian data. LASI wave-1 (31,464 adults aged 60 and above) found **about 3% with severe ADL disability and about 6% with severe IADL disability**, with **7.4% ADL dependency**. Rural surveys show far higher figures — **ADL disability 13.7% and IADL disability 38.7%** in rural Goa — associated with sex, education, financial dependence, comorbidity, alcoholism and **sensory impairment**. Functional tools are being adapted for community health worker delivery under **NPHCE**.

D. Current Practice Update

Aspect	Pathy's 6th ed. (2022)	Current position (2023–2026)
Core concept	Function measured as **deficit** (ADL/IADL loss)	**WHO shift to intrinsic capacity** — measure **reserve**, not only deficit
Number of domains	Broad, flexible list	**Standardised minimum sets** — ICOPE six domains; German S3 six dimensions in 15 minutes
Gait speed cut-off	Below 0.6 m/s abnormal	**0.8 m/s or less** widely used; slow gait now the best single predictor of incident disability
Who assesses	Interdisciplinary professional team	**Self-assessment and digital screening validated**; community health worker delivery in LMICs
Hospital framing	ACE units and geriatric assessment programmes	**4Ms Age-Friendly framework** — Mobility as a named, auditable care process
Sarcopenia	Not integrated into functional assessment	Functional assessment now **routinely linked to sarcopenia case-finding**

E. Exam One-Liners

- **Function is the common language of geriatrics.**
- **IADLs go first, ADLs later** — IADL loss is the earlier warning.
- **Two screens you must never omit: cognition and depression.**
- **Hierarchy rule:** ADL-dependent, skip frailty; robust, skip basic ADLs.
- **Record premorbid function, not just current function.**

- **TUG above 12 s; Tinetti below 24; gait speed below 0.6 m/s (Pathy) or 0.8 m/s (EWGSOP2).**
- **New functional decline equals a new drug, a new illness, or a new living situation** until proved otherwise.
- **Task analysis converts a vague complaint into a treatable diagnosis.**
- **WHO ICOPE six domains; 4Ms — What Matters, Medication, Mentation, Mobility.**
- **India (LASI): about 3% severe ADL and 6% severe IADL disability; 7.4% ADL dependency.**
- **Assessment without a linked care plan is not an assessment.**

QUESTION 4

Tests of Cognition and Rating Scales

A. 20 High-Yield Points

Principles

1. **Purpose of cognitive testing:** to establish **strengths and weaknesses of various functions relative to a standardised, norm-referenced scoring system**, so that the **nature and extent of cognitive deficits** can be determined, informing diagnosis and management and acting as a **comparator for past and future assessments**.
2. **A cognitive screen is not a diagnosis.** The MMSE and similar tools give an overall assessment of cognitive function, but **their use alone is not sufficient to establish a diagnosis of Alzheimer's disease or any dementia**.
3. **Cognitive screening is one of only two mandatory screens** (with depression) in any older person with functional disability.
4. **Key aspects of the mental status examination:** state of consciousness · general appearance and behaviour · orientation · **memory (short and long term)** · language · visuospatial function · **executive control functions** · other cognitive functions (calculations, proverb interpretation) · **insight and judgement** · thought content · mood and affect.
5. **Test all three components of memory: immediate recall** (repeating digits), **recent memory** (recalling three objects after a few minutes), and **remote memory** (details of early life).
6. **Executive function** involves the **planning, sequencing and execution of goal-directed activities**, and is **critical to the ability to perform IADLs**. Screen with the **clock-drawing test** (a component of the Mini-Cog) or **verbal fluency** (listing 12 animal names in one minute is normal).
7. **Interpretation depends on education and age.** The MMSE score is **dependent on sociocultural level and subject to a ceiling effect** — a crucial caveat in the Indian population, where illiteracy is common.
8. **Assess premorbid intellectual status** where possible (for example the National Adult Reading Test) to interpret other test scores correctly.
9. **Adapt tests to deficits.** In language impairment or dyspraxia, **recognition memory tests reduce the demand for expressive language** required by recall tests.
10. **Always include a standardised measure of mood** — depression is the commonest mimic of cognitive impairment.

Screening and Assessment Tools

11. **Recommended tools in primary care (Table 72.2):** - **Delirium: Confusion Assessment Method (CAM)** - **Dementia screening: AD8**, **Mini-Cog** - **Dementia assessment: SLUMS**, **MMSE**, **MoCA**
12. **Mini-Cog** screens **both memory (three-item recall) and executive function (clock drawing)** — quick, and relatively education-independent.
13. **MoCA and Addenbrooke's Cognitive Examination-Revised (ACE-R)** are described as **well-validated screening options**; ACE-R **incorporates the MMSE and clock-face drawing**.
14. **MMSE severity bands in Alzheimer's disease: mild 20–24, moderate 10–19, severe below 9.**
15. **Global staging scales combining cognition and ADLs: Global Deterioration Scale (GDS, Reisberg)** and the **Clinical Dementia Rating (CDR) scale**.
16. **Severe Impairment Battery (SIB)** was developed for subjects **too cognitively impaired for standard testing**.
17. **Memory-specific screens:** the **five-word test** and the **Memory Impairment Screen (MIS)** assess **anterograde verbal memory** at an initial consultation. For detailed evaluation, **free and cued recall** paradigms — the **Grober and Buschke test (Free and Cued Selective Reminding Test)** and the **California Verbal Learning Test** — distinguish mild Alzheimer's disease from healthy subjects with **more than 90% precision**, even when MMSE scores are similar.
18. **Domain-specific battery (Table 62.4 — Cardiff memory clinic):**

Domain	Tests
Premorbid ability	**National Adult Reading Test (NART)**
Cognitive screen / battery	**MMSE, MoCA, RBANS, Frontal Assessment Battery (FAB)**
Attention and concentration	Digits forward, digits backward, digit span
Language	**Boston Naming Test**, Irving Names Learning Test
Memory	Story recall (immediate and delayed), Kendrick tests, **California Verbal Learning Test**
Executive function	**Trail-Making Test A and B**, clock drawing
Visuospatial function	**Rey–Osterrieth Complex Figure**, clock drawing
Mood	**GDS-15**

Delirium Instruments

19. **Confusion Assessment Method (CAM)** — the standard; **sensitivity 94%, specificity 89%** with high inter-rater reliability. **Inattention** is tested at the bedside by **months of the year backwards, days of the week backwards, or digit span 3 or 4 backwards**. - **3D-CAM:** a structured **three-minute** screen — **sensitivity 96%, specificity 86% in patients with dementia**; **93% and 96% in those without**. - **Best single item: "months of the year backwards"** (sensitivity 83%, specificity 69%). Best two items: months backwards plus "what is the day of the week?" (sensitivity 93%, specificity 64%). - **CAM-ICU** for intubated patients — inattention tested by squeezing on the letter A in "SAVEAHAART"; level of consciousness by **RASS score other than 0**.

Practical Conduct of Testing

20. Tests must be **acceptable to the person and contain nothing belittling**. **Consent must be obtained**, the purpose explained, and **sensory and physical limitations accommodated** — adequate lighting, earphone amplifiers, suitable seating, minimal distractions. Provide **realistic reassurance with positively phrased feedback that highlights strengths as well as weaknesses**, and at the end **allow patients to comment on their own performance**. Ideally an experienced psychologist or neuropsychologist performs formal testing, though **much useful information can be obtained by any suitably trained professional**. Introduce questions **non-threateningly**, because many patients with early deficits respond defensively.

B. Comparison of the Main Cognitive Tools

Tool	Items / time	Domains	Main strength	Main limitation
AMT (Hodkinson)	10 questions, 2 min	Orientation, memory	Very quick; RCP/BGS endorsed	Crude; misses executive and visuospatial deficits
Mini-Cog	3 min	3-word recall + clock drawing	**Relatively education-independent**; screens executive function	Screen only
AD8	8 items, informant	Change over time	Informant-based, detects change	Needs a reliable informant
MMSE	30 points, 10 min	Orientation, registration, attention, recall, language, construction	Most widely known; severity bands defined	**Education- and culture-dependent; ceiling effect; poor for executive function**; copyrighted
MoCA	30 points, 10–15 min	Adds executive, abstraction, visuospatial, attention	**Superior for mild cognitive impairment and executive dysfunction**	Lower specificity; education adjustment (+1 if 12 years or less)
SLUMS	30 points, 10 min	Adds executive and clock tasks	Free to use; education-stratified cut-offs	Less international validation
ACE-R / ACE-III	100 points, 20 min	Five domains; incorporates MMSE	Differentiates **Alzheimer's from frontotemporal dementia**	Long
RBANS	30 min	Repeatable battery	Serial testing without practice effect	Requires training
Frontal Assessment Battery	6 items	Frontal/executive	Detects subcortical and frontal deficits	Narrow
CAM / 3D-CAM / CAM-ICU	3–5 min	Delirium	**Gold standard for delirium**	Needs training; misses hypoactive cases if hurried
GDS (Reisberg) / CDR	Interview	Global staging	Combines cognition and function	Time-consuming
SIB	40 min	Severe dementia	Usable when MMSE floor is reached	Specialist tool

C. Latest Updates (2023–Present)

1. 2024 NIA-AA Revised Criteria — Alzheimer's disease is now defined biologically. Diagnosis rests on **Core 1 biomarkers** — a positive **plasma p-tau217**, a positive **amyloid PET**, or abnormal **CSF ratios (p-tau181/Aβ42, t-tau/Aβ42, Aβ42/40)**. Cognitive testing now defines the **clinical stage**, not the disease itself. This is the single biggest conceptual change in the field.

2. Blood-based biomarkers have entered clinical use. Diagnostic models combining **plasma p-tau217 with age and ApoE genotype** achieve **AUC around 0.94** for Alzheimer's disease. In very elderly nursing-home and hospital populations, **plasma p-tau181, p-tau217 and GFAP differ significantly between cognitively normal and probable Alzheimer's groups**, while **Aβ42, Aβ40 and the Aβ42/40 ratio do not** — an important practical point, since amyloid ratios perform poorly in the oldest old.

3. Cognitive screens are being repositioned as triage. Rather than diagnosing, MMSE and MoCA increasingly serve to **decide who proceeds to biomarker testing**. Clinical trials now use combinations such as **CDR global score 0.5–1.0**

plus MMSE 21 or above plus a positive Core 1 biomarker for entry.

4. Mild behavioural impairment (MBI) — late-onset persistent neuropsychiatric symptoms — has emerged as an **early indicator of dementia risk that may precede measurable cognitive decline**, and is being studied alongside plasma p-tau217.

5. Digital and mobile cognitive assessment. Mobile cognitive assessment systems have demonstrated **diagnostic equivalence to MMSE and MoCA** in Alzheimer's screening; future direction is **composite risk scores combining digital tests, wearables and plasma p-tau217**. Regulatory and ethical frameworks for data security, algorithmic transparency and equitable access are still being established.

6. Standardised inclusion in assessment frameworks. Cognition/delirium is one of the **six mandatory core dimensions** of the German S3 CGA guideline (2024); **cognition** is one of the **six WHO ICOPE domains**, with **Mini-Cog, MoCA, MMSE or GPCOG** named as step-2 assessment tools; **Mentation** is one of the **4Ms** of Age-Friendly Health Systems.

7. Indian context. In LASI wave-1, **impaired cognition was present in 9.3%** of adults aged 60 and above. Culture- and education-fair instruments are essential: **Hindi Mental State Examination (HMSE)**, **Vernacular MoCA versions**, **Rowland Universal Dementia Assessment Scale (RUDAS)**, **Indian Council of Medical Research Neuro-Cognitive Tool Box**, and **AIIMS Comprehensive Neuropsychological Battery in Hindi** are used in place of a directly translated MMSE, because **the standard MMSE penalises illiteracy heavily**.

D. Current Practice Update

Aspect	Pathy's 6th ed. (2022)	Current position (2023–2026)
Basis of Alzheimer's diagnosis	Clinical, supported by cognitive tests and imaging	**Biological — 2024 NIA-AA criteria** based on Core 1 biomarkers
Role of MMSE/MoCA	Screening and severity grading	**Staging and triage** to biomarker testing; no longer diagnostic on their own
Biomarkers	CSF amyloid and tau "in some centres"	**Plasma p-tau217 in routine diagnostic pathways**; AUC around 0.94 with age and ApoE
Amyloid ratios	Presented as useful	**Aβ42/40 performs poorly in the oldest old**; p-tau217 and GFAP superior
Delivery	Face-to-face testing by trained staff	**Mobile and computerised assessment shown equivalent to MMSE/MoCA**
Preclinical detection	Subjective cognitive decline and MCI	Adds **mild behavioural impairment (MBI)** as an early neurobehavioural marker
Indian practice	MMSE with education caveat	**Education-fair tools mandatory** — HMSE, RUDAS, vernacular MoCA, ICMR NCTB

E. Exam One-Liners

- **A cognitive screen is not a diagnosis** — MMSE alone cannot diagnose Alzheimer's disease.
- **MMSE bands:** mild 20–24, moderate 10–19, severe below 9.
- **MMSE is education- and culture-dependent with a ceiling effect** — the key limitation in India.
- **MoCA beats MMSE for mild cognitive impairment and executive dysfunction.**
- **Mini-Cog = 3-word recall plus clock drawing** — screens memory and executive function in three minutes.
- **Clock drawing and verbal fluency screen executive function**, which drives **IADL** performance.
- **CAM sensitivity 94%, specificity 89%; 3D-CAM takes three minutes.**
- **Best single delirium item: months of the year backwards.**
- **ACE-R incorporates the MMSE and clock drawing** and helps separate Alzheimer's from frontotemporal dementia.
- **Free and cued recall (Grober–Buschke) separates mild Alzheimer's from normal with over 90% precision** even at similar MMSE scores.
- **Global staging: Reisberg GDS and CDR** combine cognition with function.
- **SIB is for patients too impaired for standard testing.**
- **2024 NIA-AA: Alzheimer's disease is now a biological diagnosis** — plasma **p-tau217**, amyloid PET, or CSF ratios.
- **Always pair cognitive testing with a mood scale**, and obtain consent and premorbid ability first.

QUESTION 5

Cognitive Assessment Tools

A. 20 High-Yield Points

Classification — the Framework to Open With

1. Cognitive assessment tools fall into **four functional groups**, and stating this classification first earns marks: - **Delirium instruments** — CAM, 3D-CAM, CAM-ICU, 4AT - **Brief screening tools** — Mini-Cog, AD8, AMT, GPCOG, MIS, six-item screener - **Cognitive screening batteries** — MMSE, MoCA, SLUMS, ACE-III, RUDAS - **Detailed neuropsychological batteries and staging scales** — RBANS, CVLT, Trail-Making, FAB, Boston Naming, Rey–Osterrieth; CDR and Reisberg GDS for staging
2. **A second axis is the informant dimension — performance-based** (patient does the task: MMSE, MoCA) versus **informant-based** (a relative reports change: **AD8, IQCODE**). Informant tools detect **change from a personal baseline** and are invaluable when education confounds performance tests.
3. **The tool must match the question:** *Is this delirium?* → CAM. *Is there any cognitive problem?* → Mini-Cog or AD8. *How severe and in which domain?* → MoCA, MMSE, SLUMS. *Which dementia is it?* → ACE-III with a full battery. *How advanced is it?* → CDR or Reisberg GDS.

Delirium Tools

4. **CAM** — sensitivity **94%**, specificity **89%**, high inter-rater reliability. Four features: **acute onset and fluctuating course, inattention, disorganised thinking, altered level of consciousness** (features 1 and 2 plus either 3 or 4).
5. **Inattention testing at the bedside: months of the year backwards, days of the week backwards, digit span 3 or 4 backwards**; or the examiner may simply observe **inappropriate distraction or loss of the thread of conversation**.
6. **3D-CAM** — three-minute structured version: **sensitivity 96%, specificity 86% in patients with dementia**; **93% and 96% without dementia**. **CAM-ICU** for intubated patients uses the "SAVEAHAART" letter-A task and the **RASS**.

Brief Screens

7. **Mini-Cog — three-item recall plus clock drawing**; screens **memory and executive function** in about three minutes and is **relatively education-independent**, making it the most practical screen in Indian outpatient settings.
8. **AD8** — an **eight-item informant questionnaire** detecting change over time; useful when the patient's education level makes performance testing unreliable.
9. **Abbreviated Mental Test (Hodkinson, 10 items)** — the **RCP/BGS-endorsed** memory and cognition screen; extremely fast but crude.
10. **Memory Impairment Screen and the five-word test** assess **anterograde verbal memory** specifically at a first consultation.

Screening Batteries

11. **MMSE (Folstein, 1975)** — 30 points across orientation, registration, attention/calculation, recall, language and construction. **Interpretation is complex because thresholds depend on education and age**; it has a **ceiling effect** and **tests executive function poorly**. Severity bands: **mild 20–24, moderate 10–19, severe below 9**.
12. **MoCA (Nasreddine, 2005)** — 30 points; adds **trail-making, cube copy, clock drawing, verbal fluency, abstraction and delayed recall**, so it detects **mild cognitive impairment and executive/subcortical dysfunction far better than the MMSE**. **Add 1 point if formal education is 12 years or less.**
13. **SLUMS (Saint Louis University Mental Status)** — 30 points; free of charge, with **separate cut-offs for those with and without a high-school education**.
14. **ACE-R / ACE-III** — 100 points across **attention/orientation, memory, verbal fluency, language and visuospatial function**; ACE-R **incorporates the MMSE and clock-face drawing**, and the **VLOM ratio helps separate Alzheimer's disease from frontotemporal dementia**.
15. **RUDAS (Rowland Universal Dementia Assessment Scale)** — designed to be **culturally and linguistically fair**, needing no literacy; highly relevant to multilingual, variably literate Indian populations.

Detailed and Domain-Specific Tools

16. **Domain mapping (Table 62.4):** premorbid ability — **NART**; screening battery — **MMSE, MoCA, RBANS, FAB**; attention — **digit span forward and backward**; language — **Boston Naming Test**; memory — **story recall, California Verbal Learning Test**; executive — **Trail-Making A and B, clock drawing**; visuospatial — **Rey-Osterrieth Complex Figure**; mood — **GDS-15**.
17. **Free and cued recall tests** — the **Grober and Buschke test** and the **CVLT** — separate mild Alzheimer's disease from normal ageing with **more than 90% precision**, even when MMSE scores are identical, because they demonstrate a **hippocampal amnestic pattern (cueing does not improve recall)**.

18. **Staging scales: Clinical Dementia Rating (CDR)** and the **Reisberg Global Deterioration Scale** combine cognition with ADL impact. The **Severe Impairment Battery** is used when impairment is too severe for standard testing.

Rules of Use

19. **Practical conduct:** obtain **consent**, explain the purpose, ensure **adequate lighting, hearing amplification, suitable seating and no distractions**, introduce questions **non-threateningly**, phrase feedback **positively**, and give the patient a chance to comment on their own performance at the end. Assess **premorbid intellectual status** first, and **always add a mood scale**.
20. **Two cardinal caveats:** - **Depression and delirium must be excluded before any score is attributed to dementia** — screening tools become unreliable in delirium, and depression is the commonest reversible mimic. - **No cognitive screening test is appropriate for determining decision-making capacity.** Screens of **executive function** such as the **clock-drawing test** or the **Executive Interview** correlate better with decisional capacity than the MMSE, but capacity requires its own task-specific assessment.

B. Comparison Table — Choosing the Right Tool

Clinical question	Tool of choice	Time	Notes
Is this delirium?	**CAM / 3D-CAM / CAM-ICU**	3–5 min	Sensitivity 94%, specificity 89%
Rapid screen in a busy clinic	**Mini-Cog**	3 min	Education-independent; memory + executive
Screen when the patient cannot be tested	**AD8 or IQCODE** (informant)	3 min	Detects change from baseline
General cognitive assessment	**MoCA**	10–15 min	Best for MCI; add 1 point if education 12 years or less
Severity grading in known dementia	**MMSE**	10 min	Mild 20–24, moderate 10–19, severe below 9
Free alternative with education cut-offs	**SLUMS**	10 min	Education-stratified
Differentiating dementia subtypes	**ACE-III**	20 min	VLOM ratio; Alzheimer's vs frontotemporal
Illiterate or multilingual patient	**RUDAS, HMSE, vernacular MoCA**	10 min	Culture- and literacy-fair
Detecting early Alzheimer's memory pattern	**Grober–Buschke, CVLT**	20–30 min	Cueing fails to improve recall
Frontal or subcortical deficit	**FAB, Trail-Making B, verbal fluency**	5–10 min	Subcortical screen
Global staging	**CDR, Reisberg GDS**	Interview	Combine cognition and function
Severe dementia	**SIB**	40 min	Below the MMSE floor

C. Latest Updates (2023–Present)

1. 2024 NIA-AA Revised Criteria define Alzheimer's disease **biologically** through **Core 1 biomarkers — plasma p-tau217, amyloid PET, or CSF p-tau181/Aβ42, t-tau/Aβ42 or Aβ42/40**. Cognitive tools now **stage the disease rather than diagnose it**.

2. Plasma p-tau217 is the practice-changing test. Models combining **plasma p-tau217, age and ApoE genotype** achieve **AUC about 0.94**. In the very elderly, **p-tau181, p-tau217 and GFAP discriminate probable Alzheimer's from normal cognition, while Aβ42, Aβ40 and the Aβ42/40 ratio do not** — so amyloid ratios should not be relied on in the oldest old.

3. Digital cognitive assessment. Mobile cognitive assessment systems show **diagnostic equivalence to MMSE and MoCA** in Alzheimer's screening. Future work is on **composite scores integrating digital tests, wearables (for example sleep fragmentation) and plasma p-tau217**, with cross-ethnic validation and regulatory frameworks still pending.

4. Mild behavioural impairment (MBI) — late-onset persistent neuropsychiatric symptoms — is now recognised as an early dementia-risk indicator that may **precede measurable cognitive decline**, and is being validated against plasma p-tau217.

5. Mandated inclusion in assessment frameworks. German S3 CGA guideline (2024): cognition and delirium is one of six core dimensions. **WHO ICOPE:** cognition is one of six intrinsic-capacity domains, with **Mini-Cog, MoCA, MMSE and GPCOG** as named step-2 tools. **Age-Friendly 4Ms:** cognition sits under **Mentation**. **ASCO 2023 Practical Geriatric Assessment** includes a cognitive domain for every cancer patient aged 65 and above.

6. Indian tools and data. LASI wave-1 found **impaired cognition in 9.3%** of those aged 60 and above. Because the MMSE penalises illiteracy heavily, Indian practice uses the **Hindi Mental State Examination (HMSE)**, **RUDAS**, **vernacular MoCA versions**, the **ICMR Neuro-Cognitive Tool Box**, and the **AIIMS Comprehensive Neuropsychological Battery in Hindi**.

D. Current Practice Update

Aspect	Pathy's 6th ed. (2022)	Current position (2023–2026)
What the tools do	Screen for and grade cognitive impairment	**Stage the disease** and **triage to biomarker testing**
Diagnosis of Alzheimer's	Clinical, supported by imaging and sometimes CSF	**Biological (2024 NIA-AA)** — Core 1 biomarkers
Blood tests	Not diagnostically useful	**Plasma p-tau217 in routine pathways**; GFAP adds value
Amyloid measures	CSF amyloid used in some centres	**Aβ42/40 unreliable in the oldest old**
Mode of testing	Paper-and-pencil, face to face	**Mobile and computerised testing equivalent to MMSE/MoCA**
Preclinical markers	Subjective cognitive decline, MCI	Adds **mild behavioural impairment**
Cross-cultural use	MMSE with an education caveat	**Literacy-fair instruments mandatory** in India — RUDAS, HMSE, vernacular MoCA

E. Exam One-Liners

- **Four groups of tools:** delirium instruments, brief screens, screening batteries, detailed batteries and staging scales.
- **CAM for delirium; Mini-Cog or AD8 to screen; MoCA or MMSE to assess; ACE-III to subtype; CDR or Reisberg GDS to stage.**
- **Mini-Cog = 3-word recall plus clock drawing**, and is relatively education-independent.
- **AD8 and IQCODE are informant-based** — they measure change from the person's own baseline.
- **MoCA detects MCI and executive dysfunction better than MMSE; add 1 point if education is 12 years or less.**
- **MMSE has a ceiling effect, is education-dependent, and tests executive function poorly.**
- **RUDAS, HMSE and vernacular MoCA are the literacy-fair choices for Indian patients.**
- **Grober–Buschke free and cued recall demonstrates the hippocampal amnestic pattern of Alzheimer's disease.**
- **ACE-R incorporates the MMSE and clock drawing.**
- **Exclude delirium and depression before interpreting any cognitive score.**
- **No cognitive screening test determines decision-making capacity** — executive tests correlate better than the MMSE, but capacity needs its own assessment.
- **2024 NIA-AA: plasma p-tau217, amyloid PET or CSF ratios define the disease; cognition defines the stage.**

QUESTION 6

Assessment of Decisional Capacity in an Older Person

A. 20 High-Yield Points

Concepts and Definitions

1. **Capacity and competence are not the same.** A **clinician evaluates a patient's capacity to make a decision**; **competence is a legal term** implying that a person **has been deemed competent or incompetent by a court of law**. Someone not competent to act on their own behalf **requires an agent to act for them**.
2. **Competence** refers to a person's ability to **act reasonably after understanding the nature of the situation being faced**.
3. **Capacity is decision-specific and time-specific.** A patient may have capacity for one decision and not another, and may regain capacity later.
4. **Capacity is presumed until proved otherwise.** A **diagnosis of dementia — even by type — is not a sufficient indicator of a person's ability to comprehend and express a meaningful preference**. Intellectual deficits are **spotty**: a person may get lost or forget things yet still make decisions **with appropriate prompting**.
5. **The ethical foundation is autonomy** — the capacity of each adult and cognitively competent individual to **process information and freely decide on a course of action that best suits their own values, preferences, goals and desires**. Respect for autonomy underpins **informed consent**.
6. **Preconditions of valid informed consent: competence to understand and decide**, and **voluntariness in deciding**. The **information element** is **disclosure of relevant information** about the condition and the proposed course of action.
7. **An unwise decision is not incapacity.** In a patient with capacity, autonomy means "*you may not do things to me without my permission*" — **even if a patient's decision to refuse treatment seems unwise, if they have capacity their refusal stands**, and the clinician's **duty of care does not diminish**; all other care continues without prejudice.
8. **Just as it is wrong to infantilise patients** by directing questions to others who answer faster, **it is equally wrong to prejudge their ability to participate in decisions about their own care**.

The Four Elements of Decisional Capacity

9. **Decisional capacity is predicated on four elements** — the single most examinable list: - **Understanding** — the ability to comprehend the disclosed information about the nature of the situation or medical problem, the procedures involved, and the risks and benefits of undergoing or not undergoing testing or treatment. - **Appreciation** — of the significance of the disclosed information and the potential risks and benefits **for one's own situation and condition**. - **Reasoning** — the ability to engage in a reasoning process about the risks and benefits. - **Choice** — being able to **express a choice** whether or not to undergo further testing or treatment.
10. **The four US legal standards for determining decisional capacity** (a parallel, equally examinable list): - The patient is **aware of the treatment options and expresses a treatment preference clearly and consistently**. - The patient can give a **conventionally understandable reason** for the decision that is **consistent with their past behaviour**. - The patient **remembers and can tell back the information** about diagnosis, prognosis and treatment options given by the practitioner. - The patient can **state what their decision means for their own state of health**.
11. **The sliding-scale principle (Figure 95.1):** the **standard of decisional capacity required rises with the risk of the intervention**. A **fully cognisant patient with good health literacy** can consent to high-risk options such as a **phase 1 clinical trial**; a **cognitively impaired patient lacking decisional capacity could not consent to experimental treatment** and, given reduced life expectancy in advanced dementia, would be unlikely to benefit from highly toxic therapy even if it is standard of care. **The area between is the domain of best clinical judgement.** Hence **"staging the ageing is as important as staging the cancer."**

Assessment in Practice

12. **Distinguish memory from understanding** — a person may fail a memory test yet understand the decision at hand.
13. **No cognitive screening test is appropriate to determine decision-making capacity.** Screening tools that focus on **executive function** — the **Clock Drawing Test** or the **Executive Interview** — are **more likely to be associated with decisional capacity** than the MMSE.
14. **Formal capacity instruments exist but are difficult for older adults**, because they require consideration of **hypothetical situations and complex abstract thinking**. Examples: **MacCAT-T (MacArthur Competence Assessment Tool for Treatment — the reference standard)**, **Aid to Capacity Evaluation (ACE)**, **Capacity to Consent to Treatment Instrument (CCTI)**, **Hopemont Capacity Assessment Interview**, **Understanding Treatment Disclosures**.
15. **A useful practical criterion** in routine informed consent is to **recheck with the patient after an interval** to be certain that they **understand the situation and consistently state the same preference**.

16. **Maximise capacity before declaring its absence.** Before deciding a person cannot decide: correct **sensory deficits** (a deaf patient may need a hearing aid, many older people need glasses), **treat delirium, depression, pain and infection**, review sedating drugs, use **simple language, repetition and rephrasing**, choose a **quiet, distraction-free environment**, and involve a familiar person. Written material must be legible — a leaflet is useless if **the typeface is too small, the colour contrast is poor, or there is no white space**.
17. **Formal assessment of mental state may be necessary**, but **no specific score is associated with an inability to make decisions about one's personal healthcare** — this is an important negative statement to write in an examination.
18. **Where capacity is absent:** consult any **advance care directive** and the **surrogate or proxy decision-maker**. A proxy may be formally appointed through an **enduring power of attorney**, or the patient's wishes may be recorded in an **advance directive**; if none exists, the lead clinician must decide in the patient's **best interests**, or apply for **guardianship**.
19. **Hierarchy of treatment decision-makers** (Australian model, quoted in the textbook): **enduring guardian with authority → guardian with authority → adult son or daughter → parent → sibling → primary unpaid carer → other person with a close personal relationship and frequent contact**. The clinician goes to the first person who is **18 or older, has full legal capacity, and is reasonably available and willing**.
20. **Communication and documentation:** - **Relatives must not be given the impression that the decision is theirs to make** — they may suffer significant distress from the emotional burden. Their role is to provide **valuable information about what would be in the patient's best interests**. - Where families cannot bear to decide, the humane option is **"informed assent"** — explicitly offering them the choice to **defer to the provider's judgement**, documented clearly in the record. - **Anticipate decisions early in admission** so the patient can be involved as much as possible and **proxy decision-making is avoided**. - Clinicians must disclose: **the condition being treated; the nature of the proposed treatment or procedure; anticipated results; recognised alternatives; and recognised serious risks, complications and benefits, including of non-treatment.** - **Document the assessment, the reasoning and the outcome.**

B. Structured Approach to a Capacity Assessment

Step	Action
1. Define the decision	Capacity is **decision-specific** — state exactly which decision is in question
2. Presume capacity	Start from presumption; a diagnosis alone never establishes incapacity
3. Optimise the person	Treat delirium, pain, depression, infection; correct vision and hearing; review sedatives; choose the best time of day and a quiet setting
4. Disclose	Condition, proposed treatment, expected outcome, alternatives, serious risks, and the consequences of non-treatment — in **plain language**
5. Test the four elements	**Understanding** ("tell me in your own words") · **Appreciation** ("what does this mean for you?") · **Reasoning** ("how did you weigh those up?") · **Choice** ("so what would you like to do?")
6. Apply the sliding scale	Higher risk demands a higher standard of capacity
7. Recheck	Re-ask after an interval to confirm **consistency** of preference
8. Conclude and act	If capacity present, the decision stands even if unwise. If absent, consult advance directive, then surrogate, then best interests or guardianship
9. Document	Record the specific decision, the disclosure made, the four elements, the conclusion and the reasoning

C. The Indian Legal Framework

1. **Mental Healthcare Act (MHCA), 2017** is the governing statute across India and was rewritten after India ratified the **UNCRPD in 2007**. Capacity has a **pivotal role**.
2. **Section 4, MHCA 2017:** every person is **presumed to have the capacity to make mental healthcare and treatment decisions** if they can **understand the information, appreciate the foreseeable consequences of the decision, or communicate the decision by speech, gesture or any other means**.
3. **Memory loss, cognitive deficits, or even a formal diagnosis of dementia do not imply incapacity automatically** — the Act emphasises **establishing capacity**, and gives **preference to the choices and wishes of a person who has capacity**.
4. **The opinion of the assessor or a third party that a decision is "unwise" must not influence the capacity assessment.** Capacity is **specific to the task and to the time**.
5. **Advance directives — Section 5(3):** an advance directive is **invoked only when the person becomes incapacitated** and **remains effective until they regain capacity**. The Act does not clearly distinguish advance statements from advance directives.

6. **Nominated representative:** decision-making by a nominated representative is **restricted to those lacking capacity for mental healthcare decisions**. **Supported decision-making** ranges from minimal support to complete support.
7. **Other relevant Indian law:** the **Maintenance and Welfare of Parents and Senior Citizens Act, 2007** (maintenance, protection, and transfer-of-property provisions), the **Rights of Persons with Disabilities Act, 2016** (limited guardianship), the **National Trust Act, 1999**, and **Elder Line 14567** for reporting abuse and neglect.
8. **Practical Indian caveat:** the **MacCAT-T, though the international reference standard, is not standardised for the Indian population**, and **family plays a central role in healthcare decision-making** in Indian and other Asian societies — creating real difficulties in operationalising individual autonomy and advance directives.

D. Latest Updates (2023–Present)

1. Indian evidence base is only now emerging. A 2025 descriptive cross-sectional study of mental capacity and advance directives in Indian persons with mental illness confirmed that **published Indian data remain very scarce**, that **the MacCAT-T is regarded as the gold standard but is not standardised for India**, and that the **central role of the family creates practical difficulty in determining advance directives** and enabling individual decision-making about future treatment.

2. Dementia and Indian law. A published analysis of the assessment of dementia under prevailing Indian laws confirms that under **Section 4 of MHCA 2017 a diagnosis of dementia does not itself establish incapacity**, and sets out the four components of the decision-making process as **understanding, appreciating, reasoning and communicating**. It also distinguishes **capacity** (clinical) from **competence** (legal — relating to valid contracts and financial and personal decision-making).

3. UK practice guidance (2024). NHS England guidance restates the functional test as the ability to **understand the relevant information, retain it long enough to make the decision, weigh up the arguments on each side, and communicate the decision** (by talking, signing or any other means). It provides a **practical checklist**: choose a quiet space free from distractions and noise, avoid jargon, **repeat and rephrase questions**, and **identify in advance what the "relevant information" for that specific decision is**. Essex Chambers publishes decision-specific guidance on what constitutes relevant information for different categories of decision.

4. High prevalence of impairment in institutional settings. Older adults evaluated in long-term care have **rates of capacity impairment between 44% and 69%** — an argument for routine, structured assessment rather than ad hoc judgement.

5. Convergence with capacity-based frameworks. The **German S3 CGA guideline (2024)** mandates cognition and delirium assessment as a core dimension; **WHO ICOPE** includes cognition and psychological well-being. Both feed directly into capacity evaluation, and the **Age-Friendly 4Ms** place **"What Matters"** — eliciting the person's own goals — as the first M, operationalising autonomy in routine care.

E. Current Practice Update

Aspect	Textbook position	Current position (2023–2026)
Legal framework	Generic Western frameworks; Australian guardianship hierarchy quoted	**India: MHCA 2017 governs**, with statutory presumption of capacity and nominated representatives
Effect of a dementia diagnosis	A diagnosis is not a sufficient indicator of incapacity	**Statutorily reinforced** — Section 4 MHCA: dementia does not imply incapacity
Instruments	Formal tools exist but are hard for older adults	**MacCAT-T remains the gold standard but is not standardised for Indian populations**
Cognitive tests	No screening test determines capacity; executive tests correlate better	Unchanged and reaffirmed
Role of family	Consult relatives for best interests, but do not transfer the decision	**In India, family-centred decision-making is normative**, creating tension with individual-autonomy models
Documentation	Recommended	Now expected to be **decision-specific**, recording the relevant information disclosed
Autonomy in routine care	Ethical principle	Operationalised as **"What Matters"** in the 4Ms framework

F. Exam One-Liners

- **Capacity is clinical and decision-specific; competence is legal and determined by a court.**
- **The four elements: Understanding, Appreciation, Reasoning, Choice.**
- **Capacity is presumed** — a diagnosis of dementia never establishes incapacity by itself.
- **An unwise decision is not incapacity.** If the patient has capacity, their refusal stands, and the duty of care does not diminish.
- **Sliding scale:** the higher the risk of the intervention, the higher the standard of capacity required.

- **"Staging the ageing is as important as staging the cancer."**
- **No cognitive screening test determines capacity**; **clock drawing and the Executive Interview** correlate better than the MMSE.
- **No specific MMSE score defines incapacity.**
- **MacCAT-T is the reference instrument** — but is not standardised for India.
- **Maximise capacity first:** treat delirium, depression and pain; correct hearing and vision; simplify language; choose a quiet room and the best time of day.
- **Recheck after an interval to confirm the preference is consistent.**
- **Section 4, MHCA 2017 (India):** presumption of capacity; understand, appreciate consequences, communicate by any means.
- **Section 5(3), MHCA 2017:** advance directive is invoked only on incapacity and lapses when capacity returns.
- **Never make the family feel the decision is theirs** — offer **"informed assent"** where they cannot bear to decide.
- **Anticipate decisions early in the admission** so that proxy decision-making is avoided.

QUESTION 7

Geriatric Syndromes — An Overview

A. 20 High-Yield Points

Concept and Definition

1. **A geriatric syndrome is a clinical condition in older people that does not fit into a discrete disease category.** Geriatric syndromes are **highly prevalent, multifactorial, and associated with substantial morbidity and poor outcomes**.
2. **The defining feature is a many-to-one and one-to-many relationship: multiple contributing causes converge on a single presenting problem**, and **any one problem may arise from many different causes**. This is the opposite of the classical single-disease model.
3. **Because diagnoses often do not tell the whole story in geriatrics, it is more helpful to think in terms of presenting problems.**
4. **The underlying principle is homeostenosis** — the progressive decline in the **reserve capacity of all organ systems** with increasing age, so that **several small deficits combine to cross a threshold** and produce a syndrome that no single deficit would have caused.
5. **The classical list — "the series of I's": Immobility · Instability (falls) · Incontinence · Intellectual impairment · Infection · Impairment of vision and hearing · Irritable colon · Isolation (depression) · Inanition (malnutrition) · Impecunity · Iatrogenesis · Insomnia · Immune deficiency · Impotence.**
6. **Morley's "new geriatric giants"** add **frailty, sarcopenia, anorexia of ageing and cognitive impairment** — reflecting the modern emphasis on pre-disability states.
7. **Illustrative example (immobility):** it may be caused by a **fractured hip, congestive heart failure, a recent fall with fear of falling, or pain from degenerative joint disease** — any or all together. It is essential to **explore the underlying causes with the individual** so that appropriate interventions can be implemented; **pain and fear of falling must be addressed before engaging the person in an exercise programme**.

Shared Risk Factors — the Unifying Concept

8. **Four shared risk factors run across the five best-validated geriatric syndromes** (Tinetti, Inouye, Gill and Doucette, *JAMA* 1995; Inouye et al., *JAGS* 2007): - **Older age** - **Baseline cognitive impairment** - **Baseline functional impairment** - **Impaired mobility**
9. **The five syndromes in which these were demonstrated: pressure ulcers, incontinence, falls, functional decline and delirium.**
10. **Clinical corollary:** because the risk factors are shared, **multiple risk factor reduction is more effective than the regular single-disease medical model**, and **one intervention can prevent several syndromes at once**.
11. **Landmark proof-of-concept trials: Inouye's multicomponent HELP intervention (NEJM 1999)** for delirium prevention, and **Tinetti's multifactorial intervention (NEJM 1994)** to reduce falls in the community — both showing that **targeting shared risk factors works**.
12. **The number of risk factors present determines the total risk**, which rises **proportionally** as they accumulate — demonstrated by Tinetti for dizziness (five to seven significant risk factors).

Iatrogenesis — a Special Emphasis

13. **Iatrogenesis is an all too common problem, and the risk increases with exposure to the care system** — tests, surgical interventions, medications and treatments. In every interaction the **risk–benefit balance must be considered and explained** to the individual and their proxy.
14. **Medication management** needs particular deliberation, because older adults have **marked changes in absorption, metabolism and excretion**. Use **Beers criteria and STOPP-START**, review **anticholinergic burden**, and check **QTc risk with psychotropics**.
15. **Even more dangerous is the careless, hasty application of clinical labels.** A patient who becomes confused in hospital may **not** have dementia; a person with an occasional urinary accident is **not necessarily incontinent**. **Labelling a patient as demented or incontinent is too often the first step towards nursing-home placement — a setting that can make such labels self-fulfilling prophecies.**

Assessment and Management

16. **The clinical method for any geriatric syndrome:** - Treat the **presenting problem as a syndrome, not a diagnosis** - Enumerate **all contributing factors** rather than searching for one cause - Look specifically for the **shared risk factors** — cognition, function, mobility - Search for **reversible contributors**: a **new drug or dose change (including over-the-counter), an intercurrent acute illness, or a change in living conditions** - Intervene on **as many factors as possible simultaneously** - **Link assessment to a care plan and long-term follow-up**

17. **Comprehensive geriatric assessment is the definitive vehicle** for identifying and managing geriatric syndromes, because it is the only method that systematically covers all the contributing domains at once.
18. **Criteria for targeting CGA in diabetes (Table 86.6)** show how syndromes drive referral: **presence of a geriatric syndrome (confusional state, depression, falls, incontinence, immobility, pressure sores)**; pre-frailty or frailty; several coexisting morbidities with complex drug regimens; disability from lower-limb vascular disease or neuropathy requiring rehabilitation; and **absence of a terminal illness or dementing syndrome**.
19. **Cross-syndrome prevention principles: mobilise early ("bed is bad")**, **deprescribe**, **maintain hydration and nutrition**, **correct vision and hearing**, **avoid restraints and catheters**, **screen for cognition and mood**, and **treat pain**. Because pressure injuries, falls, delirium and functional decline share risk factors, **multifactorial interventions delivered by a multidisciplinary team** are effective across all of them.
20. **The major barrier is implementation, not evidence.** Preventive strategies of proven effectiveness for **delirium and falls** exist, but **major barriers have been identified in translating research advances into clinical practice and policy**.

B. The Major Geriatric Syndromes at a Glance

Syndrome	Key shared drivers	First-line action
Falls and instability	Gait/balance impairment, drugs, vision, cognition, environment	Multifactorial assessment; TUG, Tinetti; deprescribe; strength and balance exercise; home hazards
Immobility	Pain, disease, fear, iatrogenesis, environment	Treat pain and cause; early mobilisation; physiotherapy and occupational therapy
Delirium	Cognitive impairment, sensory deficits, drugs, infection, dehydration, immobility	**CAM**; HELP-type multicomponent prevention; treat precipitants
Dementia and cognitive impairment	Age, vascular risk, sensory deficits	Mini-Cog/MoCA; exclude reversible causes; caregiver support
Incontinence	Mobility, cognition, drugs, local pathology	Bladder diary; exclude retention, infection, impaction; timed toileting
Pressure injury	Immobility, nutrition, incontinence, low weight	Braden/Norton; pressure relief; nutrition; skin care
Frailty and sarcopenia	Disease, poor intake, sarcopenia, executive dysfunction	FRAIL screen; resistance exercise plus protein 1 g/kg/day; deprescribe if more than 5 illnesses
Malnutrition and anorexia of ageing	Social, sensory, gastric, hormonal factors	MNA; **MEALS-ON-WHEELS** mnemonic
Polypharmacy and iatrogenesis	Exposure to the care system	Beers, STOPP-START; medication reconciliation
Functional decline	All of the above	CGA and rehabilitation
Depression and isolation	Loss, disability, chronic disease, loneliness	PHQ-2 then GDS-15; social prescribing
Sensory impairment	Age-related	Snellen, whispered voice test; spectacles, cataract surgery, hearing aids
Insomnia	Pain, nocturia, drugs, depression, environment	Sleep hygiene; avoid hypnotics
Elder abuse and neglect	Dependency, caregiver burden, cognitive impairment	Direct enquiry; **Elder Line 14567** in India

C. Latest Updates (2023–Present)

1. The shared-risk-factor model is now the accepted organising principle. The Inouye/Tinetti framework — **four shared risk factors (older age, baseline cognitive impairment, baseline functional impairment, impaired mobility) across five syndromes (pressure ulcers, incontinence, falls, functional decline, delirium)** — remains the definitional basis, with current work focused on **synergistic interactions between risk factors** and on the **basic mechanisms** underlying multifactorial conditions.

2. Screening tools have been packaged. The **Rapid Geriatric Assessment (RGA)** bundles four syndromes — **frailty, sarcopenia, geriatric anorexia and cognitive decline**. Administered to **16,615 people aged 65 and over in Missouri between 2015 and 2024**, it showed that nearly **40% reported five or more illnesses (37.3%)**, linking syndrome screening to multimorbidity risk.

3. Multiple geriatric syndromes are the rule, not the exception. A Chinese community study of 706 adults aged 60 and above (2022–23) assessed **ten syndromes — depressive symptoms, cognitive impairment, pain, falls, sleep disturbance, constipation, polypharmacy, multimorbidity, malnutrition and frailty** — and defined **"multiple geriatric syndromes" as two or more in one individual**. A 2023–24 hospital CGA study in Shanghai similarly evaluated **pain, sleep, constipation, fall risk, urinary incontinence, polypharmacy, nutritional risk and dementia** in a single assessment, using **Mini-Cog, Snellen chart and Timed Up and Go** for in-depth evaluation of those screening positive.

4. Standardised syndrome-based CGA is now guideline-mandated. The **German S3 guideline (2024)** requires **six core dimensions — self-help ability, mobility, cognition/delirium, affect, nutrition and social situation** — which

map almost exactly onto the major geriatric syndromes. The **Italian guideline (2024)**, from 117 studies, showed CGA **reduces hospitalisation, mortality, institutionalisation and delirium risk** and **improves prescribing appropriateness**.

5. WHO ICOPE reframes syndromes as losses of intrinsic capacity. The six domains — **mobility, cognition, vitality/nutrition, vision, hearing and psychological well-being** — are essentially the geriatric syndromes re-expressed as **capacities to be preserved rather than deficits to be treated**.

6. Frailty is now the strongest single predictor of adverse syndrome outcomes. A pooled odds ratio of **9.82 for incident ADL disability in frail older adults** has been reported, and the **NEJM 2024 review by Kim and Rockwood** established **"frailty-guided clinical care"**, stratifying management across **robust, pre-frailty, frailty and end-stage frailty**.

7. Indian burden. LASI wave-1 (31,464 adults aged 60 and above) found **44.3% with no geriatric condition, 32.7% with one, 15.9% with two and 7.1% with three or more**. Individual prevalences: **underweight 26.9%, dizziness 14.5%, impaired vision 13.7%, impaired hearing 9.6%, impaired cognition 9.3%, major depressive disorder 8.2%, injurious falls 5.7%, incontinence 4.0%**, with **7.4% ADL dependency**. **Both a higher number of geriatric conditions and a higher number of chronic conditions predicted ADL dependency.** Frailty affects **29.2%** and pre-frailty **58.8%**. The most disabling multimorbidity combination is **hypertension plus arthritis plus high depressive symptoms**.

D. Current Practice Update

Aspect	Textbook position	Current position (2023–2026)
Definition	Descriptive list of "I's"; concept acknowledged as poorly defined	**Operationalised** around four shared risk factors across five validated syndromes
Number recognised	14 classical "I's" plus new giants	Studies now routinely screen **8–10 syndromes simultaneously** in one CGA
Screening	Clinician-led, opportunistic	**Packaged instruments (Rapid Geriatric Assessment)** and **guideline-mandated core domain sets**
Framing	Deficits and presenting problems	Complemented by **WHO intrinsic capacity** — the same domains framed as capacities
Frailty	One syndrome among many	The **central organising syndrome**, used to guide treatment intensity ("frailty-guided care")
Evidence gap	Multifactorial interventions effective for falls and delirium	Evidence secure; **the barrier is implementation and policy translation**
Indian data	Not addressed	**LASI provides national prevalence**; over half of Indian elders have at least one geriatric condition

E. Exam One-Liners

- **A geriatric syndrome is a multifactorial condition that does not fit a single disease category.**
- **The core principle is homeostenosis** — reduced reserve across all organ systems.
- **Many causes, one presentation; one presentation, many causes.**
- **The series of I's:** Immobility, Instability, Incontinence, Intellectual impairment, Infection, Impairment of vision and hearing, Irritable colon, Isolation, Inanition, Impecunity, Iatrogenesis, Insomnia, Immune deficiency, Impotence.
- **New geriatric giants:** frailty, sarcopenia, anorexia of ageing, cognitive impairment.
- **Four shared risk factors:** older age, baseline cognitive impairment, baseline functional impairment, impaired mobility.
- **Five validated syndromes:** pressure ulcers, incontinence, falls, functional decline, delirium.
- **Multiple risk factor reduction beats the single-disease model** — proved by Inouye (delirium, 1999) and Tinetti (falls, 1994).
- **Iatrogenesis rises with exposure to the care system.**
- **The careless application of a label is more dangerous than the syndrome** — "demented" or "incontinent" can become a self-fulfilling prophecy leading to institutionalisation.
- **CGA is the definitive vehicle for detecting and managing geriatric syndromes.**
- **India (LASI): 55.7% of elders have at least one geriatric condition; 7.1% have three or more; ADL dependency 7.4%.**
- **The evidence is settled; the barrier is implementation.**

End of Set 1.