FROM BEDSIDE HISTORY TO CLINICAL DIAGNOSIS

AETCM

Medicine Long & Short Cases for Final MBBS

Case-Taking Proforma · History · Examination · Diagnosis

Step-by-step history taking and clinical examination for the eleven high-yield long cases and seventeen high-yield short cases of the final MBBS medicine practical examination — what to ask, what to look for, what each finding means, how to present and what the examiner will ask next

COPD · Pleural effusion · Pneumonia · Mitral stenosis · Mitral regurgitation · Aortic stenosis · Cirrhosis with ascites · CKD · Nephrotic syndrome · Stroke · Hemiplegia · Hypertension · Murmurs · PVD · DVT · Pneumothorax · Splenomegaly · Hepatomegaly · Cranial nerves · Parkinsonism · Thyroid · Anaemia · Oedema



By

Dr. Gireesh Kumar K.P.

For final MBBS students, interns and postgraduates preparing for clinical examinations in medicine

Medicine Long & Short Cases for Final MBBS

History taking and clinical examination — a case-taking proforma

COPD · Pleural effusion · Pneumonia · Mitral stenosis · Mitral regurgitation · Aortic stenosis
Cirrhosis with ascites · Chronic kidney disease · Nephrotic syndrome · Stroke · Hemiplegia
Seventeen short cases — cardiovascular, respiratory, abdomen, neurology, endocrine and general



Dr. Gireesh Kumar K.P.

Academy for Emergency, Trauma & Critical care Medicine (AETCM)
First Edition · October 2026

Disclaimer. This proforma is a learning guide for medical students to structure history taking and clinical examination at the bedside. Clinical findings, differential diagnoses and investigations are given for teaching; management must follow current national guidelines and senior supervision. Examination must always be performed with the patient's consent and privacy, and with a chaperone where appropriate. The author and publisher accept no liability for any consequence arising from the use of this information.

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Preface

The final MBBS clinical examination in medicine is decided at the bedside. A student is given a patient, forty-five minutes and a blank sheet of paper for the long case, and a few minutes and a single instruction — “examine this patient's abdomen”, “look at the face”, “listen to the heart” — for each short case. Examiners do not look for rare diagnoses. They look for a complete, ordered history, a confident examination, findings that agree with each other and a diagnosis that the student can defend.

This book is a case-taking proforma for exactly those cases. It covers the eleven long cases and seventeen short cases that appear most often in university examinations — the chronic chest, the valvular heart, the cirrhotic, the kidney, the stroke and the hemiplegic patient; and the short cases of the blood pressure, the murmur, the limb, the chest, the abdomen, the cranial nerves, the tremor, the thyroid, the pale and the swollen patient.

Every case is laid out on three pages. The first page is the history: bio data, presenting complaints and the questions that build the history of presenting illness, each with what the answer suggests. The second page completes the history and takes the student through the general and systemic examination in the order in which it is presented. The third page brings the case together — how to sum up, how to write the diagnosis, the differential diagnoses, the investigations, and the questions that examiners ask most often.

Dotted lines are spaces to write the patient's own details, so that the book can be used both as a study guide and as a working proforma on the ward. Use it to organise your thinking — but let every finding come from the patient in front of you. Ask with patience, examine with consent and gentleness, and present with clarity.



Dr. Gireesh Kumar K.P.
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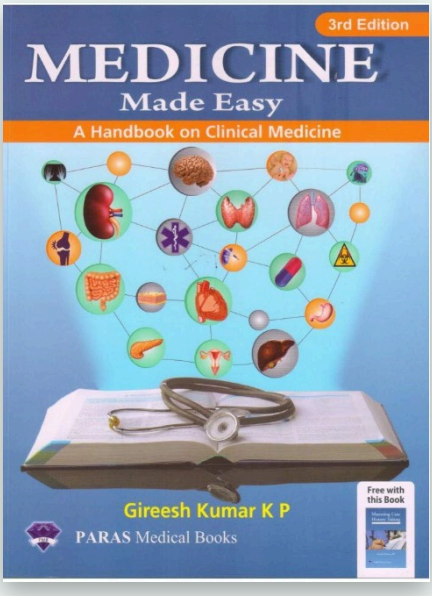
Headings followed by a dotted line are spaces to write the patient's own details. Bulleted points tell you what to ask or look for and what the finding suggests. Pages 5, 24, 34, 41, 54, 64, 74, 90, 97 and 98 feature other books by the author.

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FROM THE SAME AUTHOR

Dr. Gireesh Kumar K.P.

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CHAPTER 1 · LONG CASE · RS

Chronic Obstructive Pulmonary Disease (COPD)

Smoke or biomass exposure, progressive breathlessness, exacerbations and cor pulmonale

Bio data

Name ..... **IP No.** .....

Age ..... **Sex** .....

Occupation .....

Mines, quarries, construction, textile mills, cotton and grain dust, chemical fumes — occupational COPD

Address .....

Rural home with firewood or dung-cake chulha — biomass COPD, common in non-smoking Indian women

Age and sex

- Usually >40 years; symptoms begin after 20+ pack-years of smoking
- COPD in a young non-smoker (<40 years) or with basal emphysema → think of α1-antitrypsin deficiency

Presenting complaints (in chronological order)

1. .....
2. .....
3. .....

- Typical: breathlessness on exertion for ____ years, cough with expectoration for ____ years, increase in breathlessness / fever / change in sputum for ____ days (acute exacerbation), swelling of legs for ____ days (cor pulmonale)

History of presenting illness

Breathlessness

Onset and duration .....

- Insidious and slowly progressive over years — COPD; episodic with normal periods in between — asthma
- Grade with the **mMRC dyspnoea scale** (0: only on strenuous exercise · 1: hurrying or walking up a slope · 2: walks slower than peers or stops on level ground · 3: stops after ~100 m · 4: too breathless to leave the house or dress)
- Orthopnoea, paroxysmal nocturnal dyspnoea — rule out left heart failure; diurnal or seasonal variation, triggers — asthma

Cough and sputum

Duration .....

- Productive cough on most days for ≥3 months in 2 consecutive years — **chronic bronchitis**
- Early-morning, mucoid sputum; becomes purulent (yellow / green) during exacerbations
- Quantity: large, foul-smelling, postural sputum — bronchiectasis or lung abscess
- Haemoptysis — always rule out pulmonary TB and carcinoma lung in a smoker

Wheeze and chest tightness

- Present in COPD and asthma; ask about relief with inhalers

Exacerbations

- Number per year, hospital admissions, ICU stay, non-invasive ventilation (NIV) or intubation
- ≥2 moderate exacerbations or ≥1 hospitalisation a year → high-risk (GOLD group E)

Symptoms of complications

- Leg swelling, abdominal distension, right hypochondrial pain — **cor pulmonale**
- Morning headache, drowsiness, confusion, flapping tremor — **CO_2 retention**
- Sudden worsening of breathlessness with chest pain — **pneumothorax** (ruptured bulla)
- Loss of weight and appetite — advanced disease, TB or carcinoma

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Past history

- Similar episodes and hospitalisations; childhood asthma, recurrent chest infections, measles / whooping cough (bronchiectasis)
- Pulmonary tuberculosis — treatment taken, completed? (post-TB obstructive airway disease is common in India)
- Hypertension, diabetes, ischaemic heart disease, heart failure — common co-morbidities

Treatment history

- Inhalers: SAMA / LAMA, SABA / LABA, inhaled corticosteroid (ICS); device used and technique
- Home oxygen — hours per day; nebulisation; oral steroids, theophylline; antibiotic courses
- β-blockers, NSAIDs — may worsen bronchospasm in asthma; vaccination: influenza, pneumococcal

Personal history

Smoking

Pack-years = (cigarettes per day ÷ 20) × years smoked. Beedi smokers: 1 beedi ≈ ¼–½ cigarette. Ask about hookah and chillum.

- Current or ex-smoker; when stopped; attempts to quit; passive smoking
- Biomass exposure — hours per day of cooking over firewood / dung cake × years
- Alcohol, appetite, sleep (snoring, daytime sleepiness — overlap with obstructive sleep apnoea), bowel and bladder

Family history

- COPD, asthma, atopy; early-onset emphysema in siblings (α1-antitrypsin deficiency); TB contacts

General examination

Built / nourishment

BMI

- Patient sitting up, leaning forward with hands on knees (**tripod position**), pursed-lip breathing — emphysema
- Cachexia — advanced COPD; plethora — secondary polycythaemia
- **Central cyanosis**, clubbing (**not** a feature of COPD — think of bronchiectasis, lung abscess, carcinoma or ILD)
- Bounding pulse, warm peripheries, flapping tremor (asterixis) — CO_2 retention
- Raised JVP with prominent v wave, pedal oedema — cor pulmonale with tricuspid regurgitation
- Nicotine staining of fingers; use of accessory muscles; indrawing of intercostal spaces

Pulse

BP

Respiratory rate

SpO_2 (room air)

Temperature

JVP

Respiratory system examination

Inspection

- Barrel-shaped chest (AP : transverse diameter ≈ 1 : 1; normal 5 : 7)
- Horizontal ribs, widened subcostal angle
- Paradoxical indrawing of lower ribs — **Hoover's sign**
- Use of sternocleidomastoids, scalenes, alae nasi
- Reduced cricosternal distance (<3 fingers)

Palpation

- Trachea central; **tracheal tug** (descent with inspiration)
- Reduced chest expansion; apex beat may not be felt
- Vocal fremitus reduced bilaterally

Percussion

- Hyper-resonant note
- Upper border of liver dullness pushed down (below 6th rib)
- Cardiac dullness obliterated

Auscultation

- Vesicular breath sounds with **prolonged expiration**; diminished breath sounds in emphysema
- Polyphonic expiratory **wheeze**; early inspiratory coarse crackles
- Forced expiratory time >6 s (bedside test of airflow obstruction)
- Loud P2, parasternal heave, TR murmur — pulmonary hypertension

Other systems

- CVS: right heart failure signs · Abdomen: tender liver, ascites · CNS: drowsiness, papilloedema (CO_2 narcosis)

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Summary

Diagnosis — how to write it

Diagnosis

FORMAT

COPD (chronic bronchitis / emphysema predominant) — smoking or biomass-related — GOLD spirometry grade 1–4 — GOLD group A / B / E — with / without acute exacerbation — with / without complications (cor pulmonale, type 2 respiratory failure, secondary polycythaemia).

Differential diagnosis

Condition	Points in favour
Bronchial asthma	Younger age, atopy, episodic symptoms, normal in between, nocturnal cough, full reversibility on spirometry
Bronchiectasis	Copious purulent sputum, haemoptysis, clubbing, coarse leathery crackles, past severe childhood infection or TB
Post-TB obstructive airway disease	Old TB, fibrosis, tracheal shift, apical crepitations, reduced volume of one hemithorax
Left heart failure	Orthopnoea, PND, cardiomegaly, S3, basal fine crackles, raised BNP
Interstitial lung disease	Dry cough, clubbing, fine end-inspiratory "Velcro" crackles, restrictive spirometry

Investigations

Test	What to look for
Spirometry (post-bronchodilator)	**FEV_1/FVC <0.70** confirms airflow limitation; FEV_1 % predicted grades severity — GOLD 1 ≥80%, 2: 50–79%, 3: 30–49%, 4 <30%
Chest X-ray	Hyperinflation (>6 anterior ribs), flat diaphragm, tubular heart, bullae, prominent pulmonary arteries
Complete blood count	Polycythaemia (Hct >55%); eosinophilia (>300/µL predicts steroid response)
Arterial blood gas	Type 2 respiratory failure — PaO_2 <60, $PaCO_2$ >45 mmHg; compensated respiratory acidosis
ECG / Echo	P pulmonale, right axis deviation, RVH; pulmonary hypertension, RV function
Sputum	Gram stain, culture; AFB / CBNAAT to exclude TB
Others	HRCT for bullae and bronchiectasis; serum α1-antitrypsin if young or basal emphysema

Management in one glance

- Stop smoking / biomass exposure; vaccination; pulmonary rehabilitation
- Inhalers: LAMA or LAMA + LABA; add ICS if frequent exacerbations and eosinophils ≥300/µL
- Long-term oxygen therapy ≥15 h/day if PaO_2 ≤55 mmHg (or ≤59 with cor pulmonale / polycythaemia)
- Exacerbation: controlled O_2 (target SpO_2 88–92%), nebulised bronchodilators, prednisolone 40 mg × 5 days, antibiotics if sputum purulent, NIV for acidotic hypercapnia

EXAMINER ASKS

Pink puffer vs blue bloater? · Why is clubbing not seen in COPD? · Define chronic bronchitis and emphysema · Why controlled oxygen? · Criteria for LTOT · Causes of COPD in non-smokers in India.

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CHAPTER 2 · LONG CASE · RS

Pleural Effusion

Fluid in the pleural space — find the side, the size and, above all, the cause

Bio data

Name ..... **IP No.** .....

Age ..... **Sex** .....

Occupation .....

Asbestos exposure (shipyards, roofing, brake linings) — mesothelioma

Address .....

Age and sex

- Young adult in India — **tuberculous effusion** is the commonest cause
- Elderly smoker — malignancy (carcinoma lung); woman with breast lump — metastatic effusion
- Young woman with joint pains, rash — SLE (bilateral, small effusions)

Presenting complaints

1. .....
2. .....
3. .....

- Typical: breathlessness for ____ weeks, fever with evening rise for ____ weeks, chest pain on one side for ____ days, dry cough, loss of weight and appetite

History of presenting illness

Chest pain

- Sharp, **pleuritic** (worse on deep breathing and coughing), localised; may be referred to the shoulder tip (diaphragmatic pleura)
- Pain often **decreases as the fluid accumulates** (pleural layers separate) while breathlessness increases

Breathlessness

- Gradual onset, progressive — proportional to the amount of fluid; lying on the affected side may be more comfortable
- Sudden onset — haemothorax, pneumothorax, pulmonary embolism

Cough

- Dry, irritating — pleural irritation; productive with purulent sputum — parapneumonic effusion / empyema
- Haemoptysis — TB, carcinoma, pulmonary infarction

Fever

- Low-grade, evening rise, night sweats — **tuberculosis**
- High-grade with chills — parapneumonic effusion; swinging fever, toxicity — **empyema**

Constitutional symptoms

- Loss of weight and appetite — TB, malignancy

History to find the cause (ask systematically)

- **Heart**: exertional breathlessness, orthopnoea, PND, palpitation — cardiac failure (bilateral, right > left)
- **Liver**: jaundice, abdominal distension, haematemesis, alcohol — cirrhosis (hepatic hydrothorax, usually right)
- **Kidney**: facial puffiness, frothy urine, oliguria — nephrotic syndrome, CKD
- **Malignancy**: breast lump, haemoptysis, hoarseness, bone pains, lymph nodes
- **Connective tissue**: joint pains, rash, photosensitivity, oral ulcers — SLE, rheumatoid arthritis
- **Abdomen**: pancreatitis (left effusion), subphrenic abscess · **Thyroid**: hypothyroidism
- **Drugs**: methotrexate, amiodarone, nitrofurantoin, minoxidil; trauma to chest
- Calf pain, recent surgery or immobilisation — pulmonary embolism

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Past history

- Tuberculosis — previous episodes, ATT taken and completed; contact with a TB patient
- Similar episodes, previous pleural tapping (number, colour of fluid), intercostal drainage
- Heart disease, liver disease, kidney disease, malignancy, diabetes, HIV

Personal history

- Smoking (pack-years), alcohol (cirrhosis, pancreatitis), occupational asbestos exposure
- Diet, appetite, sleep, bowel and bladder habits

Family history

- TB in the household; malignancy

Treatment history

- ATT (drugs, duration), diuretics, antibiotics, chemotherapy; drugs causing effusion

General examination

Built / nourishment ..... **Weight** .....

- Pallor, icterus (cirrhosis), cyanosis, **clubbing** (empyema, carcinoma lung, mesothelioma)
- **Lymph nodes** — supraclavicular / cervical: TB or malignancy (examine for matting, sinuses)
- Pedal oedema, facial puffiness (nephrotic), raised JVP (heart failure), breast lump, thyroid
- Signs of liver cell failure — palmar erythema, spider naevi, gynaecomastia

Pulse ..... **BP** .....

Respiratory rate ..... **SpO_2** .....

Temperature ..... **JVP** .....

Respiratory system examination

Inspection

- Fullness of intercostal spaces on the affected side (large effusion)
- **Diminished movement** on the affected side
- Trachea and apex shifted to the opposite side
- Scars of previous tapping or ICD tube

Palpation

- **Trachea shifted to the opposite side** (large effusion); central in small effusion or when the lung beneath is collapsed
- Apex beat displaced to the opposite side
- Expansion reduced on the affected side
- **Vocal fremitus diminished or absent**

Percussion

- **Stony dull note** over the fluid
- Upper border of dullness: curve rising towards the axilla (**Ellis S-shaped curve**)
- **Traube's space dull** — left effusion
- Shifting dullness absent unless hydropneumothorax

Auscultation

- Breath sounds **diminished or absent** over the fluid
- Vocal resonance diminished
- Above the fluid: **bronchial breathing** (aegophony, whispering pectoriloquy) — compressed lung
- Pleural rub — before fluid accumulates or after it is absorbed

Size of effusion (clinical)

- Small: dullness up to 6th rib in front · Moderate: up to 4th rib · Massive: up to 2nd rib, mediastinal shift

Other systems

- CVS: S3, murmurs, pericardial rub · Abdomen: ascites, hepatosplenomegaly, ovarian mass · CNS: as relevant

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Summary

Diagnosis — how to write it

Diagnosis

FORMAT

Right / left / bilateral — small / moderate / massive — pleural effusion — probably exudative (tuberculous / malignant / parapneumonic) or transudative (cardiac / hepatic / renal) — with or without mediastinal shift — with complications if any.

Light's criteria — exudate if any one is present

Criterion	Exudate
Pleural fluid protein ÷ serum protein	> 0.5
Pleural fluid LDH ÷ serum LDH	> 0.6
Pleural fluid LDH	> ⅔ upper limit of normal serum LDH

Causes

Type	Causes
Transudate	Congestive heart failure, cirrhosis, nephrotic syndrome, hypoalbuminaemia, hypothyroidism, peritoneal dialysis
Exudate	**Tuberculosis**, parapneumonic effusion / empyema, malignancy (lung, breast, lymphoma, mesothelioma), pulmonary embolism, SLE, rheumatoid arthritis, pancreatitis, subphrenic abscess

Investigations

Test	What to look for
Chest X-ray PA	Homogeneous opacity, obliterated costophrenic angle, meniscus sign; ≥200 mL needed on PA, ~50 mL on lateral; mediastinal shift
Ultrasound thorax	Confirms and quantifies fluid, loculations, septations; guides tapping
Diagnostic thoracentesis	Appearance; protein, LDH, glucose, pH, cell count and type, Gram stain, culture, AFB, CBNAAT, **ADA**, cytology for malignant cells
Pleural fluid ADA	>40 U/L with lymphocytic exudate — tuberculosis (in India)
Glucose and pH	Glucose <60 mg/dL — TB, empyema, rheumatoid, malignancy; pH <7.2 — complicated parapneumonic, needs drainage
Others	CBC, ESR, Mantoux / IGRA, sputum AFB, CT thorax after drainage, pleural biopsy (thoracoscopic), echo, LFT, RFT, urine protein

CLINICAL PEARL

A massive effusion with the trachea in the midline suggests collapse of the underlying lung — think of carcinoma bronchus blocking the main bronchus.

EXAMINER ASKS

Why is the trachea central in some large effusions? · What is Skodaic resonance? · Indications for intercostal drainage · Why remove only 1–1.5 L at a time (re-expansion pulmonary oedema) · Chylothorax and pseudochylothorax · Light's criteria and their limitation in patients on diuretics.

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Pneumonia / Lung Consolidation

Acute fever, cough and pleuritic pain — localise, grade severity and decide where to treat

Bio data

Name ..... **IP No.** .....

Age ..... **Sex** .....

Occupation .....

Birds (psittacosis), farm animals (Q fever), air-conditioning cooling towers (Legionella), sewage / paddy fields (leptospirosis)

Address .....

Age and sex

- Elderly — atypical presentation (confusion, falls, no fever) and higher mortality
- Young adult — pneumococcus, Mycoplasma; chronic alcoholic — Klebsiella, aspiration

Presenting complaints

1. .....
2. .....
3. .....

- Typical: fever with chills for ____ days, cough with rusty sputum for ____ days, right-sided chest pain on breathing for ____ days, breathlessness for ____ days

History of presenting illness

Fever

- Acute onset, high grade with **chills and rigors** — pneumococcal (lobar) pneumonia
- Gradual onset, low grade with headache, myalgia, dry cough — atypical pneumonia (Mycoplasma, Chlamydia, Legionella)
- Fever for >2–3 weeks with evening rise — tuberculosis, lung abscess

Cough and sputum

- Initially dry → productive; **rusty sputum** — pneumococcus
- Red currant-jelly sputum — Klebsiella; foul-smelling, copious — anaerobes / lung abscess
- Haemoptysis — TB, Klebsiella, lung abscess, carcinoma, pulmonary infarction

Chest pain

- Pleuritic, localised; upper abdominal pain in lower-lobe pneumonia (may mimic acute abdomen)
- Shoulder-tip pain — diaphragmatic pleurisy

Breathlessness

- Onset and progression; inability to speak in sentences — severe pneumonia

Associated symptoms

- Confusion (CURB-65 'C'); vomiting, diarrhoea (Legionella); jaundice (Legionella, leptospirosis); myalgia, conjunctival suffusion, oliguria (leptospirosis)

Risk factors

- Recent influenza or viral fever — secondary staphylococcal pneumonia
- Alcohol binge, seizure, stroke, vomiting, dental sepsis — **aspiration pneumonia**
- Hospitalisation in last 90 days, IV antibiotics — resistant organisms
- Diabetes, CKD, liver disease, steroids, HIV, splenectomy — immunosuppression
- Travel, contact with similar illness, pets and birds

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Past history

- Previous pneumonias (recurrent in the same lobe — bronchial obstruction: tumour or foreign body), TB, COPD, asthma, bronchiectasis
- Diabetes, heart failure, CKD, cirrhosis, HIV, malignancy, seizures, stroke with dysphagia

Personal history

- Smoking, alcohol, IV drug use (right-sided endocarditis with septic emboli); similar illness in family (TB, influenza)

Drug history

- Antibiotics already taken (name, dose, days — affects choice and cultures); steroids, immunosuppressants, PPIs
- Vaccination — pneumococcal, influenza, COVID-19

General examination

Built / nourishment ..... **Consciousness / orientation** .....

- Toxic look, flushed face, **herpes labialis**
- Cyanosis, pallor, icterus; clubbing (chronic suppurative disease — lung abscess, bronchiectasis, empyema)
- Dehydration, cold peripheries, hypotension — **sepsis / septic shock**
- Oral cavity — dental caries, gingivitis (aspiration, anaerobes); lymphadenopathy

Pulse ..... **BP** .....

Respiratory rate ..... **SpO_2** .....

Temperature ..... **Capillary refill** .....

PULSE–TEMPERATURE DISSOCIATION

Relative bradycardia with high fever suggests Legionella, typhoid or Mycoplasma pneumonia.

Respiratory system examination

Inspection

- Tachypnoea, flaring of alae nasi
- Diminished movement on the affected side
- No mediastinal shift

Palpation

- Trachea and apex **central**
- Expansion reduced on the affected side
- **Vocal fremitus increased**

Percussion

- **Woody dull** note over the consolidated area (not stony dull)
- Mark the lobe — anterior: upper and middle lobes; posterior: lower lobes; axilla: all

Auscultation

- **Bronchial breathing** (tubular)
- **Vocal resonance increased**, whispering pectoriloquy, aegophony
- **Coarse crepitations** — early (crepitations of consolidation) and late (resolution)
- Pleural rub over the involved area

Stages of lobar pneumonia

Stage	Signs
Congestion (day 1–2)	Diminished breath sounds, fine crepitations
Red hepatisation (day 2–4)	Dullness, bronchial breathing, increased VR, pleural rub
Grey hepatisation (day 4–8)	Same signs, patient often improving
Resolution (after day 8)	Coarse crepitations (crepitatio redux), breath sounds return to vesicular

Other systems

- CVS: murmurs (endocarditis) · Abdomen: hepatomegaly · CNS: meningism (pneumococcal meningitis)

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Summary

Diagnosis — how to write it

Diagnosis

FORMAT

Community-acquired (or hospital-acquired / aspiration) pneumonia — right / left — upper / middle / lower lobe — stage of consolidation — probable organism — severity (CURB-65 score ____) — with / without complications (parapneumonic effusion, empyema, respiratory failure, sepsis).

Severity — CURB-65 (one point each)

Feature	Interpretation
Confusion (new)	Score 0–1: low risk — treat at home
Urea >7 mmol/L (BUN >19 mg/dL; blood urea >42 mg/dL)	Score 2: moderate — short admission or close supervision
Respiratory rate ≥30/min	Score 3: severe — admit
BP systolic <90 or diastolic ≤60 mmHg	Score 4–5: very severe — consider ICU
Age ≥65 years	CRB-65 (without urea) for use in the clinic

Differential diagnosis

Condition	Distinguishing points
Pulmonary tuberculosis	Insidious, weeks of fever and weight loss, upper lobe, cavitation; sputum AFB / CBNAAT
Pleural effusion	Stony dull, absent fremitus and breath sounds, tracheal shift away
Collapse	Dull, trachea shifted **towards** the lesion, diminished breath sounds (if bronchus blocked)
Carcinoma lung with post-obstructive pneumonia	Smoker, slow resolution, recurrent in same lobe, haemoptysis, clubbing

Investigations

Test	What to look for
Chest X-ray PA and lateral	Homogeneous opacity in a lobar distribution, **air bronchogram**, silhouette sign (loss of right heart border — middle lobe; loss of hemidiaphragm — lower lobe)
CBC, CRP, procalcitonin	Neutrophil leucocytosis — bacterial; leucopenia — severe sepsis or viral
Sputum	Gram stain, culture and sensitivity, AFB, CBNAAT
Blood	Blood culture (before antibiotics), urea, creatinine, electrolytes, LFT, sugar, ABG
Special	Urinary pneumococcal and Legionella antigens; Mycoplasma serology; HIV; leptospira IgM / scrub typhus IgM in endemic areas

Management in one glance

- Oxygen to SpO_2 94–98% (88–92% in COPD), fluids, antipyretics
- Mild (home): amoxicillin, or a macrolide / doxycycline for atypicals
- Moderate–severe: IV β-lactam (ceftriaxone / co-amoxiclav) + macrolide; aspiration — add anaerobic cover. Repeat chest X-ray at 6 weeks in smokers >50 years to exclude carcinoma

EXAMINER ASKS

Difference between consolidation and collapse · What is air bronchogram? · Why is bronchial breathing heard over consolidation? · Causes of non-resolving pneumonia · Complications of pneumococcal pneumonia.

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CHAPTER 4 · LONG CASE · CVS

Mitral Stenosis (MS)

Rheumatic heart disease — breathlessness, palpitation, haemoptysis and the mid-diastolic rumble

Bio data

Name ..... **IP No.** .....

Age ..... **Sex** .....

Occupation .....

Physical activity at work helps grade NYHA class

Address .....

Overcrowding and poor housing — rheumatic fever

Age and sex

- Young adult, usually **female** (F : M ≈ 2 : 1); in India symptoms may begin in adolescence (**juvenile MS**)
- Pregnancy often unmasks MS (increased blood volume and heart rate)
- Elderly — degenerative mitral annular calcification

Presenting complaints

1. .....
2. .....
3. .....

- Typical: breathlessness on exertion for ____ years, palpitation for ____ months, cough with blood-stained sputum for ____ days, swelling of legs for ____ weeks

History of presenting illness

Breathlessness — the commonest symptom

- Exertional, slowly progressive; grade with **NYHA class** I–IV
- Orthopnoea, paroxysmal nocturnal dyspnoea — raised left atrial pressure
- Sudden worsening — onset of AF, pregnancy, infection, anaemia, fever (tachycardia shortens diastole)
- Acute breathlessness with frothy pink sputum — acute pulmonary oedema

Palpitation

- Irregular, fast — **atrial fibrillation**; duration of each episode, precipitating factors

Haemoptysis

- Blood-streaked sputum — winter bronchitis / pulmonary oedema
- Frank, large haemoptysis — rupture of bronchial veins (**pulmonary apoplexy**)
- With pleuritic pain — pulmonary infarction

Chest pain

- Right ventricular ischaemia due to pulmonary hypertension; associated coronary disease

Symptoms of complications

- Sudden weakness of limbs, speech difficulty, loss of vision — **systemic embolism** (stroke)
- Hoarseness — **Ortner's syndrome** (left recurrent laryngeal nerve compressed by the enlarged LA or pulmonary artery); dysphagia — LA compressing oesophagus
- Leg swelling, abdominal distension, right hypochondrial pain — right heart failure
- Fever, weight loss — **infective endocarditis** (less common in pure MS); fatigue — low cardiac output

History of rheumatic fever (ask specifically)

- Fever with **migratory polyarthritis** of large joints in childhood
- Abnormal involuntary movements (**Sydenham's chorea**), skin rash or nodules
- Sore throat in childhood; **penicillin prophylaxis** (benzathine penicillin every 3–4 weeks) — taken regularly?

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Past history

- Rheumatic fever, sore throats, previous admissions for heart failure, stroke
- Previous balloon mitral valvotomy (BMV / PTMC), closed or open mitral commissurotomy, valve replacement
- Tuberculosis, diabetes, hypertension, thyroid disease (thyrotoxicosis precipitates AF)

Treatment history

- Diuretics, β-blockers / digoxin (rate control), **anticoagulants** (warfarin — INR monitoring, bleeding), penicillin prophylaxis

Menstrual and obstetric history

- Pregnancies, worsening during pregnancy, contraception (avoid oestrogen in AF / thrombo-embolism)

Personal and family history

- Socio-economic status, overcrowding; diet (salt), sleep, bowel and bladder; rheumatic fever in siblings

General examination

Built / nourishment ..... **Decubitus** .....

- **Mitral facies** — malar flush, pinkish-purple patches over the cheeks (low cardiac output, peripheral cyanosis)
- Peripheral cyanosis; pallor (endocarditis); clubbing (endocarditis — rare)
- Signs of rheumatic activity — subcutaneous nodules, erythema marginatum
- Raised JVP (prominent **a wave** in pulmonary hypertension; absent a wave and prominent v wave in AF with TR)
- Pedal oedema, sacral oedema; tender hepatomegaly

Pulse rate, rhythm ..... **Pulse deficit (in AF)** .....

BP ..... **Respiratory rate** .....

- Pulse: low volume; **irregularly irregular** in AF — count the apical rate and radial rate (pulse deficit >10 in AF)

Cardiovascular system examination

Inspection

- Apex in normal position (left ventricle is spared)
- Left parasternal pulsation (RVH)
- Pulsations in the 2nd left space (pulmonary artery)

Palpation

- **Tapping apex beat** (palpable S1)
- **Diastolic thrill** at apex (left lateral position)
- **Left parasternal heave** — RVH
- Palpable P2 — pulmonary hypertension

Auscultation

- **Loud S1** (mobile valve; soft when calcified)
- **Opening snap** after S2 — mobile, pliable valve
- **Mid-diastolic murmur** at apex: low-pitched, rumbling, best heard with the **bell**, patient in **left lateral position**, after exercise, in expiration
- **Presystolic accentuation** (absent in AF)
- Loud P2, Graham Steell murmur (PR), pansystolic murmur of TR

SEVERITY

A short S2–OS gap (<80 ms) and a long-duration murmur mean severe MS. Loudness of the murmur does not indicate severity.

Respiratory system

- Basal crackles (pulmonary venous congestion), pleural effusion · Abdomen: tender liver, ascites

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Summary

Diagnosis — how to write it

Diagnosis

FORMAT

Chronic rheumatic heart disease — severe mitral stenosis — in sinus rhythm / atrial fibrillation — with pulmonary arterial hypertension — NYHA class ____ — with / without congestive cardiac failure, infective endocarditis, embolic complications — rheumatic activity present / absent.

Differential diagnosis of a mid-diastolic murmur at the apex

Condition	Distinguishing point
Mitral stenosis	Loud S1, opening snap, presystolic accentuation, tapping apex
Carey Coombs murmur	Acute rheumatic carditis — soft, short, no OS
Austin Flint murmur	Severe aortic regurgitation — no loud S1 or OS, collapsing pulse
Left atrial myxoma	Tumour plop, murmur changes with posture, fever, emboli
Flow murmur	Large VSD, PDA, severe MR — high-flow across normal valve

Investigations

Test	What to look for
ECG	**P mitrale** (broad bifid P in II), atrial fibrillation, right axis deviation, RVH
Chest X-ray	Straightening of left heart border, **double atrial shadow**, widened carina (splaying), Kerley B lines, prominent upper-lobe veins, enlarged pulmonary artery
Echocardiography	**Mitral valve area** (normal 4–6 cm^2; severe ≤1.5 cm^2), mean gradient, PA pressure, LA size and clot (TEE), **Wilkins score** for suitability of balloon valvotomy
Others	ASO titre, ESR, CRP (rheumatic activity); blood cultures (endocarditis); INR on warfarin

Severity of MS (echocardiography)

Grade	Valve area	Mean gradient
Mild	>1.5 cm^2	<5 mmHg
Severe	≤1.5 cm^2	>10 mmHg (at normal heart rate)
Very severe	≤1.0 cm^2	—

Management in one glance

- Penicillin prophylaxis (benzathine penicillin G 1.2 MU IM every 3 weeks) — till 40 years or lifelong in severe disease
- Diuretics, β-blockers / ivabradine to prolong diastole; AF — rate control and **warfarin** (DOACs not for rheumatic MS)
- Severe symptomatic MS with favourable valve — **percutaneous balloon mitral valvotomy**; otherwise valve replacement

EXAMINER ASKS

Why is S1 loud in MS? · What is the opening snap and what does the S2–OS gap tell you? · Why does presystolic accentuation disappear in AF? · Causes of haemoptysis in MS · Contraindications to balloon valvotomy · Jones criteria.

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Mitral Regurgitation (MR)

Pansystolic murmur at the apex — rheumatic, degenerative, ischaemic or functional

Bio data

Name ..... **IP No.** .....

Age ..... **Sex** .....

Occupation .....

Address .....

Age and sex

- Young — rheumatic MR, mitral valve prolapse (MVP), Marfan syndrome
- Middle-aged and elderly — ischaemic MR (after MI), degenerative MR, functional MR due to dilated LV

Presenting complaints

1. .....
2. .....
3. .....

- Typical: breathlessness on exertion for ____ years, palpitation for ____ months, easy fatigability for ____ months

History of presenting illness

Breathlessness

- Chronic MR: slowly progressive over years, NYHA class; orthopnoea and PND late (LV failure)
- **Acute MR** — sudden severe breathlessness, pulmonary oedema, shock: rupture of chordae, papillary muscle rupture after MI, infective endocarditis

Fatigue and weakness

- Prominent symptom — reduced forward cardiac output

Palpitation

- Forceful heartbeat — large stroke volume; irregular — **atrial fibrillation**

Chest pain

- Angina, past MI — ischaemic MR; atypical chest pain with palpitation, anxiety — MVP

Symptoms of complications

- Fever, weight loss, joint pains, rash, painful finger nodules — **infective endocarditis** (MR is more prone than MS)
- Stroke, limb ischaemia — embolism (in AF)
- Leg swelling, abdominal distension — right heart failure

Clues to the cause

- Childhood migratory polyarthritis, chorea, sore throats — **rheumatic**
- Chest pain, past heart attack, diabetes, smoking — **ischaemic**
- Tall stature, long fingers, lens dislocation, family history of sudden death — **Marfan syndrome**
- Joint hypermobility, skin laxity — Ehlers–Danlos syndrome
- Breathlessness with a large heart and no valve disease earlier — **dilated cardiomyopathy** (functional MR)
- Drugs: ergot derivatives, fenfluramine; SLE (Libman–Sacks endocarditis)

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Past history

- Rheumatic fever, MI, hypertension, diabetes, endocarditis, cardiac surgery or interventions (valve repair / replacement)

Treatment history

- Diuretics, ACE inhibitors / ARNI, β-blockers, digoxin, anticoagulants, penicillin prophylaxis

Personal and family history

- Smoking, alcohol (alcoholic cardiomyopathy); family history of MVP, Marfan syndrome, cardiomyopathy, sudden death

Menstrual and obstetric history (in women)

- Pregnancies and symptoms during pregnancy; contraception

General examination

Built / nourishment ..... **Decubitus** .....

- Marfanoid habitus (arm span > height, arachnodactyly, high-arched palate) · features of endocarditis (pallor, clubbing, splinter haemorrhages, Osler's nodes, Janeway lesions, Roth spots, splenomegaly)
- JVP — raised with prominent v wave in TR; pedal oedema
- Signs of rheumatic activity

Pulse ..... **BP** .....

Respiratory rate ..... **JVP** .....

- Pulse: **brisk / short duration** (rapid ejection into the LA); irregularly irregular in AF

Cardiovascular system examination

Inspection

- Apex displaced **downwards and outwards**
- Diffuse, hyperdynamic apical impulse

Palpation

- **Hyperdynamic (volume-overloaded) apex**, displaced to 6th space, outside midclavicular line
- **Systolic thrill** at apex
- Left parasternal lift — LA expansion in late systole or RVH
- Palpable P2 — pulmonary hypertension

Auscultation

- **Soft S1** (S1 merges with the murmur)
- **Pansystolic murmur** at the apex, high-pitched, blowing, best heard with the **diaphragm**, in **expiration**, radiating to the **axilla** (and back)
- **S3** — severe MR (rapid LV filling), even without failure
- Short **mid-diastolic flow murmur** — severe MR
- Wide split S2 (early A2); loud P2 in pulmonary hypertension

Mitral valve prolapse

- **Mid-systolic click** followed by a late systolic murmur; standing moves the click earlier and lengthens the murmur; squatting has the opposite effect

Respiratory system and abdomen

- Basal crackles, pleural effusion; tender hepatomegaly, ascites (right heart failure); splenomegaly (endocarditis)

Bedside manoeuvres for systolic murmurs

Manoeuvre	Effect
Sustained handgrip (↑ afterload)	MR and VSD murmurs **louder**; AS and HOCM softer
Squatting (↑ venous return and afterload)	MR louder; MVP click **later**, murmur shorter; HOCM softer
Standing / Valsalva strain (↓ venous return)	MVP click **earlier**, murmur longer; HOCM **louder**; most others softer
Deep inspiration	Right-sided murmurs (TR) louder — Carvallo's sign

CLINICAL PEARL

Severity of MR is judged by the size of the LV, an S3 and a diastolic flow murmur — not by the loudness of the pansystolic murmur. A very severe acute MR may have a short, soft murmur.

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Summary

Diagnosis — how to write it

Diagnosis

FORMAT

Chronic rheumatic heart disease (or degenerative / ischaemic / functional) — severe mitral regurgitation — sinus rhythm / atrial fibrillation — LV function — pulmonary hypertension — NYHA class ____ — with / without heart failure or infective endocarditis.

Differential diagnosis of a pansystolic murmur

Condition	Distinguishing point
Mitral regurgitation	Apex, radiates to axilla, louder in expiration, displaced hyperdynamic apex
Tricuspid regurgitation	Lower left sternal edge, **louder in inspiration** (Carvallo's sign), giant v waves, pulsatile liver
Ventricular septal defect	Left 3rd–4th space, harsh, radiates across the precordium, thrill, normal-sized LV in small VSD
Aortic stenosis (elderly)	Ejection systolic, carotid radiation, slow-rising pulse — can be confused when heard at apex (Gallavardin phenomenon)
HOCM	Ejection systolic, increases with Valsalva and standing, jerky pulse, double apical impulse

Signs of severe MR

- Displaced hyperdynamic apex, S3, mid-diastolic flow murmur, wide split S2, signs of pulmonary hypertension or LV failure

Investigations

Test	What to look for
ECG	P mitrale, AF, **LVH** (volume overload), Q waves of old MI
Chest X-ray	Cardiomegaly (LV and LA), pulmonary venous congestion; LA calcification in rheumatic disease
Echocardiography	Mechanism (rheumatic, prolapse, flail leaflet, ischaemic, functional), severity (vena contracta ≥0.7 cm, effective regurgitant orifice ≥0.4 cm², regurgitant volume ≥60 mL — severe), **LVEF**, LV end-systolic dimension, PA pressure
TEE	Before repair, endocarditis vegetations, LA clot
Others	Blood cultures ×3 if fever; troponin, coronary angiography in ischaemic MR

Management in one glance

- Penicillin prophylaxis (rheumatic); treat heart failure — diuretics, ACE inhibitor / ARNI, β-blocker; AF — rate control + anticoagulation
- Surgery (repair preferred over replacement) — symptomatic severe MR, or asymptomatic with **LVEF ≤60%** or **LVESD ≥40 mm**, new AF or PA systolic pressure >50 mmHg
- Transcatheter edge-to-edge repair (MitraClip) — high surgical risk

EXAMINER ASKS

Why is S1 soft in MR? · Why does S3 occur in MR without heart failure? · How do you differentiate MR from TR at the bedside? · Causes of acute MR · Why surgery before LVEF falls below 60%?

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CHAPTER 6 · LONG CASE · CVS

Aortic Stenosis (AS)

Angina, syncope and breathlessness on exertion with an ejection systolic murmur

Bio data

Name ..... **IP No.** .....

Age ..... **Sex** .....

Occupation .....

Address .....

Age — points to the cause

- <30 years — congenital AS (unicuspid valve)
- 40–60 years — **bicuspid aortic valve** with calcification; rheumatic AS (almost always with mitral disease)
- >65 years — **degenerative calcific (senile) AS** — the commonest cause worldwide; more in men

Presenting complaints

1. .....
2. .....
3. .....

- Typical: chest pain on exertion for ____ months, giddiness / blackout on exertion for ____ weeks, breathlessness on exertion for ____ months

History of presenting illness

THE CLASSIC TRIAD

Angina, syncope and heart failure (breathlessness) on exertion. Once symptoms appear, average survival without valve replacement is about 5 years with angina, 3 years with syncope and 2 years with heart failure.

Chest pain (angina)

- Exertional, retrosternal, relieved by rest — increased LV oxygen demand (LVH) with reduced coronary flow; associated coronary disease in the elderly

Syncope / presyncope

- **On exertion** — fixed cardiac output cannot rise while exercising muscles vasodilate
- At rest or sudden — arrhythmia (AF, VT, heart block from calcium extending into the conduction system)

Breathlessness

- Exertional, then orthopnoea, PND — LV diastolic and later systolic dysfunction
- Sudden worsening with palpitation — onset of AF (loss of atrial kick is poorly tolerated)

Other symptoms

- Reduced exercise tolerance, fatigue — often the earliest symptom; ask what the patient has stopped doing
- Gastrointestinal bleeding, melaena — **Heyde's syndrome** (angiodysplasia with acquired von Willebrand deficiency)
- Fever, weight loss — infective endocarditis
- Sudden weakness, visual loss — calcific embolism
- Sudden death in the family (HOCM — important differential)

Clues to the cause

- Murmur known since childhood — congenital / bicuspid valve
- Rheumatic fever, other valve disease — rheumatic
- Hypertension, diabetes, smoking, dyslipidaemia, CKD — degenerative calcific AS (same risk factors as atherosclerosis)

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Past history

- Rheumatic fever, hypertension, diabetes, ischaemic heart disease, CKD, previous cardiac surgery, coarctation repair (bicuspid valve association)

Treatment history

- Diuretics (cautious), antihypertensives, statins, antiplatelets; **vasodilators and nitrates** — may cause hypotension and syncope

Personal and family history

- Smoking, alcohol, diet; family history of bicuspid valve, HOCM or sudden cardiac death

General examination

Built / nourishment ..... **Decubitus** .....

- Usually no specific general signs; look for xanthelasma, arcus (dyslipidaemia), pallor (GI bleeding), signs of endocarditis
- Elfin facies, hypercalcaemia, learning difficulty — **Williams syndrome** (supravalvular AS)

Pulse ..... **BP (both arms)** .....

Respiratory rate ..... **JVP** .....

- Pulse: **low volume, slow rising (pulsus parvus et tardus)**, anacrotic notch; best felt at the **carotid**
- BP: **narrow pulse pressure** (systolic <100 in severe AS); normal BP does not exclude severe AS in the elderly
- Radiofemoral delay — coarctation of aorta with bicuspid valve

Cardiovascular system examination

Inspection and palpation

- Apex **heaving (pressure overload)**, sustained, minimally displaced until LV fails
- **Systolic thrill** in the aortic area (right 2nd space) and carotids — best felt with the patient leaning forward in expiration
- Double apical impulse (palpable S4)

Auscultation

- **Ejection systolic murmur**, harsh, crescendo–decrescendo, in the **aortic area**, radiating to both **carotids**; best heard leaning forward in held expiration
- **Soft or absent A2**; **reversed (paradoxical) splitting** of S2 in severe AS
- **S4** (stiff hypertrophied LV)
- **Ejection click** — bicuspid, mobile valve (absent when calcified)
- Early diastolic murmur of associated AR

Signs of severe AS

- Slow-rising, low-volume pulse; narrow pulse pressure; thrill; **late-peaking** murmur; soft or absent A2; reversed split S2; S4; signs of LV failure
- In heart failure the murmur may become **soft** (low flow) — do not underestimate severity

Other systems

- Lungs: basal crackles · Abdomen: hepatomegaly (late) · Fundus: hypertensive changes

Sites of left ventricular outflow obstruction

Site	Cause	Bedside clue
Valvular	Calcific, bicuspid, rheumatic	Ejection click (bicuspid), soft A2, slow-rising pulse
Supravalvular	Williams syndrome	BP higher in the right arm, elfin facies, no click
Subvalvular membrane	Congenital	Associated AR, no ejection click
Dynamic (HOCM)	Asymmetric septal hypertrophy	Jerky pulse, louder on Valsalva and standing

CLINICAL PEARL

Always feel the carotid before you listen. A slow-rising carotid with a thrill is severe AS until proved otherwise; a brisk carotid with the same murmur suggests sclerosis or HOCM.

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Summary

Diagnosis — how to write it

Diagnosis

FORMAT

Valvular aortic stenosis — severe — probably degenerative calcific / bicuspid / rheumatic — symptomatic (angina, syncope, heart failure) — sinus rhythm — LV function — with / without heart failure or infective endocarditis.

Differential diagnosis of an ejection systolic murmur in the aortic area

Condition	Distinguishing point
Valvular aortic stenosis	Slow-rising pulse, soft A2, thrill, radiation to carotids, ejection click (bicuspid)
Aortic sclerosis	Elderly, normal pulse and A2, no thrill, short murmur, no LVH — no obstruction
HOCM	Jerky pulse, murmur at left sternal edge, **louder on Valsalva / standing**, softer on squatting, no carotid radiation
Supravalvular AS	Williams syndrome; BP higher in right arm
Flow murmur	Anaemia, pregnancy, thyrotoxicosis, AR (increased stroke volume)

Investigations

Test	What to look for
ECG	**LVH with strain** (ST depression, T inversion in V5–V6), LA enlargement, LBBB, AV block
Chest X-ray	Normal heart size until failure, **post-stenotic dilatation** of ascending aorta, valve calcification on lateral view
Echocardiography	Valve morphology (bicuspid / calcific), peak velocity, mean gradient, **aortic valve area**, LVEF, LVH
Others	Coronary angiography before surgery (>40 years); CT for calcium score and TAVI planning; exercise test **contraindicated** in symptomatic severe AS

Severity of AS (echocardiography)

Grade	Peak velocity	Mean gradient	Valve area
Mild	2.0–2.9 m/s	<20 mmHg	>1.5 cm^2
Moderate	3.0–3.9 m/s	20–39 mmHg	1.0–1.5 cm^2
Severe	≥4.0 m/s	≥40 mmHg	<1.0 cm^2

Management in one glance

- Symptomatic severe AS — **aortic valve replacement** (surgical, or transcatheter TAVI in the elderly / high-risk); asymptomatic severe AS with LVEF <50% also qualifies
- Avoid vasodilators and strenuous exertion; treat hypertension cautiously; dental hygiene

EXAMINER ASKS

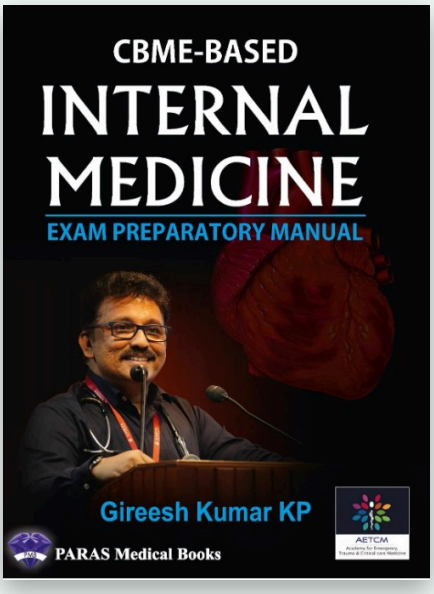
Why is the pulse slow rising? · Why syncope on exertion? · Aortic stenosis vs aortic sclerosis · Reversed splitting of S2 · Why does the murmur become soft in heart failure? · Gallavardin phenomenon.

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Chronic Liver Disease (Cirrhosis) with Ascites

Abdominal distension, jaundice, bleeding and confusion — cause, decompensation and complications

Bio data

Name ..... **IP No.** .....

Age ..... **Sex** .....

Occupation .....

Bartender, toddy tapper, healthcare worker (hepatitis B/C), sewage worker

Address .....

Area endemic for hepatitis B, schistosomiasis; source of drinking water

Age and sex

- Middle-aged man — **alcohol-related liver disease** (commonest in India), hepatitis B, MASLD (fatty liver with diabetes and obesity)
- Young — Wilson's disease, autoimmune hepatitis, hepatitis B; middle-aged woman with itching — primary biliary cholangitis

Presenting complaints

1. .....
2. .....
3. .....

- Typical: abdominal distension for ____ months, swelling of legs for ____ months, yellowish discoloration of eyes for ____ weeks, vomiting of blood / black stools for ____ days, altered behaviour for ____ days

History of presenting illness

Abdominal distension

- Onset and progression; began in the abdomen and then the legs — **ascites of liver disease** (in cardiac and renal disease, legs or face swell first)
- Abdominal pain with fever — **spontaneous bacterial peritonitis (SBP)**; umbilical hernia; breathlessness from tense ascites

Jaundice

- Onset, progression; pale stools and itching — cholestasis. Jaundice in cirrhosis means **decompensation** or superadded alcoholic / viral hepatitis

Gastrointestinal bleeding

- Haematemesis (fresh blood / coffee-ground), melaena — **variceal bleed**; number of episodes, transfusions, endoscopic banding

Altered sensorium — hepatic encephalopathy

- Sleep reversal, irritability, forgetfulness, confusion, drowsiness, coma (West Haven grade 1–4)
- Ask for precipitants: GI bleed, constipation, infection, diuretics, hypokalaemia, sedatives, high protein meal, dehydration

Other symptoms

- Fatigue, anorexia, muscle wasting (masked by fluid); bleeding gums, bruising, epistaxis — coagulopathy, low platelets
- Reduced urine output — **hepatorenal syndrome**; breathlessness — hepatic hydrothorax, hepatopulmonary syndrome
- Right upper abdominal pain, weight loss in a known cirrhotic — **hepatocellular carcinoma**

History to find the cause

- **Alcohol**: type, quantity in grams/day, duration (≥80 g/day for >10 years); last drink; withdrawal symptoms
- **Viral**: jaundice in the past, blood transfusion, injections, tattoos, IV drug use, unsafe sex, dialysis, family history of hepatitis B
- **Metabolic**: diabetes, obesity, dyslipidaemia (MASLD); tremors, behavioural change, family history (Wilson's)
- **Drugs**: methotrexate, amiodarone, isoniazid, herbal / Ayurvedic preparations
- **Cardiac**: valvular or constrictive pericardial disease — cardiac cirrhosis; loss of libido, amenorrhoea — hypogonadism

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Past history

- Previous admissions, paracentesis (number), endoscopy and banding, SBP, encephalopathy, hepatitis
- Diabetes, TB (TB peritonitis is an important cause of ascites), heart disease, kidney disease

Treatment history

- Diuretics (spironolactone, furosemide), propranolol / carvedilol, lactulose, rifaximin, albumin infusions, antiviral therapy

Personal history

- Alcohol (CAGE questions), smoking, diet and salt intake, appetite, sleep pattern, bowel habits (constipation)

Family history

- Liver disease, hepatitis B carriers, Wilson's disease, haemochromatosis

General examination

Built / nourishment .....

Consciousness / orientation .....

SIGNS OF LIVER CELL FAILURE (LOOK FOR ALL)

Icterus · **palmar erythema** · **spider naevi** (above the nipple line; >5 significant) · **gynaecomastia** · testicular atrophy · loss of axillary and pubic hair · Dupuytren's contracture and parotid enlargement (alcohol) · leukonychia (hypoalbuminaemia) · clubbing · **flapping tremor** (asterixis) · fetor hepaticus · bruises, petechiae · pedal oedema

- Pallor (bleeding, hypersplenism), muscle wasting, Kayser–Fleischer ring (Wilson's — slit lamp)
- Signs of portal hypertension: splenomegaly, dilated abdominal veins, ascites

Pulse .....

BP .....

Respiratory rate .....

Temperature .....

Abdominal examination

Inspection

- **Distended abdomen**, full flanks, **everted umbilicus**, umbilical hernia
- **Dilated veins** — flow **away from the umbilicus** (caput medusae; portal hypertension); flow from below upwards — IVC obstruction
- Divarication of recti, striae

Palpation

- Liver: in cirrhosis often **shrunken / not palpable**; if palpable — firm, sharp / nodular border; hard, irregular — HCC
- **Splenomegaly** — portal hypertension
- Dipping method (ballottement) to feel organs in tense ascites

Percussion

- **Shifting dullness** — moderate ascites (>500 mL)
- **Fluid thrill** — massive, tense ascites
- **Puddle sign** — small ascites (~120 mL), knee–elbow position
- Liver span by percussion (reduced in cirrhosis)

Auscultation

- Venous hum over the umbilicus (**Cruveilhier–Baumgarten**) — portal hypertension
- Bowel sounds; hepatic bruit — HCC, alcoholic hepatitis

Other systems

- RS: right-sided pleural effusion (hepatic hydrothorax) · CVS: hyperdynamic circulation · CNS: grade of encephalopathy, asterixis, constructional apraxia (cannot draw a star)

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Summary

Diagnosis — how to write it

Diagnosis

FORMAT

Decompensated chronic liver disease (cirrhosis) — probably alcohol-related / hepatitis B / MASLD — with portal hypertension (splenomegaly, ascites, varices) — Child–Pugh class ____ — complications (variceal bleed, hepatic encephalopathy grade ____, SBP, hepatorenal syndrome, HCC).

Child–Pugh score (A: 5–6, B: 7–9, C: 10–15)

Parameter	1 point	2 points	3 points
Bilirubin (mg/dL)	<2	2–3	>3
Albumin (g/dL)	>3.5	2.8–3.5	<2.8
INR	<1.7	1.7–2.3	>2.3
Ascites	None	Mild / controlled	Moderate–severe
Encephalopathy	None	Grade 1–2	Grade 3–4

Differential diagnosis of ascites

Cause	Clues
Cirrhosis with portal hypertension	Liver cell failure signs, splenomegaly, SAAG ≥1.1
Tuberculous peritonitis	Fever, weight loss, doughy abdomen, lymphocytic exudate, high ADA, SAAG <1.1
Malignancy (peritoneal carcinomatosis)	Weight loss, hard nodular masses, ovarian mass, blood-stained fluid, cytology
Congestive heart failure / constrictive pericarditis	Raised JVP, Kussmaul's sign, pericardial knock, SAAG ≥1.1 with high protein
Nephrotic syndrome	Facial puffiness first, frothy urine, heavy proteinuria, SAAG <1.1

Investigations

Test	What to look for
Ascitic tap	Appearance, **SAAG** (serum albumin – ascitic albumin; ≥1.1 g/dL = portal hypertension), total protein, cell count (**neutrophils ≥250/µL = SBP**), culture in blood-culture bottles, ADA, cytology
Blood	CBC (low platelets — hypersplenism), LFT, albumin, **INR**, urea, creatinine, Na^+, K^+, sugar
Aetiology	HBsAg, anti-HCV, autoimmune markers, ceruloplasmin, iron studies
Imaging	USG: coarse shrunken nodular liver, splenomegaly, portal vein >13 mm, collaterals, ascites, HCC; **AFP**; fibroscan
Endoscopy	Oesophageal and gastric varices, portal hypertensive gastropathy

Management in one glance

- Abstinence, salt restriction (<5 g/day), spironolactone ± furosemide (100 : 40 ratio); large-volume paracentesis with **albumin 8 g per litre removed** (>5 L)
- β-blocker for varices; lactulose ± rifaximin; SBP — cefotaxime + albumin; transplant (MELD)

EXAMINER ASKS

Why does ascites occur in cirrhosis? · Mechanism of spider naevi and gynaecomastia · SAAG and its use · Precipitating factors of hepatic encephalopathy · Why spironolactone first?

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Chronic Kidney Disease (CKD)

Puffiness, breathlessness, nausea and pallor — stage, cause, complications and need for dialysis

Bio data

Name ..... **IP No.** .....

Age ..... **Sex** .....

Occupation .....

Agricultural work in hot climate, pesticide exposure — CKD of unknown aetiology (CKDu)

Address .....

Uddanam (Andhra Pradesh) and other CKDu belts; source of drinking water

Age and sex

- Middle-aged and elderly — **diabetic nephropathy** (commonest), hypertensive nephrosclerosis; elderly man — obstructive uropathy
- Young — chronic glomerulonephritis, reflux nephropathy, polycystic kidney disease (ADPKD), lupus nephritis

Presenting complaints

1. .....
2. .....
3. .....

- Typical: swelling of face and legs for ____ months, decreased urine output for ____ weeks, loss of appetite with nausea and vomiting for ____ weeks, breathlessness for ____ days, easy fatigability for ____ months

History of presenting illness

Swelling

- Facial puffiness in the morning, later legs and abdomen; pitting, progressive

Urinary symptoms

- Oliguria (<400 mL/day) or anuria; **nocturia** (early loss of concentrating ability), polyuria in tubulointerstitial disease
- Frothy urine — proteinuria; red / smoky urine — glomerulonephritis; passage of stones, colicky pain
- Dysuria, frequency, fever — UTI; poor stream, hesitancy, dribbling — prostatic obstruction

Uraemic symptoms (ask each)

- Loss of appetite, **nausea, vomiting** (often in the morning), metallic taste, hiccups, ammoniacal breath
- Fatigue, weakness (anaemia), **pruritus**, restless legs, burning feet (neuropathy)
- Drowsiness, twitching, seizures — **uraemic encephalopathy**; gum / GI bleeding — platelet dysfunction
- Chest pain worse on lying down and relieved by leaning forward — **uraemic pericarditis**

Breathlessness

- Volume overload / pulmonary oedema; acidotic (Kussmaul) breathing; anaemia; pericardial effusion

Bone and mineral symptoms

- Bone pain, fractures, proximal muscle weakness — CKD–mineral bone disorder

History to find the cause

- **Diabetes**: duration, control, retinopathy (laser, vision loss), neuropathy — diabetic nephropathy is unlikely without retinopathy in type 1
- **Hypertension**: duration, treatment, control; young hypertensive — renal cause likely
- Swelling with red urine in childhood, sore throat or skin infection — **glomerulonephritis**
- Joint pains, rash, photosensitivity, oral ulcers — SLE
- Recurrent UTIs, stones, childhood bedwetting / reflux; flank mass — ADPKD
- **Drugs**: NSAIDs and painkillers (analgesic nephropathy), lithium, aminoglycosides, herbal / heavy-metal preparations

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Past history

- Diabetes, hypertension, TB (genitourinary TB), stones, surgery on urinary tract, previous AKI, contrast exposure
- Haemodialysis or peritoneal dialysis — since when, frequency, vascular access (AV fistula, catheter), complications
- Blood transfusions (sensitisation for transplant)

Treatment history

- Antihypertensives, insulin / OHAs (doses adjusted?), erythropoietin, iron, phosphate binders, vitamin D, sodium bicarbonate, diuretics, nephrotoxic drugs

Personal history

- Diet (salt, protein, potassium-rich foods, fluid intake), smoking, alcohol, sleep, appetite

Family history

- Kidney disease, dialysis, ADPKD (autosomal dominant), Alport syndrome (hearing loss), diabetes, hypertension

General examination

Built / nourishment

- **Pallor** (anaemia of CKD), sallow / lemon-yellow complexion (urochrome), uraemic frost (rare)
- Facial puffiness, **pedal and sacral oedema**, dry skin, scratch marks (pruritus)
- Bruises, petechiae; flapping tremor (uraemic), myoclonic jerks
- **AV fistula** at the wrist / elbow — thrill and bruit; dialysis catheter in neck; peritoneal catheter
- Raised JVP (overload, pericardial effusion), **Kussmaul breathing** (metabolic acidosis)
- Hearing loss and lens abnormalities (Alport)

Consciousness

Pulse

BP (lying and standing)

Respiratory rate

Weight / urine output

EXAMINE THE FUNDUS

Hypertensive and diabetic retinopathy support the cause and indicate end-organ damage.

Systemic examination

Abdomen

- Ballotable, bilateral, nodular **kidneys** — ADPKD (with liver cysts)
- Palpable bladder — obstructive uropathy; per rectal — enlarged prostate
- Renal bruit — renal artery stenosis
- Ascites, transplant kidney in the iliac fossa

CVS and RS

- Pericardial **rub**, muffled heart sounds — uraemic pericarditis
- Cardiomegaly, S3, S4, LVH (hypertension)
- Basal crackles, pleural effusion — fluid overload

CNS

- Peripheral neuropathy (glove-and-stocking), asterixis, altered sensorium

Common causes of CKD in India — bedside clues

Cause	Clue
Diabetic nephropathy	Long-standing diabetes, retinopathy, albuminuria first, normal-sized kidneys
Hypertensive nephrosclerosis	Long hypertension, LVH, hypertensive retinopathy, little proteinuria
Chronic glomerulonephritis	Young, haematuria, proteinuria, hypertension, small smooth kidneys
Chronic interstitial nephritis	Analgesic use, stones, UTIs, nocturia, polyuria, minimal proteinuria
ADPKD	Family history, bilateral flank masses, haematuria, hypertension, liver cysts
CKD of unknown aetiology (CKDu)	Young farm workers in hot belts, no diabetes or hypertension, low proteinuria

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Summary

Diagnosis — how to write it

Diagnosis

FORMAT

Chronic kidney disease — stage G____ A____ (eGFR ____ mL/min/1.73 m²) — probably due to diabetic nephropathy / hypertensive nephrosclerosis / chronic GN / ADPKD — with complications (anaemia, hyperkalaemia, metabolic acidosis, fluid overload, CKD–MBD, uraemic pericarditis) — on conservative management / haemodialysis.

KDIGO staging of CKD (CKD = abnormality of kidney structure or function for >3 months)

Stage	eGFR (mL/min/1.73 m²)	Albuminuria (ACR, mg/g)
G1	≥90 (normal or high)	A1 <30 (normal to mild)
G2	60–89	A2 30–300 (moderate)
G3a / G3b	45–59 / 30–44	A3 >300 (severe)
G4	15–29	
G5	<15 (kidney failure)	

CKD or AKI? — features that favour chronicity

- Small kidneys (<9 cm) with loss of corticomedullary differentiation on USG — **except** diabetes, amyloidosis, ADPKD, HIV nephropathy, myeloma (normal / large kidneys)
- Anaemia, hypocalcaemia with hyperphosphataemia, high PTH, renal osteodystrophy, broad casts, old creatinine values, long history of nocturia

Investigations

Test	What to look for
Renal function	Urea, creatinine, **eGFR** (CKD-EPI), Na^+, K^+, bicarbonate (ABG — metabolic acidosis), uric acid
Urine	Albumin–creatinine ratio, protein, RBC and casts, 24-h protein; culture
CBC and iron studies	Normocytic normochromic anaemia; ferritin, transferrin saturation
Mineral bone	Calcium (low), phosphate (high), ALP, **PTH** (high), vitamin D
Imaging	USG kidneys (size, echotexture, obstruction, cysts); avoid iodinated contrast
Others	HbA1c, lipids, HBsAg, anti-HCV, HIV, ECG (peaked T waves), echo; ANA, complement for GN

Indications for urgent dialysis — remember "AEIOU"

- **A**cidosis (pH <7.1) · **E**lectrolytes (refractory hyperkalaemia K^+ >6.5) · **I**ntoxication (dialysable drugs) · **O**verload (refractory pulmonary oedema) · **U**raemia (pericarditis, encephalopathy, bleeding)

Management in one glance

- Control BP (<130/80; ACE inhibitor / ARB if albuminuria), sugar; **SGLT2 inhibitor** to slow progression; avoid nephrotoxins; adjust drug doses
- Anaemia — iron, erythropoietin (Hb target 10–11.5 g/dL); phosphate binders, vitamin D; bicarbonate; low-salt diet
- Hepatitis B vaccination; AV fistula at eGFR <20–25; dialysis / transplant

EXAMINER ASKS

Causes of anaemia in CKD · Why are kidneys normal-sized in diabetic CKD? · Hyperkalaemia — ECG and treatment · CKD–MBD · Indications for dialysis.

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Nephrotic Syndrome

Massive proteinuria, hypoalbuminaemia, oedema and hyperlipidaemia — primary or secondary?

Bio data

Name ..... **IP No.** .....

Age ..... **Sex** .....

Occupation .....

Address .....

Age — points to the cause

- Child 2–6 years — **minimal change disease** (~80% of childhood nephrotic syndrome)
- Young adult — focal segmental glomerulosclerosis (FSGS), minimal change, lupus nephritis (women)
- Middle-aged / elderly — **membranous nephropathy** (look for malignancy), diabetic nephropathy, amyloidosis, myeloma

Presenting complaints

1. .....
2. .....
3. .....

- Typical: swelling of face for ____ weeks, swelling of legs and abdomen for ____ weeks, frothy urine for ____ weeks

History of presenting illness

Swelling (oedema)

- **Periorbital puffiness on waking**, settles during the day; then legs, abdomen (ascites), scrotum / vulva
- Generalised (**anasarca**) — gross hypoalbuminaemia; weight gain
- Breathlessness — pleural effusion, ascites splinting the diaphragm

Urine

- **Frothy urine** — heavy proteinuria
- Urine output — normal or reduced; **haematuria** (red / smoky urine) suggests a **nephritic** element (FSGS, MPGN, lupus, IgA)

Complications (ask each)

- Fever, abdominal pain, tenderness — **SBP / cellulitis** (loss of immunoglobulins; pneumococcal peritonitis in children)
- Calf pain, swelling of one leg, sudden breathlessness — **DVT, pulmonary embolism**
- Flank pain, haematuria — **renal vein thrombosis** (membranous nephropathy)
- Giddiness on standing, reduced urine — hypovolaemia, AKI
- Yellowish skin deposits (xanthelasma) — hyperlipidaemia

History to find a secondary cause

- **Diabetes** — duration, retinopathy
- **SLE** — joint pains, rash, photosensitivity, oral ulcers, hair loss
- **Infections** — hepatitis B and C, HIV, malaria (P. malariae), syphilis, post-streptococcal
- **Drugs** — NSAIDs, gold, penicillamine, lithium, interferon; skin-lightening creams (mercury)
- **Malignancy** — weight loss, lymph nodes, haemoptysis, bone pain (myeloma); lymphoma (Hodgkin — minimal change)
- **Amyloidosis** — chronic infections (TB, bronchiectasis), rheumatoid arthritis, macroglossia
- Recent vaccination, insect sting, atopy — minimal change disease

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Past history

- Previous episodes, relapses, steroid response (steroid-sensitive / dependent / resistant), renal biopsy result
- Diabetes, hypertension, TB, malaria, jaundice, joint disease

Treatment history

- **Steroids** (dose, duration, side effects — Cushingoid, diabetes, infections), cyclophosphamide, calcineurin inhibitors, rituximab
- Diuretics, ACE inhibitors / ARBs, statins, anticoagulants

Personal and family history

- Diet (salt, protein), smoking, alcohol; family history of renal disease (genetic FSGS, Alport)

General examination

Built / nourishment ..... **Weight** .....

- **Facial puffiness, pitting pedal oedema**, sacral oedema, scrotal oedema; **anasarca**
- Pallor (usually mild); **Muehrcke's lines** (paired white bands in nails — hypoalbuminaemia); xanthelasma
- Signs of secondary cause: malar rash, alopecia (SLE); diabetic skin changes; lymphadenopathy; macroglossia (amyloid)
- Signs of steroid use: moon face, striae, acne, buffalo hump
- Signs of complications: calf tenderness, unilateral leg swelling (DVT), cellulitis, skin breaks

Pulse ..... **BP (lying and standing)** .....

Respiratory rate ..... **Temperature** .....

Urine output (24 h) ..... **Abdominal girth** .....

- BP usually normal (minimal change); raised — FSGS, MPGN, diabetic, lupus nephritis

Systemic examination

Abdomen

- Distension, **ascites** (shifting dullness, fluid thrill)
- Hepatosplenomegaly — amyloidosis, lymphoma
- Kidneys not palpable (renal vein thrombosis — enlarged tender kidney)
- Tenderness — peritonitis

Respiratory system

- Bilateral **pleural effusions** (transudative)

CVS

- Pericardial effusion (rare), murmurs

Fundus

- Diabetic retinopathy; lipaemia retinalis (rare)

Mechanism of oedema — two theories

- **Underfill**: low albumin → low plasma oncotic pressure → fluid moves to interstitium → low effective volume → RAAS activation → salt retention
- **Overfill**: primary renal sodium retention (ENaC activation in collecting duct) — explains oedema with normal volume in adults

Primary causes — what the biopsy may show

Lesion	Typical patient	Clue and steroid response
Minimal change disease	Child; adult after NSAIDs / Hodgkin	Selective proteinuria, no haematuria, normal BP — **steroid-sensitive**
FSGS	Young adult, HIV, obesity, heroin	Haematuria, hypertension, reduced GFR — often steroid-resistant
Membranous nephropathy	Middle-aged / elderly	Anti-PLA2R positive (primary); renal vein thrombosis; look for cancer, hepatitis B, SLE
MPGN	Any age, hepatitis C	Low complement, nephritic features

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Summary

Diagnosis — how to write it

Diagnosis

FORMAT

Nephrotic syndrome — first episode / relapse — probably primary (minimal change / FSGS / membranous) or secondary (diabetic, lupus, amyloid, hepatitis B) — steroid-sensitive / resistant — with / without complications (infection, thrombosis, AKI, hyperlipidaemia).

Definition

- **Proteinuria >3.5 g/24 h** in adults (>40 mg/m²/h in children; urine protein–creatinine ratio >3.5 g/g)
- **Hypoalbuminaemia** (<3 g/dL) · **Oedema** · Hyperlipidaemia and lipiduria (oval fat bodies, Maltese crosses)

Nephrotic vs nephritic syndrome

Feature	Nephrotic	Nephritic
Proteinuria	>3.5 g/day	<3.5 g/day
Haematuria	Usually absent	Present — RBC casts
Hypertension	Usually absent	Present
Oedema	Massive, generalised	Mild, periorbital
Renal function	Usually normal	Reduced, oliguria
Example	Minimal change, membranous	Post-streptococcal GN, IgA, RPGN

Investigations

Test	What to look for
Urine	Dipstick protein 3+/4+, **24-hour protein / PCR**, microscopy (fat bodies, RBC casts if nephritic)
Blood	Serum albumin, cholesterol, triglycerides, urea, creatinine, electrolytes, CBC, sugar
Secondary causes	ANA, anti-dsDNA, C3, C4; HBsAg, anti-HCV, HIV; HbA1c; serum and urine protein electrophoresis, free light chains (>40 years); **anti-PLA2R antibody** (primary membranous)
Imaging	USG kidneys (size, renal vein thrombosis Doppler); chest X-ray (effusion)
Renal biopsy	All adults (unless clearly diabetic) and children who are steroid-resistant or atypical

Complications

- Infections (cellulitis, peritonitis), **thrombo-embolism** (loss of antithrombin III), hyperlipidaemia, AKI, hypovolaemia, protein malnutrition, vitamin D deficiency

Management in one glance

- Salt restriction, diuretics, **ACE inhibitor / ARB** (antiproteinuric), statin; anticoagulation if albumin <2–2.5 g/dL in membranous
- Minimal change: prednisolone 1 mg/kg/day; others as per biopsy (calcineurin inhibitors, rituximab, cyclophosphamide); treat secondary cause

EXAMINER ASKS

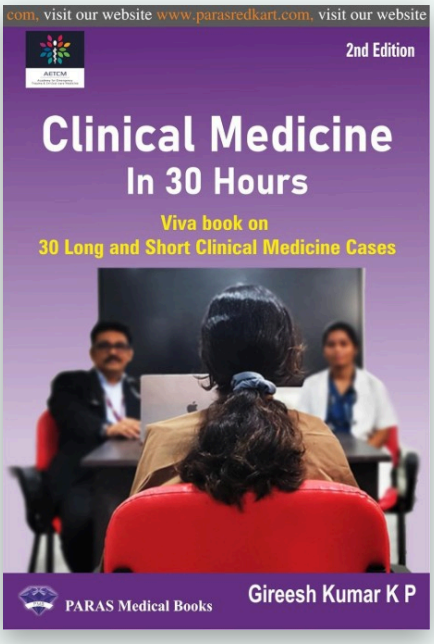
Why does thrombosis occur? · Why hyperlipidaemia? · Which nephrotic syndrome is steroid-sensitive? · Causes of secondary membranous nephropathy · Selective vs non-selective proteinuria · Indications for renal biopsy.

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Cerebrovascular Accident (Stroke)

Sudden focal deficit — time of onset, ischaemic or haemorrhagic, vascular territory and risk factors

Bio data

Name ..... **IP No.** .....

Age ..... **Sex** .....

Handedness .....

Right-handed — dominant left hemisphere; aphasia with right hemiplegia

Occupation / education .....

Needed to assess higher functions and plan rehabilitation

Address .....

Age

- >55 years — atherosclerotic ischaemic stroke, hypertensive intracerebral haemorrhage
- **Young stroke (<45 years)** — cardioembolism (rheumatic heart disease), cerebral venous thrombosis (postpartum, OCP), vasculitis, arterial dissection, TB meningitis, prothrombotic states, drugs

Presenting complaints

1. .....
2. .....
3. .....

- Typical: sudden weakness of right upper and lower limbs for ____ days, slurring of speech / inability to speak for ____ days, deviation of angle of mouth for ____ days

History of presenting illness

TIME OF ONSET

Note the exact time the patient was **last seen normal** — thrombolysis window 4.5 hours; thrombectomy up to 24 hours in selected patients.

Onset and evolution

- **Sudden**, maximal at onset — embolism; stepwise / evolving over hours — thrombosis; on waking — thrombotic (wake-up stroke)
- During exertion, straining or emotion with headache and vomiting — **haemorrhage**
- Gradual over days–weeks — think of tumour, subdural haematoma, abscess (not stroke)

Weakness

- Side, upper / lower limb, face; which is more affected (arm > leg — MCA; leg > arm — ACA); progression or recovery since onset

Speech, swallowing and vision

- Cannot speak or understand, words wrong — **aphasia** (dominant hemisphere); slurred — dysarthria
- Choking, nasal regurgitation — dysphagia (aspiration risk); double vision, vertigo, ataxia — **posterior circulation**
- Loss of half the visual field — hemianopia; sudden transient loss of vision in one eye (**amaurosis fugax**) — carotid disease

Sensory and other symptoms

- Numbness on the paralysed side; neglect (non-dominant parietal); incontinence, behavioural change
- **Headache, vomiting, loss of consciousness** at onset — haemorrhage (sudden worst headache — subarachnoid haemorrhage)
- Seizures at onset — haemorrhage, venous thrombosis, embolic cortical stroke

Previous transient episodes

- **TIA** — focal deficit lasting <24 h (usually minutes) with complete recovery

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Risk factors (ask each)

- **Hypertension**, **diabetes**, dyslipidaemia, smoking, alcohol, obesity, sedentary life
- **Heart**: palpitation (AF), rheumatic heart disease, recent MI, prosthetic valve, heart failure
- Previous stroke / TIA, peripheral vascular disease, migraine with aura, OCP, pregnancy / puerperium
- Snoring (sleep apnoea); polycythaemia, sickle cell disease; HIV, syphilis, TB

Past history

- Previous strokes (residual deficit), TIAs, MI, AF, valve disease, TB, seizures, head injury

Drug history

- Antiplatelets, **anticoagulants** (warfarin, DOACs — bleeding risk, time of last dose), antihypertensives, statins; adherence

Personal history

- Smoking (pack-years), alcohol, tobacco chewing, diet, physical activity, sleep, bowel and bladder

Family history

- Stroke, hypertension, diabetes, premature coronary disease, sudden death

General examination

Consciousness (GCS) ..... **Orientation** .....

- Pallor, cyanosis, clubbing (endocarditis, congenital heart disease), xanthelasma, arcus
- Signs of endocarditis; neck stiffness (SAH, meningitis); bed sores, aspiration

Pulse (rhythm — AF?) ..... **BP (both arms)** .....

Respiratory rate ..... **Temperature** .....

- **Carotid bruit**; peripheral pulses; fundus — hypertensive / diabetic retinopathy, papilloedema, emboli

CVS examination

- Irregular rhythm (AF), murmurs (MS, MR, endocarditis), cardiomegaly, prosthetic valve sounds

Nervous system examination

Higher mental functions

- Consciousness, orientation, memory, **speech**: fluent vs non-fluent, comprehension, repetition, naming — type of aphasia
- Neglect, apraxia, agnosia (parietal lobe)

Cranial nerves

- **UMN facial palsy** — lower face weak, forehead spared (bilateral cortical supply)
- Visual fields (homonymous hemianopia), gaze deviation (towards the lesion in cortical stroke; away in pontine), palatal movement, tongue deviation (towards the weak side)
- Brainstem: crossed cranial nerve palsy with contralateral hemiplegia

Motor system

- Bulk normal (wasting late, disuse)
- **Tone**: flaccid in the acute phase (cerebral shock) → **spastic**, clasp-knife later
- **Power** (MRC grade 0–5) in each group; pronator drift
- **Reflexes**: brisk, clonus; **extensor plantar** (Babinski)
- Abdominal reflexes lost on the paralysed side
- Posture: arm flexed, leg extended; **circumduction gait**

Sensory system, cerebellum, gait

- Hemianaesthesia (thalamus / internal capsule); cortical sensations (two-point, stereognosis) lost in parietal lesions
- Ataxia, nystagmus (cerebellar stroke); gait if possible

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Summary

Diagnosis — how to write it

Diagnosis

FORMAT

Acute / old cerebrovascular accident — ischaemic (thrombotic / embolic / lacunar) or haemorrhagic — in the territory of the left / right middle cerebral artery (or other) — causing right / left hemiplegia with UMN facial palsy and aphasia — day _____ — risk factors (hypertension, diabetes, AF, RHD) — complications.

Localisation by vascular territory

Artery	Features
Middle cerebral (MCA)	Contralateral hemiplegia and hemianaesthesia **face and arm > leg**, homonymous hemianopia, aphasia (dominant) or neglect (non-dominant), gaze towards the lesion
Anterior cerebral (ACA)	Contralateral weakness **leg > arm**, urinary incontinence, abulia, grasp reflex
Posterior cerebral (PCA)	Contralateral homonymous hemianopia with macular sparing, alexia without agraphia, thalamic pain
Vertebrobasilar	Vertigo, diplopia, dysarthria, dysphagia, ataxia, **crossed signs**, quadriparesis, coma
Lacunar (small vessel)	Pure motor, pure sensory, ataxic hemiparesis, dysarthria–clumsy hand; no cortical signs

Ischaemic vs haemorrhagic — clinical clues (CT is essential)

- Haemorrhage: onset during activity, severe headache, vomiting, early drop in consciousness, very high BP, neck stiffness, on anticoagulants
- Ischaemia: TIAs before, onset at rest or on waking, AF, carotid bruit, conscious patient with large deficit

Investigations

Test	What to look for
Non-contrast CT brain (urgent)	Haemorrhage (hyperdense) vs infarct (hypodense after 6–24 h); early signs — hyperdense MCA, loss of grey–white differentiation
MRI brain with DWI	Early ischaemia, posterior fossa and lacunar infarcts; MR / CT angiography for large-vessel occlusion
Blood	**Capillary glucose** (exclude hypoglycaemia), CBC, coagulation profile, lipids, HbA1c, urea, creatinine, electrolytes
Cardiac	**ECG, Holter** (AF), echocardiography (clot, vegetations, valve disease, PFO)
Vessels	**Carotid Doppler** — stenosis ≥70% symptomatic: endarterectomy
Young stroke	Vasculitis screen, antiphospholipid antibodies, homocysteine, protein C/S, VDRL, HIV, MR venography

Management in one glance

- ABC, glucose, swallow screen before oral feeding; BP — permit up to 220/120 unless thrombolysis (then <185/110)
- Ischaemic: **IV alteplase / tenecteplase within 4.5 h**; mechanical thrombectomy for large-vessel occlusion; aspirin 24 h after thrombolysis (or at once if not thrombolysed); statin; anticoagulation for AF (timing by infarct size)
- Haemorrhagic: BP control, reverse anticoagulation, neurosurgery when indicated; DVT prevention, physiotherapy, rehabilitation

EXAMINER ASKS

Why is the forehead spared in UMN facial palsy? · Why is tone flaccid initially? · Contraindications to thrombolysis · Lacunar syndromes · Causes of young stroke · TIA and ABCD² score.

CHAPTER 11 · LONG CASE · CNS

Hemiplegia

Weakness of one half of the body — localise the lesion along the corticospinal tract and find the cause

Bio data

Name ..... **IP No.** .....

Age ..... **Sex** .....

Handedness .....

Occupation .....

Address .....

Age and the cause

- Elderly — vascular (infarct, haemorrhage), tumour (glioma, metastasis), chronic subdural haematoma
- Young — embolism from RHD, venous thrombosis, demyelination (multiple sclerosis), tuberculoma, TB meningitis with arteritis, vasculitis, trauma
- Child — congenital (cerebral palsy), infections, moyamoya

Presenting complaints

1. .....

2. .....

- Typical: weakness of left upper and lower limbs for ____ months; difficulty in walking for ____ months

History of presenting illness

Mode of onset — the single most useful clue to the cause

Onset	Likely cause
Sudden (seconds–minutes)	Vascular — embolism, thrombosis, haemorrhage; also trauma
Acute (hours–days)	Infection — abscess, encephalitis; demyelination; venous thrombosis
Gradual (weeks–months)	**Tumour**, tuberculoma, chronic subdural haematoma
Episodic, recovers	TIA, Todd's palsy after seizure, hemiplegic migraine, hypoglycaemia

Progression and distribution

- Static / improving — vascular; progressive — space-occupying lesion
- Arm vs leg involvement; started in one limb and spread (march) — cortical tumour
- Face involved on the same side, on the opposite side (crossed), or spared — level of lesion

Symptoms that localise

- Cortical: seizures, aphasia, sensory inattention, visual field loss, behavioural change
- Brainstem: diplopia, vertigo, dysphagia, dysarthria, facial numbness on the opposite side
- Spinal cord (high cervical): no facial or speech involvement, neck pain, **bladder involvement**, sensory level

Symptoms of raised intracranial pressure

- Early-morning headache, projectile vomiting, blurring of vision — tumour, abscess, subdural haematoma

Clues to specific causes

- Fever, ear discharge, headache — **brain abscess**; fever, neck stiffness — TB meningitis
- Weight loss, cough, haemoptysis, breast lump — **metastasis**; past TB — tuberculoma
- Fall or head injury weeks earlier, alcoholic or on anticoagulants — **chronic subdural haematoma**
- Previous episodes of visual loss, weakness or numbness that recovered — **multiple sclerosis**
- Palpitation, RHD — embolism; postpartum headache with seizures — cortical venous thrombosis; HIV — toxoplasmosis, lymphoma

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Past, personal and family history

- Hypertension, diabetes, heart disease, TB, HIV, malignancy, seizures; previous strokes
- Smoking, alcohol; drug history (anticoagulants, OCP, antiepileptics); family history of stroke, neurocutaneous syndromes

Bladder and bowel

- Urgency, retention, incontinence — bilateral (cord, parasagittal) lesions; constipation

General examination

Consciousness ..... **Orientation** .....

- Pallor, fever, lymph nodes, neurocutaneous markers; ear (CSOM — abscess), breast (metastasis), endocarditis, carotid bruit

Pulse ..... **BP** .....

Respiratory rate ..... **Temperature** .....

- **Fundus** — papilloedema (raised ICP), optic atrophy (MS), hypertensive retinopathy

Nervous system examination

Higher functions and speech

- Aphasia (left hemisphere), neglect / anosognosia (right parietal), dementia (frontal tumour)

Cranial nerves — level of the lesion

- Same-side UMN facial palsy — lesion above the facial nucleus (cortex, internal capsule, upper midbrain)
- **Crossed hemiplegia**: cranial nerve palsy on the side of the lesion + hemiplegia on the opposite side — brainstem
- Visual fields, pupils, eye movements, palate, tongue; **no cranial nerve involvement** — spinal cord

Motor system (compare both sides)

Feature	Upper motor neuron	Comment in hemiplegia
Bulk	Normal / disuse atrophy	Wasting late
Tone	Spastic, clasp-knife	Flaccid in acute stage (neural shock)
Power	Pyramidal pattern	Extensors of arm and flexors of leg weaker
Reflexes	Exaggerated, clonus	Abdominal and cremasteric lost on affected side
Plantar	**Extensor**	Hoffmann's sign may be positive

Sensory system

- Pattern: whole half-body (thalamus, internal capsule), cortical sensation loss (parietal), **dissociated or crossed** (brainstem, Brown-Séquard)

Cerebellum, gait, spine and skull

- Ataxic hemiparesis (lacunar); **hemiplegic (circumduction) gait**
- Skull — bruit, tenderness, burr-hole scars; spine — deformity, tenderness

MRC grading of muscle power

Grade	Meaning
0	No contraction
1	Flicker or trace of contraction
2	Active movement with gravity eliminated
3	Active movement against gravity
4	Against gravity and some resistance (4−, 4, 4+)
5	Normal power

DETECT SUBTLE WEAKNESS

Pronator drift with eyes closed, slowed finger tapping, inability to hop on one leg and a reduced arm swing may be the only signs of a mild hemiparesis.

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Summary

Diagnosis — how to write it

Diagnosis

FORMAT

Right / left hemiplegia — spastic / flaccid — with or without UMN facial palsy and aphasia — lesion at the level of cortex / internal capsule / brainstem / high cervical cord — of vascular / infective / neoplastic / demyelinating origin.

Localisation of hemiplegia

Level	Features
Cortex	Monoplegia or face–arm weakness, aphasia (dominant), cortical sensory loss, seizures, hemianopia
Internal capsule	**Dense hemiplegia** of face, arm and leg equally, hemianaesthesia, hemianopia; no cortical signs
Midbrain — Weber	Ipsilateral **III nerve palsy** + contralateral hemiplegia
Pons — Millard–Gubler	Ipsilateral **VI and VII (LMN)** palsy + contralateral hemiplegia
Medulla — medial (Dejerine)	Ipsilateral **XII** palsy + contralateral hemiplegia and loss of posterior column sensation
High cervical cord	Hemiplegia sparing the face; Brown-Séquard — ipsilateral weakness and posterior column loss, contralateral pain and temperature loss

Differential diagnosis by cause

Cause	Pointers
Vascular	Sudden onset, risk factors, improving course
Space-occupying lesion	Gradual, progressive, headache, vomiting, papilloedema, seizures
Infection	Fever, CSOM, meningism, immunosuppression — abscess, tuberculoma, toxoplasmosis
Demyelination	Young woman, relapses and remissions, optic neuritis, internuclear ophthalmoplegia
Trauma / subdural	Head injury, fluctuating consciousness, alcohol, anticoagulants
Others	Todd's palsy (after seizure), hypoglycaemia, hemiplegic migraine — transient, recover fully

Investigations

- **CT / MRI brain** (with contrast if SOL, infection or demyelination suspected); MRI spine for cord lesions
- CBC, ESR, glucose, lipids, renal function, ECG, echo, carotid Doppler (vascular); chest X-ray (TB, metastasis)
- CSF (after imaging excludes mass effect) — TB meningitis, MS (oligoclonal bands); HIV, VDRL; biopsy for tumour

CLINICAL PEARL

Hemiplegia with a cranial nerve palsy on the opposite side is a brainstem lesion — the cranial nerve tells you the level, the side of the cranial nerve tells you the side of the lesion.

EXAMINER ASKS

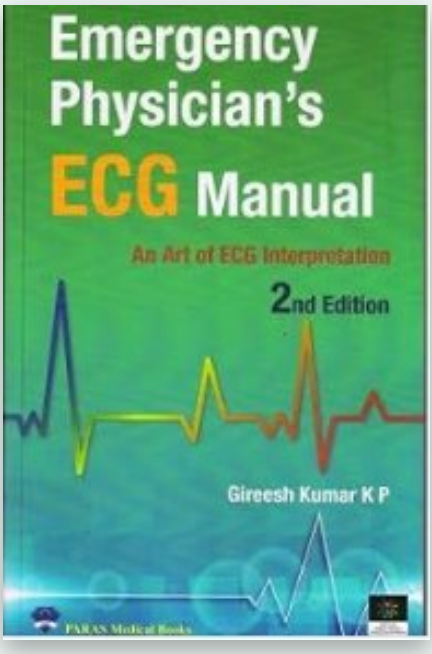
Why is the face spared in a cord lesion? · Clasp-knife vs lead-pipe rigidity · Why are abdominal reflexes lost? · Pure motor hemiplegia · Wernicke's vs Broca's aphasia · Why does the arm flex and the leg extend?

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CHAPTER 12 · SHORT CASE · CVS

Hypertension Assessment

Measure BP correctly, grade it, look for target-organ damage and a secondary cause

THE INSTRUCTION

"Check this patient's blood pressure", "Examine this hypertensive patient" or "Look at the fundus". You are expected to measure BP properly, grade it, and then look for target-organ damage and secondary causes.

Bio data

Name ..... **Age / Sex** .....

Occupation .....

Focused history

Hypertension

- Duration, how detected (screening / symptoms), highest recorded BP, treatment and **adherence**, home BP readings
- Symptoms: headache (occipital, early morning), giddiness, epistaxis, blurred vision — most patients have **no symptoms**

Target-organ damage

- **Heart**: exertional breathlessness, orthopnoea, chest pain, palpitation
- **Brain**: transient weakness, speech or visual disturbance (TIA / stroke), memory loss
- **Kidney**: nocturia, frothy urine, swelling; **Eyes**: blurred or reduced vision
- **Legs**: pain on walking (claudication) — peripheral arterial disease

Clues to secondary hypertension (ask each)

- Age <30 years, sudden onset, resistant hypertension (≥3 drugs including a diuretic)
- **Renal**: swelling, haematuria, UTIs, family history of polycystic kidneys
- **Phaeochromocytoma**: episodic headache, sweating, palpitation, pallor (paroxysms)
- **Primary aldosteronism**: muscle weakness, cramps, polyuria (hypokalaemia)
- **Cushing's**: weight gain, purple striae, easy bruising, steroid use
- **Thyroid**: heat or cold intolerance, weight change; **sleep apnoea**: loud snoring, daytime sleepiness
- **Coarctation**: leg claudication, cold feet in a young person
- **Drugs**: NSAIDs, steroids, OCP, decongestants, erythropoietin, cyclosporine, alcohol, liquorice

Cardiovascular risk factors

- Diabetes, smoking, dyslipidaemia, obesity, family history of premature heart disease, salt intake, physical inactivity

General examination

Built ..... **BMI / waist circumference** .....

- **Cushingoid** features (moon face, striae, buffalo hump), **acromegaly** (large hands, coarse face), thyroid swelling
- Neurofibromas, café-au-lait spots (phaeochromocytoma), pallor and puffiness (renal), xanthelasma
- Radio-femoral delay, weak femoral pulses (coarctation); all peripheral pulses and bruits

Pulse (rate, rhythm, radio-femoral delay) ..... **Pallor / puffiness** .....

Drug and personal history

- Antihypertensives — names, doses, timing, side effects (ankle swelling — amlodipine; cough — ACE inhibitor; gout — thiazide)
- Salt intake (pickles, papads, dried fish), alcohol, smoking, tobacco, physical activity, stress, sleep
- Family history of hypertension, early stroke or MI, kidney disease

CLINICAL PEARL

Hypertension in a patient under 30, or with hypokalaemia, a renal bruit or BP that was controlled and suddenly is not — search for a secondary cause.

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Measuring blood pressure — step by step

1. Patient seated for **5 minutes**, back supported, feet on the floor, no caffeine, smoking or exercise for 30 minutes, bladder empty
2. Arm supported at **heart level**; bare arm; correct cuff — bladder covers **80% of arm circumference** (large cuff for obese arm; small cuff overestimates BP)
3. **Palpatory method first**: inflate until the radial pulse disappears — note the level — avoids missing an **auscultatory gap**
4. Inflate 20–30 mmHg above the palpatory level; deflate at **2 mmHg per second** with the diaphragm over the brachial artery
5. **Systolic** — first Korotkoff sound (phase I); **diastolic** — disappearance of sounds (phase V; phase IV if sounds persist to zero, as in pregnancy, AR, thyrotoxicosis)
6. Measure in **both arms** at first visit (use the higher arm thereafter); difference >10–15 mmHg — subclavian stenosis, coarctation, dissection
7. Take **two or more readings** 1–2 minutes apart and average them
8. Lying and **standing** BP (after 1 and 3 minutes) — postural hypotension: fall ≥20 systolic / ≥10 diastolic (elderly, diabetics, on treatment)
9. Lower-limb BP (thigh cuff, popliteal artery) in the young — normally 10–20 mmHg higher than the arm; lower in coarctation

ERRORS TO AVOID

Small cuff, arm below heart level, talking, a single reading, rapid deflation, missing the auscultatory gap, terminal digit preference (readings ending in 0).

Examination for target-organ damage

Heart

- Apex — **heaving (pressure overload)**, displaced if LV dilated; **loud A2**, **S4**; early diastolic murmur (AR from aortic root dilatation); signs of heart failure

Fundus (dilate if necessary)

- Arteriolar narrowing, AV nipping, haemorrhages, cotton-wool spots, hard exudates, **papilloedema** (malignant hypertension)

Kidney and abdomen

- **Renal bruit** (renal artery stenosis — para-umbilical, epigastric, flanks); ballotable kidneys (ADPKD); aortic aneurysm

Brain and limbs

- Residual hemiparesis, cognitive impairment; peripheral pulses, carotid bruits, ankle-brachial index

Other signs

- Radio-radial and radio-femoral delay; thyroid; urine dipstick for protein and blood

Fundoscopy — how to do it

1. Darkened room; patient looks at a distant point; dilate the pupil if needed (avoid in suspected angle-closure glaucoma)
2. Your right eye for the patient's right eye, ophthalmoscope in your right hand; start at +10 D, 30 cm away — look for the **red reflex**
3. Come close, reduce the lens power to focus; follow a vessel to the **optic disc** — colour, margins, cup–disc ratio
4. Follow each of the four arcades — **arteries** (thinner, brighter) vs veins; AV crossings; then the **macula** (ask the patient to look at the light)

Signs of a secondary cause on examination

Cause	Signs
Renal parenchymal disease	Pallor, puffiness, oedema, ballotable kidneys (ADPKD), proteinuria
Renovascular	**Abdominal (renal) bruit**, other atherosclerotic disease, flash pulmonary oedema
Coarctation of aorta	**Radio-femoral delay**, weak femorals, BP arms > legs, scapular collaterals, ejection murmur at the back
Cushing's syndrome	Moon face, buffalo hump, purple striae, proximal myopathy, bruising
Phaeochromocytoma	Neurofibromas, café-au-lait spots, pallor and tremor during attacks, postural drop
Endocrine others	Acromegaly (coarse face, large hands), hypothyroidism, hyperthyroidism (wide pulse pressure)

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How to present

EXAMPLE

"This 52-year-old man has a blood pressure of 168/104 mmHg in the right arm and 164/102 in the left arm, sitting, with no postural drop — grade 2 hypertension. He has a heaving apex in the 5th space, a loud A2 and an S4 suggesting LVH, and grade II hypertensive retinopathy. There are no features of a secondary cause. My diagnosis is essential hypertension, grade 2, with target-organ damage to the heart and eye."

Grading of blood pressure (office BP, adults — ISH / ESH)

Category	Systolic (mmHg)	Diastolic (mmHg)
Normal	<130	<85
High-normal	130–139	85–89
Grade 1 hypertension	140–159	90–99
Grade 2 hypertension	≥160	≥100
Isolated systolic hypertension	≥140	<90

DIAGNOSTIC THRESHOLD

Indian guidelines and ISH 2020 diagnose hypertension at ≥140/90 mmHg; ACC/AHA 2017 uses ≥130/80. Confirm with home BP (≥135/85) or 24-hour ambulatory BP (≥130/80).

Hypertensive retinopathy (Keith–Wagener–Barker)

Grade	Fundus
I	Mild generalised arteriolar narrowing, increased light reflex (copper wiring)
II	Definite narrowing with focal constriction and **AV nipping**
III	Grade II + flame haemorrhages, **cotton-wool spots**, hard exudates
IV	Grade III + **papilloedema** — malignant (accelerated) hypertension

Hypertensive emergency vs urgency

- **Emergency**: BP usually >180/120 **with acute target-organ damage** — encephalopathy, stroke, acute LVF, ACS, aortic dissection, AKI, eclampsia — lower BP with IV drugs in ICU (by ~25% in the first hour)
- **Urgency**: severe BP without acute organ damage — oral drugs, lower over 24–48 hours

Basic investigations

- Urine (protein, blood, ACR), creatinine and eGFR, **K^+** (low in aldosteronism), Na^+, glucose, lipids, Hb, **ECG** (LVH — Sokolow–Lyon SV1 + RV5/V6 >35 mm), echo, fundus
- For secondary causes: renin–aldosterone ratio, plasma metanephrines, renal Doppler, TSH, overnight dexamethasone test, sleep study

Treatment in one glance

- Lifestyle for all: salt <5 g/day, weight loss, DASH diet, exercise, stop smoking, limit alcohol
- Drugs: **ACE inhibitor / ARB, calcium-channel blocker (amlodipine), thiazide-like diuretic** — start with two in a single pill if BP ≥160/100
- Target <130/80 for most adults (<140/90 in frail elderly); β-blockers for angina, post-MI, heart failure, AF

EXAMINER ASKS

What is the auscultatory gap? · Why use the palpatory method first? · Pseudohypertension and Osler's manoeuvre · White-coat vs masked hypertension · Causes of resistant hypertension · Which drugs are first line?

CHAPTER 13 · SHORT CASE · CVS

Mitral and Aortic Murmurs

"Listen to this patient's heart" — time the murmur, find its site, radiation and character, and name the lesion

THE INSTRUCTION

"Examine the precordium" or "Auscultate this patient's heart". Feel the pulse first, then inspect, palpate and auscultate — never go straight to the stethoscope.

Bio data

Name ..... **Age / Sex** .....

Focused history (if permitted)

- Breathlessness (NYHA class), orthopnoea, PND, palpitation, chest pain, syncope, haemoptysis
- Rheumatic fever in childhood, penicillin prophylaxis; fever and weight loss (endocarditis)
- Previous valve surgery or balloon valvotomy; anticoagulants

General examination — clues before you listen

Finding	Suggests
Malar flush	Mitral stenosis
Low-volume, slow-rising pulse	Aortic stenosis
Collapsing (water-hammer) pulse, wide pulse pressure	Aortic regurgitation
Irregularly irregular pulse	Atrial fibrillation — MS, MR
Prominent carotid pulsations (Corrigan's), head nodding (de Musset's)	Aortic regurgitation
Clubbing, splinter haemorrhages, Osler's nodes	Infective endocarditis
Marfanoid habitus, high-arched palate	MVP, aortic root dilatation, AR
Raised JVP with giant v waves	Tricuspid regurgitation
Sternotomy / thoracotomy scar	Valve surgery, closed mitral valvotomy (left lateral thoracotomy)
Metallic click audible without stethoscope	Prosthetic valve

Peripheral signs of aortic regurgitation (wide pulse pressure)

- **Corrigan's** (dancing carotids), **de Musset's** (head nodding), **Quincke's** (capillary pulsation in nail bed), **Traube's** (pistol-shot femorals), **Duroziez's** (to-and-fro femoral murmur on compression), **Hill's** sign (popliteal systolic BP >20–60 mmHg higher than brachial), Müller's sign (uvula pulsation)

Inspection and palpation of the precordium

- Precordial bulge (cardiomegaly in childhood), scars, visible apical impulse, pulsations
- **Apex beat**: position (normal: 5th left intercostal space, just inside the midclavicular line) and character

Apex character	Lesion
Tapping (palpable S1)	Mitral stenosis
Heaving, sustained, not displaced	Aortic stenosis, hypertension (pressure overload)
Hyperdynamic, displaced down and out	MR, AR (volume overload)

- Parasternal heave (RVH — pulmonary hypertension); palpable P2; thrills (systolic / diastolic — area)

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Auscultation — the routine

1. Listen in all four areas with the **diaphragm** and **bell**: mitral (apex), tricuspid (lower left sternal edge), pulmonary (2nd left space), aortic (2nd right space) and **Erb's point** (3rd left space)
2. Time the murmur against the **carotid pulse** (systolic murmurs coincide with the carotid upstroke)
3. Identify S1 and S2 — intensity, splitting; added sounds: S3, S4, opening snap, ejection click
4. Describe each murmur: **timing, site of maximum intensity, grade, character, pitch, radiation, and variation with respiration and posture**
5. Special positions: **left lateral** with the bell for MS; **sitting up, leaning forward in held expiration** with the diaphragm for AR (left sternal edge) and AS
6. Dynamic manoeuvres: inspiration (right-sided murmurs louder), Valsalva, squatting, handgrip

Grading of murmurs (Levine)

Grade	Description
1	Very faint, heard only with effort
2	Faint but readily heard
3	Moderately loud, **no thrill**
4	Loud, **with thrill**
5	Very loud, heard with stethoscope partly off the chest
6	Heard with stethoscope off the chest

The four classic valve lesions

Lesion	Murmur	Site / radiation	Associated findings
MS	Mid-diastolic rumble, presystolic accentuation	Apex, localised; bell, left lateral	Loud S1, opening snap, tapping apex, malar flush, AF
MR	Pansystolic, blowing	Apex → axilla	Soft S1, S3, displaced hyperdynamic apex
AS	Ejection systolic, harsh, crescendo–decrescendo	Aortic area → carotids	Slow-rising pulse, soft A2, heaving apex, thrill
AR	Early diastolic, high-pitched, decrescendo	Left sternal edge (3rd–4th space); sitting forward, expiration	Collapsing pulse, wide pulse pressure, Austin Flint murmur, displaced apex

CLINICAL PEARL

An early diastolic murmur heard best at the **right** sternal edge suggests aortic root disease (Marfan syndrome, syphilis, dissection) rather than valve-leaflet disease.

Mixed and multivalvular disease — which lesion dominates?

- **MS vs MR**: dominant MS — tapping apex, loud S1, normal-sized LV; dominant MR — displaced hyperdynamic apex, soft S1, S3
- **AS vs AR**: dominant AS — slow-rising pulse, narrow pulse pressure; dominant AR — collapsing pulse, wide pulse pressure, displaced apex
- Rheumatic **mitral + aortic** disease is common; isolated aortic disease in an old patient — degenerative or bicuspid

Splitting of the second heart sound

Type	What you hear	Causes
Normal (physiological)	Split on inspiration, single on expiration	Normal
Wide	Split in both phases, wider on inspiration	RBBB, pulmonary stenosis, MR (early A2)
Fixed wide	Same in both phases	**Atrial septal defect**
Reversed (paradoxical)	Split on expiration, single on inspiration	LBBB, severe AS, HOCM, RV pacing
Single S2	No split	Severe AS (absent A2), pulmonary atresia, elderly

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How to present

EXAMPLE

"The pulse is 88/min, irregularly irregular, with a pulse deficit of 12. The apex is in the 5th space in the midclavicular line and is tapping. There is a left parasternal heave. On auscultation S1 is loud, P2 is loud and there is an opening snap followed by a mid-diastolic rumbling murmur at the apex, best heard in the left lateral position with the bell, without presystolic accentuation. My diagnosis is chronic rheumatic heart disease with severe mitral stenosis, atrial fibrillation and pulmonary hypertension."

Differential diagnosis of murmurs

Murmur	Causes
Ejection systolic	AS, aortic sclerosis, HOCM, pulmonary stenosis, ASD (flow across pulmonary valve), **flow murmurs** (anaemia, pregnancy, fever, thyrotoxicosis)
Pansystolic	MR, TR, VSD
Late systolic	Mitral valve prolapse, papillary muscle dysfunction
Early diastolic	AR, pulmonary regurgitation (Graham Steell in pulmonary hypertension)
Mid-diastolic	MS, TS, Austin Flint (AR), Carey Coombs (rheumatic carditis), flow murmur (VSD, PDA, ASD, severe MR)
Continuous	PDA (machinery murmur), AV fistula, ruptured sinus of Valsalva, venous hum

Innocent murmur — features

- Soft (grade ≤2), ejection systolic, at the left sternal edge, no thrill, normal heart sounds, varies with posture, no cardiomegaly, asymptomatic

Bedside signs of severity

Lesion	Signs of severe disease
MS	Short S2–OS gap, long murmur, signs of pulmonary hypertension (loud P2, RV heave), right heart failure
MR	Displaced hyperdynamic apex, S3, mid-diastolic flow murmur, wide split S2
AS	Slow-rising low-volume pulse, narrow pulse pressure, thrill, soft / absent A2, reversed split, late-peaking murmur
AR	Wide pulse pressure (diastolic <50 mmHg), long murmur, Austin Flint murmur, displaced apex, signs of LVF

REVISED JONES CRITERIA (2015)

For moderate / high-risk populations such as India — evidence of preceding streptococcal infection + 2 major, or 1 major + 2 minor. **Major**: carditis (clinical or subclinical on echo), arthritis (polyarthritis, monoarthritis or polyarthralgia), chorea, erythema marginatum, subcutaneous nodules. **Minor**: monoarthralgia, fever ≥38 °C, ESR ≥30 mm/h and / or CRP ≥3 mg/dL, prolonged PR interval.

Investigations

- **ECG** (LVH, RVH, P mitrale, AF), **chest X-ray** (chamber enlargement, pulmonary congestion, valve calcification), **echocardiography** (diagnosis, severity, LV function), blood cultures if fever, ASO titre and ESR (rheumatic activity)

EXAMINER ASKS

How do you time a murmur? · Why is AR best heard leaning forward? · Mechanism of the Austin Flint murmur · Causes of a collapsing pulse · Splitting of S2 — normal, wide, fixed, reversed · Which murmurs increase with inspiration?

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CHAPTER 14 · SHORT CASE · CVS

Peripheral Vascular Disease

Claudication, rest pain and the pulseless limb — level of block and limb viability

THE INSTRUCTION

"Examine this patient's lower limbs" or "Examine the peripheral pulses". Look for ischaemia, feel all pulses on both sides, and find the level of arterial occlusion.

Bio data

Name ..... **Age / Sex** .....

Occupation .....

Focused history

Pain

- **Intermittent claudication**: cramping pain in a muscle group on walking a fixed distance, relieved by **standing still** for a few minutes — note the **claudication distance**
- Site of pain indicates the level: **calf** — femoropopliteal; **thigh and buttock** — aorto-iliac; foot (instep) — tibial vessels or Buerger's disease
- **Rest pain**: severe burning pain in the toes / forefoot, worse at night and on elevating the leg, relieved by **hanging the leg down** — critical limb ischaemia
- Pain with tingling relieved by bending forward, walking uphill easier — **neurogenic (spinal) claudication**

Other symptoms

- Ulcers, gangrene (black toes), coldness, numbness, colour change; non-healing wounds after trivial injury
- **Leriche syndrome**: buttock claudication + erectile dysfunction + absent femoral pulses (aorto-iliac occlusion)
- Sudden pain, pallor, paralysis — **acute limb ischaemia** (embolism from AF / MI, thrombosis)
- Raynaud's phenomenon, migratory superficial thrombophlebitis — **Buerger's disease**

Risk factors

- **Smoking** (beedi / cigarette — strongest; essential in Buerger's), **diabetes**, hypertension, dyslipidaemia, CKD, family history
- Coronary disease (angina, MI) and stroke — atherosclerosis is generalised

General examination

Pulse ..... **BP (both arms)** .....

- Xanthelasma, arcus, tobacco staining, signs of diabetes; irregular pulse (AF — embolic source)
- Upper-limb pulses, **carotid bruits**; abdominal aortic aneurysm (expansile pulsation)

Past and drug history

- Previous angioplasty, bypass, amputation; stroke, MI; diabetes with foot ulcers; antiplatelets, statins, cilostazol

Vascular vs neurogenic claudication

Feature	Vascular	Neurogenic (spinal stenosis)
Pain brought on by	Walking a fixed distance	Standing, walking a variable distance
Relief	Standing still, 2–5 min	**Sitting or bending forward**
Walking uphill	Worse	Easier (spine flexed)
Pulses	Reduced / absent	Normal
Skin	Ischaemic changes	Normal

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Examination of the lower limbs — step by step

Inspection (both limbs, exposed from groin to toes; look between the toes and at the heels)

- **Colour**: pallor, cyanosis, mottling; **dependent rubor** ("sunset foot")
- Trophic changes: **shiny, hairless skin**, thickened brittle nails, wasting of muscles
- **Ulcers** (punched-out, on toes, heel and pressure points, painful), **gangrene** — dry (line of demarcation) or wet; amputation stumps
- Venous guttering — collapsed veins in an elevated ischaemic limb

Palpation

- **Temperature** with the back of the hand — compare both sides and move proximally; cold level indicates block
- **Capillary refill time** (press the nail bed or pulp — normal <2 s; delayed in ischaemia)
- Tenderness, oedema, sensation (diabetic neuropathy)

Peripheral pulses (compare both sides)

Pulse	How to feel it
Femoral	Mid-inguinal point (midway between ASIS and pubic symphysis), just below the inguinal ligament
Popliteal	Patient supine, knee flexed 30–45°, both thumbs on the tibial tuberosity, fingertips press deep in the popliteal fossa; or prone with knee flexed
Posterior tibial	Behind the medial malleolus, midway to the heel, with the foot relaxed and inverted
Dorsalis pedis	Lateral to the extensor hallucis longus tendon on the dorsum, proximal part of 1st web space
Others	Radial, ulnar, brachial, subclavian, carotid; abdominal aorta

- Grade: 2+ normal, 1+ diminished, 0 absent; listen for **bruits** over femoral, iliac, aorta, renal, carotid

Special tests

- **Buerger's test**: lift the leg — pallor at an angle <20° (**Buerger's angle**) suggests severe ischaemia; then hang it over the bed edge — slow return of colour and reactive **rubor** (positive)
- **Ankle-brachial index (ABI)** with a hand-held Doppler: ankle systolic ÷ brachial systolic

ABI	Interpretation
1.0–1.4	Normal
0.9 or less	Peripheral arterial disease
<0.5	Severe disease, critical ischaemia
>1.4	Non-compressible, calcified arteries (diabetes, CKD) — use toe pressure

- Neurological examination of the foot (monofilament, vibration) — diabetic foot

Ulcers of the leg — tell them apart

Feature	Arterial	Venous	Neuropathic (diabetic)
Site	Toes, heel, shin, pressure points	**Gaiter area**, above medial malleolus	Sole, under metatarsal heads
Pain	**Very painful**	Mild ache	**Painless**
Edge / floor	Punched-out, pale floor	Sloping edge, granulation, exudate	Punched-out, callus around
Surrounding skin	Cold, hairless, absent pulses	Pigmentation, eczema, lipodermatosclerosis	Warm, dry, pulses present

Upper limbs

- **Allen's test** (before radial puncture): occlude both radial and ulnar, patient clenches; release the ulnar — colour returns in <7 s if the palmar arch is patent

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How to present

EXAMPLE

"This 60-year-old smoker has absent left popliteal, posterior tibial and dorsalis pedis pulses with a normal femoral pulse. The left foot is cold up to the ankle, pale on elevation with a Buerger's angle of 30° and dependent rubor. There is a dry gangrene of the left second toe. The right lower limb is normal. My diagnosis is chronic atherosclerotic peripheral arterial disease with left femoropopliteal occlusion and critical limb ischaemia."

Level of block from the pulses

Pulse lost from	Level of block	Claudication in
Femoral (and below)	Aorto-iliac	Buttock, thigh, calf
Popliteal (femoral present)	Femoropopliteal (adductor canal)	Calf
Pedal (popliteal present)	Tibial / peroneal	Foot, calf

Fontaine and Rutherford classification

Stage	Features
I	Asymptomatic
II	Intermittent claudication (IIa >200 m, IIb <200 m)
III	Ischaemic rest pain
IV	Ulceration or gangrene — III and IV = **chronic limb-threatening ischaemia**

Differential diagnosis

Condition	Points
Atherosclerotic PAD	Older, smoker, diabetic, other vascular disease, femoropopliteal disease
Thromboangiitis obliterans (Buerger's)	Young male (<45) smoker, distal small vessels, upper limbs too, migratory thrombophlebitis, Raynaud's
Embolism	Sudden onset, AF / MI / valve disease, the 6 Ps — pain, pallor, pulselessness, paraesthesia, paralysis, poikilothermia (perishing cold)
Takayasu arteritis	Young woman, upper limb claudication, unequal arm pulses and BP, bruits
Spinal canal stenosis	Pulses normal, relief on sitting / bending forward

Investigations

- **Doppler** (ABI, waveforms), duplex ultrasound, **CT / MR angiography**, digital subtraction angiography before intervention
- Glucose, HbA1c, lipids, renal function, ECG, echo (embolic source), ESR / CRP (vasculitis)

Management in one glance

- **Stop smoking**, supervised walking exercise, antiplatelet (aspirin / clopidogrel), statin, control diabetes and BP, foot care; cilostazol for claudication
- Revascularisation (angioplasty / stent, bypass) for rest pain, ulcer, gangrene or disabling claudication; amputation for non-viable limb

EXAMINER ASKS

Claudication vs neurogenic claudication · Buerger's angle · Why rest pain is relieved by hanging the leg · Why ABI is falsely high in diabetes · Differences between atherosclerosis and Buerger's disease · Dry vs wet gangrene.

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CHAPTER 15 · SHORT CASE · CVS

Deep Vein Thrombosis (DVT) Signs

The swollen painful leg — confirm DVT clinically, score probability and look for the cause

THE INSTRUCTION

"Examine this patient's legs" or "This patient has a swollen leg — examine". Compare both limbs, measure, look for signs of DVT and its complications, and look for the cause.

Bio data

Name ..... **Age / Sex** .....

Occupation .....

Long-distance drivers, sedentary work, long flights

Focused history

Symptoms

- **Unilateral leg swelling**, pain or heaviness in the calf / thigh, worse on standing and walking
- Redness, warmth; duration and progression
- Many DVTs are **asymptomatic** (especially in bedridden patients)

Symptoms of complications

- **Sudden breathlessness, pleuritic chest pain, haemoptysis, syncope — pulmonary embolism**
- Massive swelling, blue leg — phlegmasia caerulea dolens (venous gangrene risk)

Risk factors — Virchow's triad (stasis, vessel injury, hypercoagulability)

- **Immobility**: bed rest >3 days, paralysis (stroke), plaster cast, long travel
- **Surgery** (especially hip / knee replacement, pelvic, abdominal) and **trauma** (fractures of pelvis, femur)
- **Malignancy** (active, chemotherapy), weight loss
- **Pregnancy and puerperium**, oral contraceptives, hormone replacement
- Previous DVT / PE, family history of thrombosis (thrombophilia — protein C / S, antithrombin deficiency, factor V Leiden), antiphospholipid syndrome (recurrent miscarriages)
- Nephrotic syndrome, heart failure, polycythaemia, inflammatory bowel disease, obesity, varicose veins, IV drug use (femoral injection), central venous catheters

General examination

Pulse ..... **BP** .....

Respiratory rate ..... **SpO_2** .....

- Tachycardia, tachypnoea, hypoxia — **pulmonary embolism**
- Signs of malignancy (lymph nodes, abdominal mass, breast), pregnancy, varicose veins, signs of nephrotic syndrome

Past and drug history

- Previous DVT or PE, anticoagulants taken (duration, INR control, bleeding), recent surgery or admission, hormonal therapy, chemotherapy
- Bleeding history — peptic ulcer, haemorrhagic stroke, recent surgery (contraindications to anticoagulation)

CLINICAL PEARL

Unprovoked DVT in a middle-aged or elderly patient may be the first sign of an occult cancer — ask about weight loss and examine the breast, abdomen, rectum and lymph nodes.

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Examination of the leg — step by step

Inspection (compare both legs)

- **Unilateral swelling** of the calf or whole leg (iliofemoral DVT — swelling up to the groin)
- Colour: red, warm skin; bluish (cyanotic) leg; **dilated superficial veins** (collaterals) over the leg or abdominal wall
- Ulcers, varicose veins, scars of surgery, injection sites in the groin

Palpation

- **Pitting oedema** — extent (ankle, calf, thigh)
- **Local warmth**; **tenderness along the course of the deep veins** — calf, popliteal fossa, adductor canal, femoral triangle
- **Calf muscle tension** (firmness) compared with the other side
- Palpable **cord** — thrombosed vein
- Peripheral pulses — to exclude arterial disease (pulses are normal in DVT; absent in phlegmasia)

Measurement

- **Calf circumference** 10 cm below the tibial tuberosity — **difference >3 cm** is significant (Wells criterion)
- Thigh circumference 15 cm above the upper border of the patella

Classical signs (unreliable — sensitivity low; never rely on them)

- **Homans' sign**: pain in the calf on passive dorsiflexion of the foot with the knee straight — **not recommended** (low accuracy; risk of dislodging clot)
- **Moses' sign**: tenderness on squeezing the calf from front to back, not from side to side
- **Neuhof's sign**: deep tenderness and thickening on palpation of the posteromedial calf
- **Lowenberg's test**: pain below a BP cuff on the calf inflated to <150 mmHg (normal tolerance up to 180)

CLINICAL DIAGNOSIS IS UNRELIABLE

Clinical signs are absent in about half of DVTs and present in many legs without DVT. Use the **Wells score** and confirm with **compression ultrasonography**.

Examine for complications and cause

- Chest: tachypnoea, pleural rub, loud P2, RV heave (pulmonary embolism)
- Abdomen and pelvis: mass, ascites, hepatomegaly, pregnancy; per rectal / pelvic examination when indicated
- Breast, lymph nodes, prostate — occult malignancy
- Chronic changes in old DVT — **post-thrombotic syndrome**: oedema, pigmentation, lipodermatosclerosis, venous ulcer over the medial malleolus

Wells score for DVT

Clinical feature	Points
Active cancer (treatment within 6 months or palliative)	+1
Paralysis, paresis or recent plaster immobilisation of the leg	+1
Bedridden >3 days or major surgery within 12 weeks	+1
Localised tenderness along the deep venous system	+1
Entire leg swollen	+1
Calf swelling >3 cm compared with the other leg	+1
Pitting oedema confined to the symptomatic leg	+1
Collateral superficial (non-varicose) veins	+1
Previous documented DVT	+1
Alternative diagnosis at least as likely as DVT	−2

- Score ≥2 — **DVT likely** → compression ultrasound; score ≤1 — unlikely → **D-dimer**; a normal D-dimer excludes DVT

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How to present

EXAMPLE

"This 45-year-old woman, 10 days after abdominal hysterectomy, has swelling of the left leg up to the knee. The left calf is 4 cm larger than the right, warm and tender along the deep veins, with pitting oedema and dilated superficial veins. Peripheral pulses are normal. There are no signs of pulmonary embolism. Her Wells score is 4 — DVT likely. My diagnosis is left-sided provoked deep vein thrombosis of the calf and popliteal veins."

Differential diagnosis of a swollen painful leg

Condition	Points
Cellulitis	Fever, spreading erythema with a margin, skin break, lymphangitis
Ruptured Baker's cyst	Knee arthritis, sudden calf pain, crescent bruise below the malleolus
Calf muscle tear / haematoma	Sudden pain during exertion, bruising
Superficial thrombophlebitis	Tender, red, cord-like superficial vein
Lymphoedema / venous insufficiency	Chronic, painless, non-pitting (lymphoedema), varicose veins
Bilateral oedema	Systemic cause — heart, kidney, liver, hypoproteinaemia, drugs

Pulmonary embolism — recognise it at the bedside

Feature	Finding
Symptoms	Sudden breathlessness, pleuritic chest pain, haemoptysis, syncope, anxiety
Signs	Tachycardia, tachypnoea, hypoxia, low-grade fever, pleural rub, raised JVP, loud P2, RV heave
Massive PE	Hypotension (systolic <90 mmHg), shock, cardiac arrest — consider thrombolysis
Scores	Wells score for PE and PERC rule guide D-dimer and CT pulmonary angiography

Investigations

- **Compression duplex ultrasonography** (non-compressible vein — diagnostic), D-dimer, CBC, coagulation profile, renal and liver function (before anticoagulation)
- If PE suspected: ECG (sinus tachycardia, S1Q3T3), ABG, **CT pulmonary angiography**, echo
- Unprovoked DVT: look for malignancy; thrombophilia screen in the young, recurrent or family history

Management in one glance

- Anticoagulation: LMWH / fondaparinux → warfarin (INR 2–3), or a DOAC (rivaroxaban / apixaban); **3 months** for provoked, longer for unprovoked or cancer
- Early mobilisation with compression; thrombolysis / catheter-directed therapy for phlegmasia or massive PE; IVC filter if anticoagulation is contraindicated

Prevention (thromboprophylaxis)

- Assess every admitted patient; early mobilisation, leg exercises, hydration; LMWH or fondaparinux in medical and surgical high-risk patients; mechanical compression when bleeding risk is high

EXAMINER ASKS

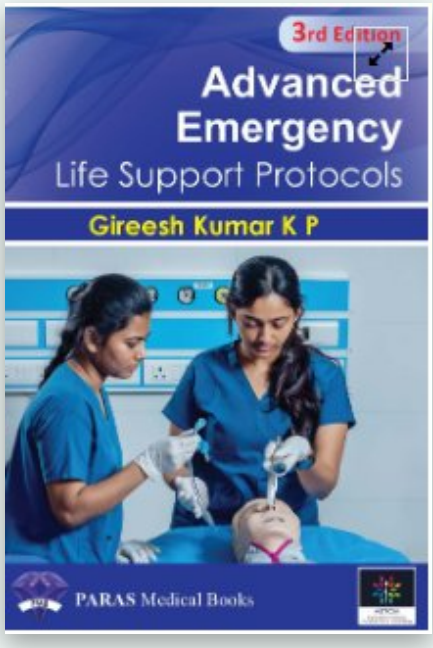
Virchow's triad · Why is Homans' sign not recommended? · Wells score · Role of D-dimer · Phlegmasia alba vs caerulea dolens · Post-thrombotic syndrome.

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CHAPTER 16 · SHORT CASE · RS

Pleural Effusion — Signs

"Examine this patient's chest" — elicit the signs of fluid, find the side, size and mediastinal position

THE INSTRUCTION

"Examine the respiratory system", "Examine the back of the chest" or "Percuss this patient's chest". In a short case you will rarely get time for a full history — be quick, systematic and compare both sides at every step.

Bio data

Name ..... **Age / Sex** .....

Quick focused history (if permitted — 1 minute)

- Breathlessness (duration, progression), pleuritic chest pain, cough, fever with evening rise, weight loss
- Known TB, heart, liver or kidney disease, cancer; previous pleural tapping or chest tube

Preparing the patient

- Introduce yourself, take consent, expose the chest to the waist (with a chaperone for women), patient **sitting up at 45°** for the front and **sitting forward** for the back, good light

General survey — what to note before touching the chest

Finding	Significance
Breathlessness at rest, use of accessory muscles	Large effusion, underlying lung disease
Preferred posture — lying on the affected side	Good lung uppermost for ventilation
Pallor, cachexia, lymph nodes (supraclavicular, axillary)	Tuberculosis, malignancy
Clubbing	Empyema, carcinoma lung, mesothelioma
Raised JVP, pedal oedema	Heart failure (bilateral effusion)
Icterus, signs of liver cell failure	Hepatic hydrothorax (right side)
Facial puffiness, anasarca	Nephrotic syndrome
Breast lump, mastectomy scar	Metastatic effusion
Scars on the back — tapping, ICD tube, biopsy	Previous drainage

Pulse ..... **Respiratory rate** .....

BP ..... **SpO_2 / Temperature** .....

Inspection of the chest

- **Shape** of chest, symmetry; fullness or bulging of intercostal spaces on one side (large effusion, empyema)
- **Movements**: reduced on the affected side — compare by looking from the foot end and from behind
- Apical impulse displaced away; visible pulsations, dilated veins; drooping of the shoulder (fibrosis — not in effusion)
- Swelling of the chest wall — **empyema necessitans** (pus tracking to the chest wall)

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Palpation

1. **Trachea**: index finger in the suprasternal notch — equal space on either side? **Shifted to the opposite side** in a large effusion
2. **Apex beat**: displaced to the opposite side (right effusion → displaced to the left, beyond the midclavicular line)
3. **Chest expansion**: hands on both sides, thumbs meeting in the midline — measure the separation on deep breath (front at the level of the 4th rib, back at the lower angle of the scapula); **reduced on the affected side**
4. **Vocal fremitus**: ulnar border or palm of the hand while the patient says "one, one, one" (or "ninety-nine") — compare corresponding areas on both sides; **reduced or absent** over fluid
5. Tenderness of the chest wall (empyema, rib secondaries), local warmth

Percussion

- Percuss from above downwards, front, axilla and back, comparing both sides; also the clavicles directly
- **Stony dull note** over the fluid — the most important sign; **flat**, with a feeling of resistance under the finger
- Upper limit of dullness: highest in the axilla, curving down to the front and back — **Ellis S-shaped (Damoiseau) curve**
- **Skodaic resonance**: a band of tympanitic (boxy) resonance **above the fluid** — relaxed, partially collapsed lung
- **Traube's space** dull — left-sided effusion (also splenomegaly, full stomach)
- **Grocco's triangle**: a paravertebral triangle of dullness on the **opposite** side — large effusion
- Shifting dullness in the chest — **hydropneumothorax** (horizontal fluid level that moves with posture)

Auscultation

- **Breath sounds diminished or absent** over the fluid
- **Vocal resonance diminished or absent** over the fluid
- At the **upper border** of the fluid: **bronchial breathing** of tubular / distant quality, **aegophony** (bleating, nasal "E" → "A"), whispering pectoriloquy — compressed lung
- **Pleural rub** — early or resolving effusion; disappears as fluid separates the layers
- **Succussion splash** — hydropneumothorax (shake the patient gently, listen)

Examine other systems quickly

- CVS — apex position, S3, murmurs, pericardial rub; abdomen — liver, spleen, ascites; breasts and lymph nodes

DO NOT FORGET

The back of the chest — most effusions are best demonstrated posteriorly. Always percuss the axillae.

Estimating the amount of fluid

Amount	Clinical and radiological guide
Minimal (<300 mL)	Blunted costophrenic angle on X-ray; ultrasound needed; few clinical signs
Moderate (300–1500 mL)	Dull up to the 4th–6th rib anteriorly; no or slight mediastinal shift
Massive (>1500 mL)	Dull to the 2nd rib or whole hemithorax, **mediastinal shift** to the opposite side

Common mistakes in the respiratory short case

- Examining only the front — **always examine the back and axillae**, with the patient sitting forward and arms folded
- Not comparing corresponding points on both sides at each step
- Percussing too hard (misses small areas) or with the finger not pressed firmly into the intercostal space
- Calling a dull note "stony dull" without the feel of resistance, or missing Traube's space
- Forgetting the trachea and apex — they decide between effusion, collapse and consolidation
- Listening through clothes; not asking the patient to breathe through the open mouth

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How to present

EXAMPLE

"On examination of the respiratory system, the trachea and apex are shifted to the left. Movements and expansion are reduced on the right side. Vocal fremitus is absent over the right infrascapular, infra-axillary and mammary areas, with a stony dull note, the upper level forming an S-shaped curve in the axilla. Breath sounds and vocal resonance are absent in the same areas, with bronchial breathing and aegophony at the upper border. My diagnosis is a moderate to massive right-sided pleural effusion with mediastinal shift."

Effusion, consolidation, collapse, fibrosis and pneumothorax — the comparison examiners love

Sign	Effusion	Consolidation	Collapse (obstructed)	Fibrosis	Pneumothorax	Hydropneumo-thorax
Movement	↓	↓	↓	↓	↓	↓
Trachea	Away	Central	**Towards**	**Towards**	Away	Away
Fremitus	↓ / absent	↑	↓	↑ or ↓	↓ / absent	↓
Percussion	**Stony dull**	Dull	Dull	Dull / impaired	**Hyper-resonant**	Resonant above, dull below
Breath sounds	↓ / absent	**Bronchial**	↓ / absent	Bronchial (cavity) or ↓	↓ / absent	↓; succussion splash
VR	↓	↑	↓	↑ or ↓	↓	↓
Added	Rub, aegophony above	Crepitations, rub	—	Coarse crepitations	Coin sound	Splash

Why the trachea may be central in a large effusion

- Underlying **collapse** (bronchial carcinoma blocking a bronchus) pulls the mediastinum back
- **Mesothelioma** or malignant infiltration freezing the mediastinum; loculated / encysted effusion
- Bilateral effusions

Common causes of effusion by side

- **Bilateral**: heart failure, nephrotic syndrome, hypoalbuminaemia, SLE, lymphoma
- **Right**: heart failure (right > left), cirrhosis (hepatic hydrothorax), Meigs' syndrome, amoebic liver abscess
- **Left**: pancreatitis, oesophageal rupture, aortic dissection, Dressler's syndrome
- **Either side**: tuberculosis (commonest in India), parapneumonic, malignancy, pulmonary embolism

Next steps

- Chest X-ray, **ultrasound-guided diagnostic tap** — appearance, protein, LDH (Light's criteria), glucose, ADA, cell count, Gram stain, AFB / CBNAAT, cytology
- Therapeutic tap or intercostal drain for breathlessness, empyema (pus, pH <7.2, organisms), haemothorax

EXAMINER ASKS

Why is the note stony dull? · What is Skodaic resonance? · Grocco's triangle · How much fluid is needed for clinical signs (~500 mL) and on X-ray (~200 mL PA)? · Why is there bronchial breathing above an effusion? · Causes of haemorrhagic effusion.

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CHAPTER 17 · SHORT CASE · RS

Consolidation — Signs

Bronchial breathing, increased fremitus and dullness with the trachea central

THE INSTRUCTION

"Examine this patient's chest" (often a patient with pneumonia on day 3–5). The defining triad — **dull note, increased vocal fremitus / resonance and bronchial breathing**, with the trachea in the midline.

Bio data

Name ..... **Age / Sex** .....

Quick focused history (if permitted)

- Fever with chills, cough with rusty / purulent sputum, pleuritic chest pain, breathlessness — days
- Past TB, COPD, diabetes, alcohol; antibiotics already taken

General survey

Finding	Significance
Toxic look, flushed face, herpes labialis	Pneumococcal pneumonia
Tachypnoea (>30/min), cyanosis, confusion	Severe pneumonia (CURB-65)
Hypotension, cold peripheries	Septic shock
Clubbing	Lung abscess, bronchiectasis, carcinoma, empyema
Lymph nodes, weight loss	Tuberculosis, carcinoma

Pulse ..... **Respiratory rate** .....

BP ..... **SpO_2 / Temperature** .....

Inspection

- Respiratory distress, alae nasi flaring, accessory muscles; **reduced movement on the affected side**
- No bulge, no retraction (lung volume is normal in consolidation)

Palpation

- **Trachea and apex central** (no volume change)
- Expansion reduced on the affected side
- **Vocal fremitus increased** — solid lung conducts vibration better than air-filled lung
- Pleural rub may be palpable (friction fremitus)

Mapping the lobes on the chest wall

Lobe	Where its signs are found
Right upper lobe	Front above the 4th rib (supraclavicular, infraclavicular, mammary); back — suprascapular
Right middle lobe	Front between the 4th and 6th ribs (right mammary and lower axilla); **loss of the right heart border** on X-ray
Right lower lobe	**Back** below the spine of the scapula (interscapular, infrascapular), lower axilla
Left upper lobe (with lingula)	Front above the 6th rib, axilla; lingula — loss of the left heart border
Left lower lobe	Back below the scapular spine, lower axilla; loss of the left hemidiaphragm outline

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Percussion

- **Dull (woody, not stony) note** over the consolidated lobe; percuss lightly to detect small areas
- Normal resonance elsewhere; Traube's space may be dull with left lower-lobe consolidation

Auscultation — the key findings

- **Bronchial breath sounds**: harsh, high-pitched, hollow, with an **expiratory phase as long as or longer than inspiration** and a **gap** between the two phases (vesicular: inspiration longer, no gap)
- Types: **tubular** (consolidation — high-pitched), **cavernous** (low-pitched, over a cavity), **amphoric** (metallic — open pneumothorax, large cavity)
- **Vocal resonance increased**; **bronchophony**, **whispering pectoriloquy** (whispered "one-two-three" heard clearly), **aegophony**
- **Crepitations**: fine–medium early (crepitatio indux) → coarse, wet in resolution (crepitatio redux)
- **Pleural rub** — leathery, creaking, in both phases, not changed by coughing, heard close to the ear

REMEMBER

Increased fremitus with dullness = solid lung with a patent bronchus (consolidation). Decreased fremitus with dullness = fluid, thickened pleura or collapse with a blocked bronchus.

WHY BRONCHIAL BREATHING?

Solid lung (alveoli filled with exudate) transmits sound from the bronchi directly to the chest wall without filtering the high frequencies — provided the **bronchus is patent**. If the bronchus is blocked (collapse), breath sounds are absent.

Distinguishing added sounds

Sound	Character	Clue
Crepitations (crackles)	Discontinuous, bubbling; mostly inspiratory	Fine — fibrosis, early pneumonia, LVF; coarse — bronchiectasis, resolution; may change with coughing
Wheeze (rhonchi)	Continuous, musical; mostly expiratory	Polyphonic — asthma, COPD; monophonic fixed — tumour narrowing a bronchus
Pleural rub	Creaking, close to the ear	Both phases, unchanged by cough, pain on breathing
Stridor	Inspiratory, loud, harsh	Upper airway obstruction — emergency

Examine the rest

- Other lung fields (bilateral pneumonia), heart (murmurs — endocarditis with septic emboli), abdomen (liver, spleen), meninges (neck stiffness)
- Look for **complications**: effusion (stony dull area with absent breath sounds at the base), empyema, lung abscess (cavernous breathing), respiratory failure

Severity at the bedside — CURB-65

- **C**onfusion · **U**rea >7 mmol/L · **R**espiratory rate ≥30 · **BP** <90 systolic or ≤60 diastolic · age ≥**65** — 0–1 home, 2 admit, ≥3 severe (consider ICU)

Signs of a non-resolving or complicated pneumonia

- Persistent fever after 72 h of appropriate antibiotics, new dullness with absent breath sounds (effusion / empyema), cavernous breathing (abscess), clubbing appearing, weight loss — think of TB, carcinoma, resistant organism or wrong diagnosis

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How to present

EXAMPLE

"The patient is febrile and tachypnoeic with a respiratory rate of 28/min. The trachea and apex are central. Movements are reduced over the right lower chest. Vocal fremitus is increased in the right infrascapular and infra-axillary areas, where the note is dull. Over the same area there are bronchial breath sounds of tubular type, increased vocal resonance with whispering pectoriloquy, coarse crepitations and a pleural rub. My diagnosis is consolidation of the right lower lobe, probably community-acquired pneumonia."

Differential diagnosis of bronchial breathing

Condition	Distinguishing points
Consolidation	Trachea central, dull note, increased fremitus, crepitations
Above a pleural effusion	Distant tubular breathing at the upper border, stony dullness below
Collapse with patent bronchus	Trachea pulled **towards** the lesion, reduced volume
Fibrosis	Trachea pulled towards, flattened chest, coarse crepitations, often upper lobe (TB)
Cavity	Cavernous / amphoric breathing, post-tussive crepitations

Causes of consolidation

- **Infective pneumonia** — bacterial (pneumococcus, Klebsiella, Staphylococcus), atypical, tuberculous (caseous)
- **Non-infective** — pulmonary infarction, pulmonary haemorrhage, organising pneumonia, alveolar cell carcinoma, lymphoma, eosinophilic pneumonia

Investigations

- **Chest X-ray** — homogeneous opacity limited by fissures, **air bronchogram**, silhouette sign
- CBC, CRP, procalcitonin, blood culture, sputum Gram stain / culture, AFB and CBNAAT, urea, electrolytes, ABG
- Urinary pneumococcal / Legionella antigen; scrub typhus / leptospira serology in endemic areas; HIV

Complications of pneumonia

- Pulmonary: parapneumonic effusion, **empyema**, lung abscess, respiratory failure, ARDS, delayed resolution
- Systemic: septic shock, AKI, meningitis, endocarditis, pericarditis, septic arthritis, jaundice, herpes labialis

Clues to the organism

Organism	Bedside clue
Pneumococcus	Sudden onset, rigors, rusty sputum, herpes labialis, lobar consolidation
Staphylococcus aureus	After influenza, IV drug use; cavities, pneumatoceles, empyema
Klebsiella	Alcoholics, diabetics; red currant-jelly sputum, upper lobe, bulging fissure
Atypical (Mycoplasma, Legionella)	Dry cough, headache, myalgia, few chest signs, relative bradycardia, diarrhoea (Legionella)
Anaerobes (aspiration)	Alcohol, seizures, poor dentition; foul sputum, right lower / posterior segments
Tuberculosis	Weeks of fever, weight loss, upper lobe, cavitation, no response to usual antibiotics

EXAMINER ASKS

What is the difference between bronchial and vesicular breathing? · Why is the trachea central? · Crepitatio indux and redux · Silhouette sign · Air bronchogram — what is it and where else is it seen? · How to distinguish crackles from a pleural rub?

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CHAPTER 18 · SHORT CASE · RS

Pneumothorax — Signs

Hyper-resonance with absent breath sounds — recognise tension pneumothorax, an emergency

THE INSTRUCTION

"Examine this patient's chest" — often a patient with an intercostal drain in place. Identify the side, decide whether it is **tension**, and assess the drain.

Bio data

Name ..... **Age / Sex** .....

Quick focused history (if permitted)

- **Sudden** onset of unilateral pleuritic chest pain and breathlessness — at rest or on exertion
- Tall, thin young man, smoker — **primary spontaneous pneumothorax** (rupture of apical blebs)
- Known COPD, asthma, TB, cystic lung disease, pneumonia (Staphylococcus, Pneumocystis in HIV) — **secondary**
- Trauma, central line insertion, pleural tap, mechanical ventilation, acupuncture — **traumatic / iatrogenic**
- Recurrence, previous drains or pleurodesis; catamenial (with menstruation — endometriosis)

Past and personal history

- **Smoking** (cigarettes and cannabis — strong risk of recurrence), occupation (divers, pilots), recent air travel or scuba diving
- Previous pneumothorax (recurrence ~30% after the first primary episode), lung disease, HIV, connective-tissue disease

General survey

Finding	Significance
Tall, thin (Marfanoid) young adult	Primary spontaneous pneumothorax, Marfan syndrome
Severe distress, cyanosis, sweating, agitation	Large or **tension** pneumothorax
Tachycardia >120, hypotension, raised JVP	**Tension** — obstructed venous return
Barrel chest, pursed lips	COPD with bullae (secondary)
Subcutaneous emphysema (crepitus in the neck / chest)	Air leak — trauma, drain, ventilation
Intercostal drain in situ	Check site, bubbling, swinging, sutures

Pulse ..... **Respiratory rate** .....

BP ..... **SpO_2** .....

Inspection

- **Reduced movement** on the affected side; the side may look fuller (hyper-expanded) with widened intercostal spaces
- Trachea deviated **away** (tension); apex shifted away
- Surgical emphysema, drain site, scars of previous thoracotomy / pleurodesis

CLINICAL PEARL

In a ventilated patient a sudden rise in airway pressure, fall in SpO_2 and BP, and unilateral absent breath sounds is a tension pneumothorax until proved otherwise.

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Palpation

- **Trachea shifted to the opposite side** — large / tension pneumothorax (central in a small one)
- Apex displaced to the opposite side (left pneumothorax may make the apex impalpable)
- **Expansion reduced**; **vocal fremitus reduced or absent**
- **Crepitus** of subcutaneous emphysema — crackling under the fingers like crumpling tissue paper

Percussion

- **Hyper-resonant (tympanitic) note** over the affected side
- **Obliteration of liver dullness** (right) or **cardiac dullness** (left)
- Dull below a horizontal level that shifts with posture — **hydropneumothorax**

Auscultation

- **Breath sounds diminished or absent**; vocal resonance diminished
- **Amphoric breathing** — open (bronchopleural fistula) pneumothorax
- **Coin test (bell sound)**: one coin held flat on the front of the chest is struck with another coin; a clear ringing, bell-like sound is heard with the stethoscope on the back of the same side
- **Succussion splash** — hydropneumothorax
- **Hamman's sign** — crunching sound synchronous with the heartbeat (left pneumothorax, pneumomediastinum)

TENSION PNEUMOTHORAX — A CLINICAL DIAGNOSIS

Severe distress, **trachea shifted away**, hyper-resonance, absent breath sounds, **tachycardia, hypotension, distended neck veins**, falling SpO_2. Do **not** wait for an X-ray — needle decompression (2nd intercostal space in the midclavicular line, or 4th–5th space in the mid-axillary line) followed by an intercostal drain.

Examining an intercostal drain (ICD) — "TOBS"

Check	What to look for
Tube site	4th–5th intercostal space in the mid-axillary line (**safe triangle**: anterior border of latissimus dorsi, lateral border of pectoralis major, line at the level of the nipple); sutures, dressing, surgical emphysema
Oscillation (swinging)	Fluid column rises on inspiration and falls on expiration — tube is patent and in the pleural space; **no swing** — blocked, kinked, or lung fully expanded
Bubbling	Air leak on coughing / expiration — ongoing pneumothorax or bronchopleural fistula; continuous bubbling — leak in the system
System	Underwater seal bottle **below the chest** level, tube end 2–3 cm under water, never clamp a bubbling drain; nature and volume of fluid drained

Examine the rest

- The other lung (bullae, COPD, fibrosis), the heart (displacement, Hamman's sign), signs of Marfan syndrome (arm span, palate, lens), HIV markers

Complications of an intercostal drain

Complication	Clue
Malposition	No swing, no drainage, persistent lung collapse on X-ray
Blocked / kinked tube	Swing stops while the lung is still collapsed
Surgical emphysema	Crepitus spreading around the drain site, neck, face
Bleeding, organ injury	Blood in the drain, falling Hb — intercostal vessels, liver, spleen
Infection	Fever, pus at the site, empyema
Re-expansion pulmonary oedema	Cough and breathlessness after rapid drainage of a large, long-standing collapse

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How to present

EXAMPLE

"This 22-year-old tall, thin man is breathless at rest with a respiratory rate of 26/min and SpO_2 92% on air. Pulse 104/min, BP 118/76 mmHg, JVP not raised. The trachea is central and movements are reduced on the right. Vocal fremitus is reduced, the note is hyper-resonant with loss of liver dullness, and breath sounds and vocal resonance are markedly diminished over the whole right hemithorax. The coin test is positive. My diagnosis is a right-sided primary spontaneous pneumothorax, not under tension."

Classification

Type	Features
Primary spontaneous	No known lung disease; tall, thin young smokers; apical blebs
Secondary spontaneous	Underlying lung disease — COPD, TB, cystic fibrosis, ILD, Pneumocystis, cavitating pneumonia, cancer; poorly tolerated
Traumatic / iatrogenic	Rib fractures, penetrating injury, central line, pleural tap, biopsy, ventilation
Tension	One-way valve — rising pressure, mediastinal shift, circulatory collapse

Differential diagnosis of hyper-resonance with diminished breath sounds

- **Large bulla** in COPD (no mediastinal shift away; CT distinguishes — never drain a bulla)
- Emphysema (bilateral), large cavity; congenital lobar emphysema; diaphragmatic hernia with gas-filled bowel (bowel sounds in the chest)

Investigations

- **Chest X-ray PA (erect)**: visible **visceral pleural line** with no lung markings beyond; size — **≥2 cm** interpleural distance at the hilum level = large (BTS); mediastinal shift; deep sulcus sign on supine film
- Ultrasound: absent lung sliding, "barcode" sign, **lung point**; CT for bullae and recurrent cases; ABG

Size of pneumothorax

Guideline	Large pneumothorax
British Thoracic Society	Visible rim ≥**2 cm** between lung margin and chest wall at the level of the hilum (~50% volume)
American (ACCP)	Apex-to-cupola distance ≥**3 cm**

CLINICAL PEARL

Treat the patient, not the X-ray — a breathless patient with a small pneumothorax and poor lung reserve (COPD) needs a drain; an asymptomatic young adult with a moderate one may need only aspiration or observation.

Management in one glance

- Small primary, minimal symptoms — observation, oxygen (speeds reabsorption), follow-up X-ray
- Large or symptomatic primary — **needle aspiration** or small-bore drain; secondary — intercostal drain and admission
- Persistent air leak (>5 days), recurrence, bilateral, occupation (pilots, divers) — **VATS pleurodesis / bullectomy**; advise to stop smoking and avoid flying until resolved

EXAMINER ASKS

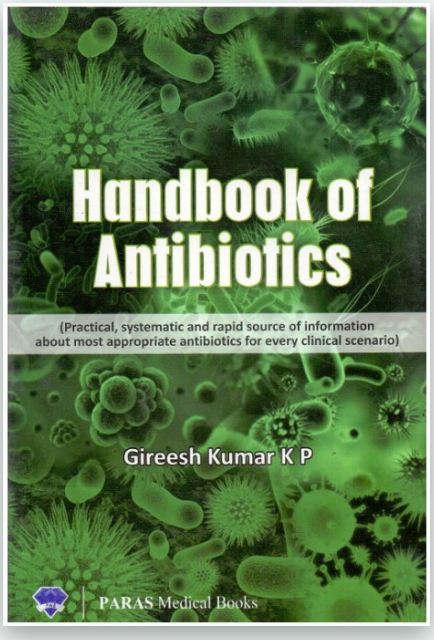
Clinical features of tension pneumothorax · Safe triangle · Why does the drain swing? · Why should a bubbling drain never be clamped? · Coin test · Pneumothorax vs bulla on X-ray.

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CHAPTER 19 · SHORT CASE · ABDOMEN

Splenomegaly

Feel, confirm and grade the spleen — then let its size and the company it keeps suggest the cause

THE INSTRUCTION

"Examine this patient's abdomen" or "Palpate the abdomen". A palpable spleen is one of the commonest short cases in India — malaria, cirrhosis with portal hypertension, haematological disease and kala-azar.

Bio data

Name ..... **Age / Sex** .....

Native place .....

Kala-azar (Bihar, Jharkhand, West Bengal, eastern UP), malaria-endemic areas

Quick focused history (if permitted)

- Dragging pain or heaviness in the left upper abdomen, early satiety; sudden severe pain — **splenic infarct** (sickle cell, CML, endocarditis)
- **Fever** (malaria, enteric, kala-azar, endocarditis, TB), weight loss, night sweats (lymphoma, CML), itching after a bath (polycythaemia vera)
- Bleeding, bruising, recurrent infections — cytopenias (marrow disease, hypersplenism)
- Jaundice, haematemesis, alcohol — **portal hypertension**; jaundice since childhood, transfusions, gallstones — **haemolytic anaemia** (thalassaemia, spherocytosis)
- Joint pains, rash — SLE, rheumatoid arthritis (Felty's syndrome)

General examination — the company the spleen keeps

Finding	Think of
Pallor	Haemolytic anaemia, leukaemia, kala-azar, malaria, hypersplenism
Icterus	Haemolysis, cirrhosis, malaria
Generalised lymphadenopathy	Lymphoma, CLL, ALL, TB, HIV, infectious mononucleosis
Petechiae, purpura, gum bleeding	Acute leukaemia, aplastic marrow, hypersplenism
Signs of liver cell failure, dilated abdominal veins	Cirrhosis with portal hypertension
Clubbing, splinter haemorrhages, murmur	**Infective endocarditis**
Frontal bossing, malar prominence (chipmunk facies)	**Thalassaemia major**
Darkening of skin, wasting	**Kala-azar** (visceral leishmaniasis)
Plethora, red face, itching	Polycythaemia vera
Sternal tenderness	Leukaemia (acute and CML)
Leg ulcers	Sickle cell disease, spherocytosis

Pulse ..... **Temperature** .....

BP ..... **Weight** .....

Inspection of the abdomen

- Fullness or visible mass in the left hypochondrium moving **downwards and medially towards the right iliac fossa** with respiration
- Dilated veins (portal hypertension), scars (splenectomy, previous surgery)

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Palpation of the spleen — step by step

1. Patient supine, abdomen relaxed, knees slightly flexed; you on the patient's right
2. Start palpating from the **right iliac fossa**, moving diagonally towards the **left costal margin** — a huge spleen can be missed if you start in the left hypochondrium
3. Use the flat of the right hand with fingers pointing towards the left costal margin; ask the patient to breathe in deeply — the spleen descends to meet the fingertips
4. Left hand placed behind the lower left ribs, lifting them forward (**bimanual palpation**)
5. If not felt, turn the patient to the **right lateral position** (45°) and repeat; **Middleton's method** — patient's left fist under the left lower ribs, examiner from the left side hooks the fingers under the costal margin
6. Percuss **Traube's space** — dullness supports splenomegaly; **Castell's sign** (dullness in the lowest left intercostal space in the anterior axillary line on full inspiration)

Features of the spleen to describe

- **Size**: in cm below the left costal margin along its long axis (towards the umbilicus / right iliac fossa)
- Surface (smooth / nodular), edge (sharp, rounded), **notch** on the anterior border, consistency (soft — acute infection; firm — chronic; hard — CML, myelofibrosis, malaria), tenderness (infarct, acute infection), **splenic rub** (infarct)

Confirm it is the spleen, not the left kidney

Feature	Spleen	Left kidney
Position	Superficial, anterior	Deep, in the loin
Movement with respiration	Moves **downwards and medially**	Moves vertically, less
Notch	Present	Absent
Fingers between mass and costal margin	**Cannot** get above it	**Can** get above it
Percussion over the mass	**Dull**	Resonant band (colon in front)
Bimanual palpation (ballotable)	Not ballotable	Ballotable

Grading of splenomegaly (Hackett's, used in malaria surveys)

Class	Spleen
0	Not palpable even on deep inspiration
1	Palpable on deep inspiration only
2	Palpable, not beyond a horizontal line halfway between the costal margin and umbilicus
3	Beyond that line but not below the umbilicus
4	Below the umbilicus, not beyond the line halfway between umbilicus and pubic symphysis
5	Beyond class 4

Then examine

- Liver (hepatosplenomegaly), ascites, lymph nodes in all regions (including epitrochlear), testes, heart (murmur), fundus (Roth spots, retinal haemorrhages in leukaemia)

Examining the lymph nodes — always with a spleen

1. **Cervical**: from behind the seated patient, head slightly flexed — submental, submandibular, pre- and post-auricular, occipital, anterior and posterior triangles, **supraclavicular** (Virchow's node on the left)
2. **Axillary**: support the patient's arm; feel the medial (central), anterior (pectoral), posterior (subscapular), lateral and apical groups
3. **Epitrochlear** (above the medial epicondyle) — enlarged in CLL, lymphoma, secondary syphilis, HIV; **inguinal** — horizontal and vertical groups
4. Describe: site, number, **size**, **consistency** (rubbery — lymphoma; hard — metastasis; firm, matted — TB), tenderness, **matting**, fixity, overlying skin (sinus — TB)

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How to present

EXAMPLE

"This 35-year-old man from Bihar has pallor and hyperpigmentation of the skin. Abdominal examination reveals a firm, non-tender spleen palpable 12 cm below the left costal margin, moving down and medially with respiration, with a palpable notch; I cannot get above it and it is dull to percussion. The liver is palpable 3 cm. There is no lymphadenopathy or ascites. My diagnosis is massive splenomegaly with mild hepatomegaly, probably due to visceral leishmaniasis; differentials are chronic malaria and CML."

Causes by size

Size	Common causes
Massive (>8 cm, crossing the midline or into the RIF)	**CML**, myelofibrosis, **kala-azar**, chronic malaria (hyper-reactive malarial splenomegaly), thalassaemia major, Gaucher's disease, splenic lymphoma
Moderate (4–8 cm)	Above + portal hypertension (cirrhosis, non-cirrhotic portal fibrosis, extrahepatic portal vein obstruction), lymphomas, CLL, haemolytic anaemias, polycythaemia vera
Mild (<4 cm)	Acute infections — malaria, **enteric fever**, infective endocarditis, viral hepatitis, infectious mononucleosis, septicaemia, TB, brucellosis; SLE, rheumatoid arthritis, megaloblastic anaemia

INDIAN SCENARIO

A young adult with moderate splenomegaly, normal liver function, previous haematemesis and no signs of liver failure — think of **non-cirrhotic portal fibrosis** or **extrahepatic portal vein obstruction**.

Hepatosplenomegaly — common causes

- Infections — malaria, enteric, kala-azar, viral hepatitis, TB, brucellosis; haematological — leukaemias, lymphomas, myeloproliferative disorders, thalassaemia; infiltrative — amyloidosis, Gaucher's; congestive — Budd–Chiari, constrictive pericarditis

Investigations

- **CBC with peripheral smear** (blasts, myelocytes — CML; target cells — thalassaemia; spherocytes; parasites), reticulocyte count, ESR
- Malarial antigen / smear, rK39 strip test (kala-azar), blood culture, Widal, LFT, HBsAg / anti-HCV
- **Ultrasound with Doppler** (size, portal vein, liver texture, collaterals), Hb electrophoresis / HPLC, Coombs test
- **Bone marrow** aspiration and biopsy, BCR-ABL (CML), JAK2 (polycythaemia, myelofibrosis), lymph node biopsy, upper GI endoscopy (varices)

Hypersplenism

- Splenomegaly + cytopenia(s) + compensatory marrow hyperplasia + correction after splenectomy

Splenectomy — vaccinate first

- Pneumococcal, Hib and meningococcal vaccines (2 weeks before elective splenectomy); lifelong risk of overwhelming post-splenectomy infection

EXAMINER ASKS

How does the spleen differ from the left kidney? · Why start palpating from the right iliac fossa? · How much must the spleen enlarge to be palpable (2–3 times)? · Causes of massive splenomegaly in India · Splenic rub · Traube's space.

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CHAPTER 20 · SHORT CASE · ABDOMEN

Hepatomegaly

Measure the liver span, describe the edge and surface, and decide whether it is congestive, infective, infiltrative or malignant

THE INSTRUCTION

"Palpate this patient's liver" or "Examine the abdomen". Percuss the upper border before you feel the lower edge — a liver pushed down by hyperinflated lungs is not an enlarged liver.

Bio data

Name ..... **Age / Sex** .....

Occupation / habits .....

Alcohol, IV drug use, healthcare work

Quick focused history (if permitted)

- Right upper abdominal pain or heaviness — stretching of the capsule (congestion, abscess, hepatitis, tumour)
- Fever — **amoebic / pyogenic liver abscess**, hepatitis, malaria, enteric, TB; fever with rigors and jaundice — cholangitis
- Jaundice, dark urine, itching; weight loss and anorexia — malignancy (HCC, metastases)
- Breathlessness, orthopnoea, leg swelling — **congestive heart failure**
- Alcohol (quantity, duration), diabetes and obesity (fatty liver), drugs, blood transfusion
- Diarrhoea / dysentery in the past — amoebic liver abscess

General examination

Finding	Think of
Raised JVP, pedal oedema, murmurs	Congestive hepatomegaly (right heart failure, TR, constrictive pericarditis)
Icterus	Hepatitis, obstructive jaundice, cirrhosis, malignancy
Signs of liver cell failure	Alcoholic liver disease, cirrhosis
Cachexia, lymph nodes (Virchow's node)	Malignancy — metastatic liver
Toxic, febrile, intercostal tenderness	**Liver abscess**
Pallor, lymphadenopathy, splenomegaly	Leukaemia, lymphoma, infections (malaria, kala-azar)
Obesity, acanthosis nigricans	MASLD (fatty liver)
Macroglossia, nephrotic features	Amyloidosis
Kayser–Fleischer ring	Wilson's disease

Pulse ..... **Temperature** .....

BP ..... **JVP** .....

Inspection

- Fullness of the right hypochondrium / epigastrium moving with respiration; **intercostal bulging** (abscess); visible pulsation (TR)
- Dilated veins, scars, caput medusae

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Examination of the liver — step by step

1. **Upper border by percussion**: percuss down the right midclavicular line from the 2nd intercostal space — normal upper border of dullness at the **5th intercostal space** (lowered in emphysema, raised by a subdiaphragmatic mass, effusion or collapse)
2. **Lower edge by palpation**: begin in the **right iliac fossa**, flat hand, fingers pointing up (or radial border of the index finger parallel to the costal margin); move up 1–2 cm with each inspiration until the edge meets the fingers
3. **Hooking method** from above if the edge is hard to define; **dipping** in the presence of ascites
4. Measure **liver span** in the right midclavicular line — normal **12–15 cm** in men, 10–13 cm in women (percussion from upper border to lower edge)
5. Describe: size (cm below the costal margin in the MCL and in the epigastrium below the xiphisternum), **edge** (sharp / rounded), **surface** (smooth, nodular), **consistency** (soft, firm, hard), **tenderness**, **pulsation**
6. Pulsatile liver — bimanual: one hand behind the lower right ribs, the other in front — **expansile systolic pulsation** in TR
7. Auscultate over the liver — **bruit** (HCC, alcoholic hepatitis), **friction rub** (perihepatitis, tumour, biopsy), venous hum
8. Examine for spleen, ascites, gall bladder (palpable in obstruction — **Courvoisier's law**), lymph nodes, testes, rectum

Character of the liver and what it suggests

Character	Causes
Soft, smooth, tender	Acute hepatitis, congestive cardiac failure
Firm, smooth, non-tender	Fatty liver, early cirrhosis, chronic hepatitis, lymphoma, amyloid
Hard, nodular (knobbly), irregular	**Metastases**, hepatocellular carcinoma, macronodular cirrhosis
Tender, localised bulge, intercostal tenderness	**Liver abscess** (amoebic — usually right lobe, single)
Pulsatile	Tricuspid regurgitation
Riedel's lobe (tongue-like projection of the right lobe)	Normal variant — commoner in women; mistaken for a mass or gall bladder

Signs to elicit when abscess is suspected

- **Intercostal tenderness** (press the lower right intercostal spaces), **point tenderness**, bulging of the intercostal spaces, raised and immobile right hemidiaphragm on X-ray
- Right basal crackles or effusion, rub; signs of rupture into peritoneum, pleura or pericardium

> **CLINICAL PEARL**
> A liver felt 3 cm below the costal margin with a normal span (upper border lowered) is a **ptosed** liver — COPD, asthma, right pleural effusion, subphrenic abscess — not hepatomegaly.

The gall bladder

- Palpate at the tip of the right 9th costal cartilage (lateral border of rectus) — a pear-shaped, smooth swelling moving with respiration, continuous with the liver, **cannot get above it**
- **Murphy's sign**: fingers under the costal margin, patient breathes in — catches breath with pain (acute cholecystitis)
- **Courvoisier's law**: a palpable gall bladder in a jaundiced patient is unlikely to be due to stones — think of carcinoma of the head of pancreas or periampullary carcinoma

Look for these associated signs at the same time

Sign	Significance
Splenomegaly	Portal hypertension, haematological disease, chronic infection
Ascites	Cirrhosis, Budd–Chiari, malignancy, heart failure
Raised JVP with hepatojugular reflux	Congestive hepatomegaly
Hard supraclavicular node, rectal shelf	Gastrointestinal malignancy with liver metastases
Pleural effusion / basal crackles on the right	Liver abscess, heart failure

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How to present

EXAMPLE

"The patient is icteric and cachectic with a hard left supraclavicular lymph node. The liver is palpable 8 cm below the right costal margin in the midclavicular line and 10 cm below the xiphisternum, with a span of 19 cm. It is hard, with an irregular nodular surface and a rounded edge, non-tender and non-pulsatile, with no bruit or rub. The spleen is not palpable and there is no ascites. My diagnosis is hepatomegaly, probably due to metastatic malignancy, with a primary in the stomach (Virchow's node)."

Causes of hepatomegaly

Group	Causes
Congestive	Right heart failure, TR, constrictive pericarditis, Budd–Chiari syndrome
Infective	Viral hepatitis, **liver abscess** (amoebic, pyogenic), malaria, enteric fever, kala-azar, TB, leptospirosis, hydatid cyst, schistosomiasis
Infiltrative	**Fatty liver** (alcohol, MASLD), amyloidosis, glycogen storage, haemochromatosis, Gaucher's
Malignant	**Metastases** (GI tract, breast, lung), hepatocellular carcinoma, lymphoma, leukaemia
Haematological	Thalassaemia, myelofibrosis, CML, haemolytic anaemias
Others	Early cirrhosis, primary biliary cholangitis, Wilson's disease, sarcoidosis

MASSIVE HEPATOMEGALY

More than 8 cm below the costal margin — Metastases, HCC, alcoholic fatty liver / hepatitis, congestive heart failure, amyloidosis, myeloproliferative disorders, hydatid cyst.

Tender hepatomegaly

- Congestive cardiac failure, acute hepatitis (viral, alcoholic), liver abscess, HCC, Budd–Chiari, perihepatitis (Fitz-Hugh–Curtis)

Investigations

- **LFT** (bilirubin, ALT, AST, ALP, GGT, albumin), INR, CBC, peripheral smear, viral markers (HBsAg, anti-HCV, IgM anti-HAV / HEV)
- **Ultrasound** abdomen (texture, focal lesions, abscess, biliary dilatation, Doppler of hepatic veins / portal vein); CT / MRI for masses
- **AFP** (HCC), amoebic serology, blood culture, malarial antigen; echocardiography (congestive); liver biopsy for infiltrative disease

Hepatomegaly with jaundice — localise

- Smooth tender liver + raised ALT — hepatitis; palpable gall bladder + itching, pale stools — malignant biliary obstruction (Courvoisier); hard nodular liver + weight loss — malignancy

Amoebic vs pyogenic liver abscess

Feature	Amoebic	Pyogenic
Patient	Young adult man, alcohol, dysentery	Elderly, diabetic, biliary disease
Lesion	Usually single, right lobe	Often multiple
Jaundice	Uncommon	Common (cholangitis)
Pus	"Anchovy sauce", sterile	Foul, organisms on culture
Treatment	Metronidazole ± aspiration	Antibiotics + drainage

EXAMINER ASKS

How do you measure the liver span? · Causes of a pulsatile liver · Why is the liver not palpable in late cirrhosis? · Riedel's lobe · Courvoisier's law · Amoebic vs pyogenic liver abscess.

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CHAPTER 21 · SHORT CASE · ABDOMEN

Ascites — Examination

Prove free fluid, estimate its amount, and use the abdomen and the tap to find the cause

THE INSTRUCTION

"Examine this patient's abdomen" in a distended abdomen. The examiner wants the **five Fs** considered, demonstration of **shifting dullness** and **fluid thrill**, and a sensible cause.

Bio data

Name ..... **Age / Sex** .....

Quick focused history (if permitted)

- Duration and progression of distension; which swelled first — abdomen (liver, peritoneum), legs (heart), face (kidney)
- Jaundice, haematemesis, alcohol, hepatitis — **cirrhosis**
- Fever, night sweats, weight loss, abdominal pain — **tuberculous peritonitis**
- Weight loss, anorexia, abdominal lump, vaginal bleeding, altered bowel habit — **malignancy**
- Breathlessness, orthopnoea — heart failure, constrictive pericarditis; frothy urine, facial puffiness — nephrotic syndrome

Five Fs of abdominal distension

- **F**luid · **F**at · **F**latus · **F**aeces · **F**etus (also **f**ibroid / ovarian tumour — huge cyst)

General examination

Finding	Think of
Signs of liver cell failure, icterus	Cirrhosis
Raised JVP, Kussmaul's sign, pulsus paradoxus	Constrictive pericarditis, heart failure
Facial puffiness, anasarca	Nephrotic syndrome
Cachexia, Virchow's node, hard lymph nodes	Abdominal malignancy
Low-grade fever, cervical nodes, wasting	Tuberculosis
Pedal oedema	Before ascites — cardiac / renal; after — liver, IVC obstruction
Breast lump, pelvic mass	Metastatic / ovarian malignancy (Meigs' syndrome — benign ovarian tumour with ascites and right effusion)

Pulse ..... **BP** .....

Abdominal girth (at umbilicus) ..... **Weight** .....

Past and treatment history

- Previous tapping (frequency, volume, colour of fluid), albumin infusions, SBP episodes, ATT, chemotherapy
- Diuretics (spironolactone, furosemide) — doses, response, side effects (gynaecomastia, hyperkalaemia, hyponatraemia, renal impairment)
- Diabetes, hypertension, heart disease, kidney disease, hepatitis, blood transfusions

SPONTANEOUS BACTERIAL PERITONITIS

Fever, abdominal pain or tenderness, worsening encephalopathy, unexplained AKI or simply clinical deterioration in a cirrhotic with ascites — tap the ascites at once (neutrophils ≥250/µL).

ORDER OF EXAMINATION

General examination → inspection → palpation (dipping) → percussion for fluid → auscultation → hernial orifices, genitalia, per rectal → back, chest and heart. Present in the same order.

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Inspection

- **Uniform distension**, **full flanks**, umbilicus transverse slit → **everted** (ascites) — in ovarian cyst the umbilicus is vertically stretched and flanks are not full
- Divarication of recti, umbilical / inguinal hernia, shiny stretched skin, striae
- Dilated veins — direction of flow (**away from umbilicus** — portal hypertension; below upwards — IVC obstruction)
- Scrotal or penile oedema; visible peristalsis (obstruction)

Palpation

- Tense or lax abdomen; tenderness (SBP, TB, malignancy); **doughy** feel (TB peritonitis)
- Organs by the **dipping method** — a quick push displaces fluid to feel the liver, spleen or a mass
- Masses: omental cake (TB, carcinomatosis), ovarian mass, nodular liver

Percussion — the tests for free fluid

1. **Percussion of flanks**: central resonance (bowel floats) with **dull flanks**, forming a horseshoe of dullness with concavity upwards; ovarian cyst — central dullness with resonant flanks
2. **Shifting dullness** (moderate ascites, ~**500–1000 mL**): percuss from the umbilicus to the flank until the note becomes dull; keep the finger there, turn the patient to the **opposite side**, wait **30–60 seconds**; the dull area becomes **resonant**
3. **Fluid thrill** (massive, tense ascites, >2 litres): patient's or assistant's **hand edge pressed along the midline** (to damp transmission through fat); tap one flank, feel the impulse on the other
4. **Puddle sign** (small ascites, ~**120 mL**): patient on knees and elbows for a few minutes; percuss the most dependent part (periumbilical) — dullness; or the stethoscope test (flick the flank, sound changes as you move the stethoscope away from the puddle)
5. Upper border of liver dullness, Traube's space, bladder (dullness above pubis — empty the bladder before examining)

Auscultation

- Bowel sounds; **venous hum** at umbilicus; hepatic bruit; **peritoneal rub**

Complete the examination

- **Per rectal** examination — rectal shelf (Blumer's shelf), mass; **per vaginal** — pelvic mass (with chaperone)
- Hernial orifices, scrotum (hydrocele, oedema), back (sacral oedema, renal angle), chest (effusion), heart (pericardial knock, raised JVP)

Grade (International Ascites Club)	Clinical
Grade 1 (mild)	Detected only by ultrasound
Grade 2 (moderate)	Symmetrical distension, shifting dullness
Grade 3 (large / tense)	Marked distension, fluid thrill, tense abdomen

Diagnostic paracentesis — how it is done

1. Consent; empty bladder; supine or slightly tilted to the side of the tap
2. Site: **left iliac fossa** — junction of the lateral and middle thirds of the line joining the umbilicus to the anterior superior iliac spine (avoid scars, inferior epigastric vessels and dilated veins); or 2 cm below the umbilicus in the midline
3. Aseptic precautions; local anaesthetic; **Z-track technique** (skin pulled 2 cm before needle entry) to prevent leak
4. Aspirate 30–60 mL for cell count, albumin, protein, culture (inoculate blood-culture bottles at the bedside), ADA, cytology
5. Large-volume paracentesis: give **albumin 6–8 g per litre removed** when >5 L is drained

COAGULOPATHY IS NOT A CONTRAINDICATION

Paracentesis is safe even with a raised INR in cirrhosis; avoid it in DIC and in the presence of bowel distension at the site.

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How to present

EXAMPLE

"This 48-year-old man has icterus, spider naevi, palmar erythema and gynaecomastia. The abdomen is uniformly distended with full flanks and an everted umbilicus; dilated veins around the umbilicus fill away from it. The liver is not palpable; the spleen is palpable 4 cm by dipping. Flanks are dull with central resonance, and shifting dullness is present; there is no fluid thrill. My diagnosis is moderate ascites with splenomegaly due to portal hypertension in decompensated chronic liver disease, probably alcohol-related."

Ascites vs other causes of distension

Feature	Ascites	Ovarian cyst / huge mass
Flanks	Full, dull	Not full, **resonant**
Centre	Resonant (bowel floats)	**Dull**
Umbilicus	Everted, transverse	Vertically stretched
Shifting dullness	Present	Absent
Margins	No definable margins	Upper and lateral margins felt; cannot get below it (pelvic origin)

Causes by serum–ascites albumin gradient (SAAG)

SAAG	Causes
≥1.1 g/dL (portal hypertension)	**Cirrhosis**, alcoholic hepatitis, congestive heart failure, constrictive pericarditis, Budd–Chiari, portal vein thrombosis, non-cirrhotic portal fibrosis, massive liver metastases
<1.1 g/dL	**Tuberculous peritonitis**, peritoneal carcinomatosis, **nephrotic syndrome**, pancreatic ascites, chylous ascites, serositis (SLE)

PROTEIN HELPS FURTHER

SAAG ≥1.1 with ascitic protein ≥2.5 g/dL suggests a cardiac cause (heart failure, constriction, Budd–Chiari); with protein <2.5 g/dL — cirrhosis.

Ascitic fluid analysis

Test	Interpretation
Appearance	Straw — cirrhosis; turbid — infection; **blood-stained** — malignancy, TB, trauma; **milky** — chylous (lymphoma, filariasis, trauma)
Cell count	**Neutrophils ≥250/μL — SBP**; lymphocyte-predominant — TB, malignancy
ADA	>39 U/L — tuberculous peritonitis
Others	Total protein, glucose, LDH, amylase (pancreatic), triglycerides (chylous), Gram stain, culture in blood-culture bottles, AFB, CBNAAT, **cytology**

Other investigations

- USG abdomen with Doppler, LFT, INR, renal function, urine protein, echo, CA-125, CT abdomen, diagnostic laparoscopy for peritoneal biopsy (TB, malignancy)

EXAMINER ASKS

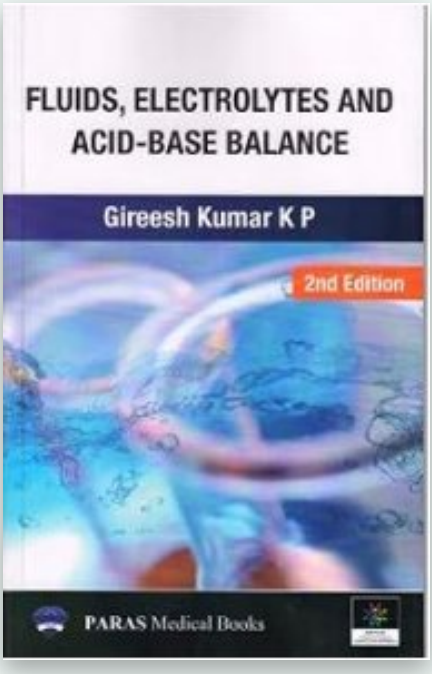
Minimum fluid for shifting dullness, fluid thrill and puddle sign · Why must the bladder be emptied first? · SAAG — principle and use · Ascites with a high protein · Refractory ascites · Diagnostic criteria for SBP.

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CHAPTER 22 · SHORT CASE · CNS

Cranial Nerve Palsies

"Examine the cranial nerves" — test all twelve quickly, find the palsy, decide UMN or LMN, and localise

THE INSTRUCTION

"Examine this patient's cranial nerves", "Look at the face" or "Examine the eye movements". The common short cases are **facial palsy (Bell's)**, **III nerve palsy**, **VI nerve palsy**, **multiple lower cranial nerve palsies (bulbar palsy)** and **Horner's syndrome**.

Bio data

Name ..... **Age / Sex** .....

Quick focused history (if permitted)

- Onset (sudden — vascular, Bell's; gradual — tumour), double vision, drooping of the eyelid, deviation of the mouth, drooling, inability to close the eye, hyperacusis, loss of taste
- Difficulty in swallowing, nasal regurgitation, change of voice, slurred speech — bulbar palsy
- Headache, vomiting, fever, ear discharge, vesicles in the ear (Ramsay Hunt), diabetes, hypertension, trauma, weakness of limbs

Examination of the cranial nerves — the quick routine

Nerve	Test
I	Smell each nostril separately with familiar, non-irritant odours (coffee, soap) — eyes closed
II	**Visual acuity** (Snellen, each eye with glasses), **fields by confrontation** (and red pin for central scotoma), **colour vision** (Ishihara), **fundus**; pupils — **direct and consensual light reflex**, accommodation, swinging-flashlight test (RAPD)
III, IV, VI	Ptosis, pupil size, eye position at rest; **eye movements** in an "H" pattern — ask about diplopia, note nystagmus; convergence
V	Sensation in all three divisions (forehead, cheek, chin); **corneal reflex**; motor — clench teeth (masseter, temporalis), open mouth against resistance (jaw deviates to the weak side — pterygoids); **jaw jerk**
VII	Raise eyebrows, close eyes tightly (bury the eyelashes), show teeth, puff cheeks, whistle; taste in anterior ⅔ of tongue; hyperacusis
VIII	Whisper test, finger rub; **Rinne and Weber** with 512 Hz fork; nystagmus, Romberg (vestibular)
IX, X	Say "aah" — palatal movement and **uvula deviation (to the normal side)**; gag reflex; voice, cough, swallowing a sip of water
XI	Shrug shoulders against resistance (trapezius); turn head against resistance (sternocleidomastoid — turns the head to the **opposite** side)
XII	Tongue at rest — wasting, **fasciculations**; protrude — **deviates to the weak side**; push against cheek

General examination

- Pulse, BP, blood sugar (diabetes — III, VI, VII palsies); ear and parotid (VII); neck stiffness; limbs — hemiplegia (crossed signs)

Pulse ..... **BP** .....

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Facial nerve palsy — UMN or LMN?

Feature	UMN (supranuclear)	LMN (nuclear / infranuclear)
Upper face (frowning, eye closure)	**Spared** (bilateral cortical supply)	**Involved** — cannot wrinkle forehead or close the eye
Lower face	Weak — opposite side to the lesion	Weak — **same side** as the lesion
Bell's phenomenon	Absent	**Present** — eyeball rolls up on attempted closure
Emotional movements	Preserved (voluntary lost)	Lost
Associated	Hemiplegia on the same side as the facial weakness	Hyperacusis, loss of taste, ear vesicles, parotid swelling

Localising an LMN facial palsy along its course

- **Pons** — with VI nerve palsy and contralateral hemiplegia (Millard–Gubler)
- **Cerebellopontine angle** — with V, VIII palsy (acoustic neuroma)
- **Facial canal** — above the chorda tympani: loss of taste; nerve to stapedius: **hyperacusis**; geniculate ganglion: **Ramsay Hunt** (vesicles in the ear canal); **Bell's palsy**
- **Stylomastoid foramen / parotid** — only facial weakness (parotid tumour, trauma)

Oculomotor (III) nerve palsy

- **Ptosis** (complete), eye **"down and out"**, **dilated, non-reacting pupil** — surgical (compressive) III palsy: **posterior communicating artery aneurysm**, uncal herniation
- **Pupil-sparing** III palsy — medical (ischaemic): **diabetes**, hypertension
- Midbrain — with contralateral hemiplegia (Weber), contralateral tremor / ataxia (Benedikt)

Abducens (VI) and trochlear (IV) nerve palsies

- **VI**: eye turned **inwards** (convergent squint), cannot abduct, horizontal diplopia worse on looking to the affected side — raised ICP (**false localising sign**), diabetes, Gradenigo's syndrome (petrous apicitis), pontine lesion
- **IV**: vertical diplopia on looking down and in (reading, descending stairs), **head tilt to the opposite side** — trauma, diabetes

Horner's syndrome

- **Partial ptosis, miosis, enophthalmos (apparent), anhidrosis** on the same side; pupil dilates slowly in the dark
- Causes: lateral medullary syndrome, cervical cord lesion, **Pancoast tumour** (apex of lung), cervical lymph nodes, carotid dissection, thyroid malignancy

Bulbar and pseudobulbar palsy

Feature	Bulbar (LMN — IX, X, XII)	Pseudobulbar (UMN — bilateral corticobulbar)
Tongue	**Wasted, fasciculating**	Small, stiff, spastic
Jaw jerk	Absent / normal	**Brisk**
Speech	Nasal	Slow, strained ("Donald Duck")
Emotions	Normal	**Emotional lability** (pathological laughing / crying)
Causes	Motor neuron disease, GBS, myasthenia, syringobulbia, polio	Bilateral strokes, MND, multiple sclerosis

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How to present

EXAMPLE

"There is weakness of the entire right half of the face — the patient cannot wrinkle the right forehead or close the right eye, Bell's phenomenon is present, the nasolabial fold is flattened and the angle of the mouth deviates to the left on showing the teeth. Taste is lost over the anterior two-thirds of the right side of the tongue and there is hyperacusis. Other cranial nerves and the limbs are normal; there are no vesicles in the ear. My diagnosis is right LMN facial palsy with the lesion in the facial canal proximal to the nerve to stapedius — most likely Bell's palsy."

Multiple cranial nerve palsies — localise by the combination

Nerves involved	Site
III, IV, V_1, VI (± Horner's)	**Cavernous sinus** / superior orbital fissure — thrombosis, aneurysm, tumour, Tolosa–Hunt
V, VII, VIII (± cerebellar signs)	**Cerebellopontine angle** — acoustic neuroma, meningioma
IX, X, XI	**Jugular foramen** (Vernet syndrome) — glomus tumour, metastasis
IX, X, XI, XII	Base of skull / retroparotid space; with Horner's — Villaret syndrome
V, IX, X, XI + crossed sensory loss, Horner's, ataxia	**Lateral medullary (Wallenberg) syndrome** — PICA / vertebral artery occlusion
Multiple, bilateral (incl. bilateral VII)	Basal meningitis (TB), carcinomatous meningitis, **GBS**, sarcoidosis, leprosy, diabetes

Investigations

- Blood sugar, HbA1c, BP, ESR; **MRI brain** with contrast (and MR angiography for a painful pupil-involving III palsy — emergency)
- CSF (basal meningitis, GBS), audiometry (CP angle), nerve conduction, acetylcholine-receptor antibodies (myasthenia — fatigable ptosis, no pupil change)

Treatment of Bell's palsy in one glance

- **Prednisolone** 1 mg/kg (60 mg) daily for 5 days then taper, within 72 hours; add acyclovir / valacyclovir in Ramsay Hunt; **eye protection** — lubricant drops, taping the eye shut at night; most recover in 3–6 months

Extraocular muscles — actions and nerve supply (LR_6 SO_4, rest III)

Muscle	Tested by looking	Nerve
Lateral rectus	Outwards (abduction)	VI
Medial rectus	Inwards (adduction)	III
Superior rectus	Up and out	III
Inferior rectus	Down and out	III
Inferior oblique	Up and in	III
Superior oblique	**Down and in**	IV

RULES OF DIPLOPIA

Diplopia is maximal in the direction of action of the weak muscle; the **false image is the outer one** and belongs to the affected eye (cover each eye to see which image disappears).

EXAMINER ASKS

Why is the forehead spared in UMN palsy? · Why is the pupil involved in compressive but spared in diabetic III palsy? · False localising signs · Bell's phenomenon · Crocodile tears.

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CHAPTER 23 · SHORT CASE · CNS

Parkinsonism — Signs

Tremor, rigidity, bradykinesia and postural instability — idiopathic Parkinson's disease or a mimic?

THE INSTRUCTION

"Look at this patient", "Examine this patient's gait" or "Examine the upper limbs". Parkinsonism is often diagnosed from the doorway — the face, posture, tremor and walk.

Bio data

Name ..... **Age / Sex** .....

Occupation .....

Manganese miners, welders, pesticide exposure

Quick focused history (if permitted)

- **Onset — unilateral, gradual**, after 55–60 years (idiopathic PD); symmetrical, early falls — atypical parkinsonism
- Tremor at rest, slowness of movement (buttoning, writing — **micrographia**), stiffness, shuffling walk, freezing, falls
- Soft monotonous speech, drooling, difficulty in swallowing, turning in bed
- **Non-motor**: loss of smell, constipation, REM sleep behaviour disorder (acting out dreams), depression, anxiety, urinary urgency, postural giddiness, memory loss, hallucinations
- **Drugs**: **haloperidol**, other antipsychotics, **metoclopramide**, prochlorperazine, flunarizine / cinnarizine, sodium valproate, reserpine — drug-induced parkinsonism
- Head injuries (boxers), stroke risk factors (vascular parkinsonism), encephalitis, carbon monoxide exposure, family history (young onset)
- Response to levodopa, "on–off" fluctuations, dyskinesias

General inspection — before touching the patient

Feature	What you see
Face	**Mask-like (hypomimia)**, reduced blinking, staring, greasy (seborrhoeic) skin, drooling
Posture	**Stooped (simian)** — flexed neck, trunk, elbows, knees
Tremor	**Resting "pill-rolling" tremor**, 4–6 Hz, often asymmetrical; reduced on action, worse with stress or mental arithmetic
Movements	Paucity of spontaneous movements; reduced arm swing
Speech	Hypophonic, monotonous, festinant

Pulse ..... **BP — lying and standing** .....

- **Postural hypotension** (fall ≥20/10 mmHg) — autonomic involvement; prominent and early in multiple system atrophy

Past, treatment and family history

- Psychiatric illness (antipsychotics), vertigo or migraine (flunarizine / cinnarizine), vomiting (metoclopramide), epilepsy (valproate)
- Levodopa — dose, frequency, duration of benefit (**wearing-off**), **dyskinesias**, hallucinations, impulse-control disorders (gambling, shopping) with dopamine agonists
- Family history of parkinsonism or tremor; Wilson's disease in siblings (young patients)

CLINICAL PEARL

Asymmetric onset, rest tremor and a clear response to levodopa favour idiopathic Parkinson's disease. Symmetrical onset, early falls, early dementia or autonomic failure, and a poor response to levodopa are red flags for another cause.

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Examination — step by step

Tremor

- Observe at rest with hands on the lap; distract (count backwards) to bring it out; outstretched hands — tremor may re-emerge after a few seconds (re-emergent tremor); finger–nose — tremor lessens

Rigidity

- Passively flex and extend the wrist and elbow, rotate the forearm slowly
- **Lead-pipe rigidity** — uniform resistance throughout the range; **cogwheel rigidity** — rigidity with superimposed tremor (ratchet-like), best felt at the wrist
- **Froment's manoeuvre** (activation): ask the patient to open and close the opposite hand — rigidity increases

Bradykinesia (the essential feature)

- **Finger tapping** (index on thumb, 10 times, as fast and wide as possible), hand opening and closing, pronation–supination, foot tapping — look for **slowness and progressive decrement** in speed and amplitude
- Ask the patient to write a sentence — **micrographia**; draw a spiral

Gait and posture

- Difficulty in rising from the chair, **stooped posture**, **short shuffling steps**, reduced arm swing (often unilateral), **festination** (hurrying steps), **freezing** at doorways, **turning en bloc** (many steps to turn)
- **Pull test**: stand behind, warn the patient, pull briskly at the shoulders — more than two steps backwards or falling = **postural instability** (late in PD; early in PSP)

Other signs

- **Glabellar tap (Myerson's sign)**: repeated tapping between the eyebrows causes continued blinking (normally stops after a few taps)
- **Vertical gaze palsy** (down-gaze) suggests **PSP**; **reflexes normal, plantars flexor** (extensor — MSA, vascular)
- Cerebellar signs (MSA-C), apraxia and cortical sensory loss (corticobasal degeneration), cognitive testing (dementia with Lewy bodies)

Types of increased tone — know the difference

Type	Feel	Lesion
Lead-pipe rigidity	Uniform resistance in both directions, throughout the range	Extrapyramidal
Cogwheel rigidity	Ratchet-like jerks (rigidity + tremor)	Extrapyramidal
Clasp-knife spasticity	Resistance builds then suddenly gives way; velocity-dependent; flexors of arm, extensors of leg	Pyramidal (UMN)
Paratonia (gegenhalten)	Resistance rises with speed of movement	Frontal lobe, dementia

Differentiating tremors

Feature	Parkinsonian	Essential tremor	Cerebellar (intention)
When	**At rest**	**Posture and action**	On reaching a target
Site	Hands (pill-rolling), chin, legs	Hands, **head (titubation)**, voice	Limbs, trunk
Alcohol	No effect	**Improves**	Worse
Associated	Rigidity, bradykinesia	Family history, normal tone	Ataxia, nystagmus, dysarthria

Parkinson's plus syndromes — red flags

Condition	Clues
Progressive supranuclear palsy (PSP)	**Vertical gaze palsy**, early backward falls, axial rigidity, "surprised" stare
Multiple system atrophy (MSA)	**Early autonomic failure** (postural hypotension, urinary incontinence, erectile dysfunction), cerebellar signs, stridor
Dementia with Lewy bodies	**Early dementia**, fluctuating cognition, visual hallucinations
Corticobasal degeneration	Asymmetric apraxia, **alien limb**, cortical sensory loss, myoclonus

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How to present

EXAMPLE

"This 64-year-old man has a mask-like face with reduced blinking and a stooped posture. There is a resting pill-rolling tremor of the right hand, 4–5 Hz, which diminishes on movement. Tone shows cogwheel rigidity at the right wrist and lead-pipe rigidity at the right elbow, increased by Froment's manoeuvre. Finger tapping is slow with decrement, more on the right. Gait is shuffling with reduced right arm swing and turning en bloc; the pull test is normal. Eye movements, reflexes and plantars are normal and there is no postural drop. My diagnosis is parkinsonism, asymmetrical, most probably idiopathic Parkinson's disease, Hoehn and Yahr stage 1."

Diagnosis of Parkinson's disease (MDS clinical criteria)

- **Bradykinesia** plus **rest tremor** and / or **rigidity** — with supportive features: dramatic response to levodopa, levodopa-induced dyskinesias, rest tremor, loss of smell; absence of red flags

Causes of parkinsonism

Type	Causes
Idiopathic	**Parkinson's disease** (~75%)
Drug-induced	Antipsychotics, metoclopramide, flunarizine, valproate — symmetrical, rest tremor less common, recovers (weeks–months) after stopping
Vascular	Multiple lacunes — **lower-body parkinsonism**, broad-based shuffling gait, pyramidal signs, poor levodopa response
Parkinson's plus	PSP, MSA, DLB, corticobasal degeneration
Others	Wilson's disease (young — check KF ring), post-encephalitic, CO and manganese poisoning, head trauma, normal pressure hydrocephalus (gait, dementia, urinary incontinence)

Hoehn and Yahr staging

Stage	Features
1	Unilateral disease
2	Bilateral, balance not impaired
3	Bilateral, mild–moderate, postural instability, physically independent
4	Severe disability, still able to walk or stand unassisted
5	Wheelchair-bound or bedridden unless aided

Investigations

- Diagnosis is **clinical**; MRI brain to exclude vascular, NPH and structural causes; serum ceruloplasmin and slit lamp in patients <50 years; DAT scan in doubtful cases

Treatment in one glance

- **Levodopa + carbidopa** (most effective), dopamine agonists (pramipexole, ropinirole) in the young, MAO-B inhibitors (rasagiline), COMT inhibitors (entacapone), amantadine (dyskinesia); physiotherapy; deep brain stimulation in selected patients

EXAMINER ASKS

Lead-pipe vs cogwheel vs clasp-knife rigidity · Why is the tremor reduced on action? · Glabellar tap · Drugs causing parkinsonism · Features of Parkinson's plus syndromes · Long-term complications of levodopa.

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CHAPTER 24 · SHORT CASE · ENDOCRINE

Thyroid Enlargement (Goitre)

Is it the thyroid, what kind of swelling, is the patient eu-, hypo- or hyperthyroid, and are there pressure effects or malignancy?

THE INSTRUCTION

"Examine this patient's neck" or "Examine the thyroid". Inspect from the front, palpate from **behind**, test movement with swallowing and tongue protrusion, and always assess the **thyroid status**.

Bio data

Name ..... **Age / Sex** .....

Native place .....

Sub-Himalayan belt and other iodine-deficient areas — endemic goitre

Quick focused history (if permitted)

- Swelling in front of the neck — duration, rate of growth (sudden increase — haemorrhage into a nodule or malignancy), pain (thyroiditis, haemorrhage, anaplastic carcinoma)
- **Pressure symptoms**: difficulty in breathing (worse on lying or raising arms), **dysphagia**, **hoarseness** (recurrent laryngeal nerve — malignancy)
- **Hyperthyroid symptoms**: weight loss with good appetite, heat intolerance, sweating, palpitation, tremor, diarrhoea, irritability, menstrual change, eye symptoms
- **Hypothyroid symptoms**: weight gain, cold intolerance, constipation, lethargy, dry skin, menorrhagia, hoarse voice
- History of **neck irradiation** in childhood, family history of thyroid cancer or MEN 2 (medullary carcinoma), iodine intake, goitrogens (cabbage, cassava), drugs (amiodarone, lithium)

General examination

- Built, anxiety / lethargy, **pulse** (tachycardia, AF — hyperthyroid; bradycardia — hypothyroid), warm moist or cool dry hands, **fine tremor**, eye signs, proximal myopathy, pretibial myxoedema
- Lymph nodes of the neck (papillary carcinoma), voice (hoarseness)

Pulse ..... **BP** .....

Weight ..... **Temperature** .....

Inspection of the neck (patient sitting, neck slightly extended)

- Swelling in the lower anterior neck — site (one lobe, isthmus, both), shape (butterfly — diffuse), surface, skin over it (dilated veins — retrosternal extension)
- **Moves up with deglutition** (give the patient a sip of water) — thyroid is invested by the pretracheal fascia attached to the larynx
- **Moves with protrusion of tongue** — **thyroglossal cyst** (not the thyroid)
- Lower border visible on swallowing? — if not, suspect **retrosternal extension**
- Pemberton's sign: raise both arms above the head for a minute — facial plethora, distended neck veins, stridor = retrosternal goitre obstructing the thoracic inlet

Past, treatment and family history

- Previous thyroid surgery (scar), radioactive iodine, antithyroid drugs or thyroxine (dose, duration), steroid therapy; pregnancies
- Family history of goitre, autoimmune thyroid disease, medullary carcinoma, phaeochromocytoma or hyperparathyroidism (**MEN 2**)

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Palpation — from behind (patient's neck slightly flexed)

1. Thumbs on the back of the neck, fingers on the lobes; feel each lobe by **Lahey's method** (push the opposite lobe across with one hand and palpate with the other) — also **Crile's method** (thumb on the nodule while swallowing)
2. Confirm **movement with swallowing**; confirm you **can get below** the lower border (not in retrosternal goitre)
3. Describe: **size** (each lobe and isthmus), **shape**, **surface** (smooth — diffuse; nodular), **consistency** (soft, firm, hard — malignancy, Riedel's), **tenderness** (thyroiditis), **mobility** (fixed — malignancy), **margins**
4. **Number of nodules** — solitary, multinodular, diffuse
5. **Trachea** — position from the front (deviated by a large lobe); **Kocher's test** — gentle pressure on the lateral lobes causes **stridor** if the trachea is compressed
6. **Carotid pulsation** — normally felt behind the lobe; absent (**Berry's sign**) — malignancy infiltrating or encasing the carotid sheath
7. **Cervical lymph nodes** — all groups, especially the deep cervical chain (papillary carcinoma)
8. **Thrill** at the upper pole — Graves' disease (increased vascularity)

Percussion and auscultation

- Percuss over the manubrium — **dullness** suggests retrosternal extension
- **Bruit** over the upper poles — **Graves' disease** (differentiate from venous hum and transmitted carotid murmur)

Assessment of thyroid status — bedside signs

Sign	Hyperthyroid	Hypothyroid
Pulse	Tachycardia, **AF**, collapsing	Bradycardia
Hands	Warm, moist, **fine tremor**, palmar erythema, onycholysis	Cold, dry, coarse, puffy
Eyes	Lid retraction, lid lag, exophthalmos (Graves')	Periorbital puffiness
Reflexes	Brisk	**Delayed relaxation** (ankle jerk)
Weight / skin	Loss; thin, warm skin	Gain; dry, rough, yellowish skin
Others	Proximal myopathy, pretibial myxoedema	Hoarse voice, slow speech, carpal tunnel

INDICES

Wayne's and Billewicz indices score symptoms and signs at the bedside for hyperthyroidism and hypothyroidism respectively — a total score guides the clinical diagnosis before tests.

WHO grading of goitre

- **Grade 0** — no palpable or visible goitre · **Grade 1** — palpable but not visible with the neck in the normal position · **Grade 2** — visible with the neck in the normal position

Retrosternal goitre — suspect it when

- Lower border cannot be reached on swallowing, **dullness over the manubrium**, dilated veins over the upper chest, **Pemberton's sign**, tracheal deviation, stridor, dysphagia, hoarseness, SVC obstruction

Is it the thyroid? — the swelling that moves

Swelling	Moves with swallowing	Moves with tongue protrusion
Thyroid swelling	**Yes**	No
Thyroglossal cyst	Yes	**Yes**
Lymph node, dermoid cyst	No	No
Subhyoid bursa	Yes	No (above the thyroid cartilage)

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How to present

EXAMPLE

"This 35-year-old woman has a swelling in the lower anterior neck which moves up with deglutition and not with protrusion of the tongue. Both lobes and the isthmus are uniformly enlarged, smooth, soft to firm and non-tender, with a bruit over both upper poles. I can get below the swelling, the trachea is central, Kocher's test is negative and carotids are palpable. There are no cervical lymph nodes. She has a pulse of 112/min, warm moist palms, fine tremor and lid retraction with lid lag. My diagnosis is diffuse toxic goitre — Graves' disease, WHO grade 2."

Types of goitre — clinical classification

Type	Examples
Diffuse, euthyroid	Physiological (puberty, pregnancy), **iodine deficiency** (simple colloid goitre)
Diffuse, toxic	**Graves' disease**
Diffuse, hypothyroid / variable	**Hashimoto's thyroiditis** (firm, bosselated), subacute thyroiditis (painful, tender)
Multinodular	Non-toxic multinodular goitre; **toxic multinodular goitre** (Plummer's disease — no eye signs)
Solitary nodule	Dominant nodule of MNG, adenoma (toxic — hot), cyst, **carcinoma**
Malignant	Papillary (young, nodes), follicular (blood spread — bone, lung), medullary (MEN 2, calcitonin), anaplastic (elderly, rapid, hard, fixed), lymphoma (Hashimoto's)

Features suggesting malignancy in a thyroid swelling

- Rapid growth, **hard**, irregular, **fixed**, **hoarseness** (vocal cord palsy), **cervical lymphadenopathy**, absent carotid pulsation, Horner's syndrome, history of irradiation, age <20 or >60, male sex, bone pain

Differential diagnosis of a midline neck swelling

- Thyroglossal cyst (moves with tongue protrusion), dermoid cyst (no movement), sublingual dermoid, enlarged pyramidal lobe, subhyoid bursitis, lymph node (Delphian)

Investigations

- **TSH**, free T4 (± free T3); **anti-TPO** (Hashimoto's), **TRAb** (Graves')
- **Ultrasound neck** (nodules — TI-RADS features: hypoechoic, microcalcification, taller-than-wide, irregular margins), **FNAC** (Bethesda classification)
- Radionuclide scan (hot vs cold nodule) when TSH is low; X-ray neck / CT for retrosternal extent and tracheal compression; indirect laryngoscopy (vocal cords) before surgery

FNAC — Bethesda system

Class	Category	Usual action
I	Non-diagnostic	Repeat FNAC under USG
II	Benign	Follow-up
III	Atypia of undetermined significance	Repeat FNAC / molecular test
IV	Follicular neoplasm	Lobectomy (capsular invasion decides)
V	Suspicious for malignancy	Surgery
VI	Malignant	Total thyroidectomy ± neck dissection

EXAMINER ASKS

Why does the thyroid move with deglutition? · Berry's sign · Retrosternal goitre — signs · Hot vs cold nodule · Which thyroid cancer spreads by blood? · Why check vocal cords before thyroidectomy?

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CHAPTER 25 · SHORT CASE · ENDOCRINE

Myxoedema (Hypothyroidism) — Signs

The slow, puffy, cold, hoarse patient — recognise hypothyroidism from the face, hands, voice and reflexes

THE INSTRUCTION

"Look at this patient's face", "Examine the hands" or "Test the ankle jerks". The diagnosis is often made from the end of the bed — then confirm with specific signs and look for the cause and complications.

Bio data

Name ..... **Age / Sex** .....

- Commonest in **middle-aged women** (F : M ≈ 8 : 1); congenital hypothyroidism in neonates (screening)

Quick focused history (if permitted)

- **Tiredness, lethargy**, slowness of thought and action, **cold intolerance**, **weight gain** with poor appetite, **constipation**
- Dry skin, hair loss (including **outer third of eyebrows**), hoarse voice, puffy face and eyes
- **Menorrhagia**, infertility, galactorrhoea; depression, memory loss, slow speech, hearing loss
- Tingling in hands (carpal tunnel syndrome), muscle cramps and stiffness, joint pains
- Breathlessness, swelling of legs (pericardial effusion, heart failure); snoring (sleep apnoea)
- **Cause**: previous thyroid surgery or **radioactive iodine**, neck irradiation, drugs (**amiodarone, lithium**, antithyroid drugs), goitre (Hashimoto's), postpartum, iodine deficiency; pituitary symptoms (headache, visual loss, amenorrhoea — secondary hypothyroidism)
- Other autoimmune diseases — vitiligo, pernicious anaemia, type 1 diabetes, Addison's disease

Hypothyroidism in special groups

- **Elderly** — presents as confusion, dementia, depression, falls, hypothermia, heart failure; few classical features
- **Children** — growth retardation, delayed puberty, poor school performance; **neonates** — prolonged jaundice, constipation, umbilical hernia, large tongue (cretinism) — screen at birth
- **Pregnancy** — miscarriage, pre-eclampsia, impaired neurodevelopment of the baby; needs higher thyroxine dose

General inspection — the myxoedema facies

Feature	What you see
Face	**Puffy, pale, expressionless**, periorbital puffiness, **coarse features**, yellowish tinge (carotenaemia)
Hair	**Dry, coarse, brittle**, sparse; **loss of the lateral third of the eyebrows** (Queen Anne's sign)
Skin	**Dry, rough, cold**, thick, non-pitting swelling (myxoedema), pale (anaemia)
Tongue	Large (macroglossia)
Voice	**Hoarse, croaky, slow**, deep-pitched
Movements	Slow; patient looks apathetic

Pulse ..... **BP** .....

Temperature ..... **Weight / BMI** .....

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Examination — step by step

Hands

- **Cold, dry, rough** skin, puffy fingers, brittle nails; **carpal tunnel syndrome** — Tinel's and Phalen's signs; slow-relaxing grip

Pulse and blood pressure

- **Bradycardia**, low volume; BP — **diastolic hypertension** (raised peripheral resistance), narrow pulse pressure

Neck

- **Goitre** — Hashimoto's (firm, bosselated), iodine deficiency; **scar** of thyroidectomy; no goitre in atrophic thyroiditis or post-ablation

Reflexes — the key sign

- **Delayed relaxation of the ankle jerk** ("hung-up" reflex) — best seen with the patient **kneeling on a chair**; contraction is normal, relaxation is slow

Other systems

- **CVS**: soft heart sounds, cardiomegaly — **pericardial effusion**; heart failure
- **RS**: pleural effusion; **Abdomen**: distension, ileus, ascites (rare)
- **CNS**: proximal myopathy, slow speech, cerebellar ataxia, depression, dementia, **carpal tunnel**; deafness
- Non-pitting oedema of legs; **pretibial puffiness**; anaemia

Signs that suggest a pituitary (secondary) cause

- No goitre, no myxoedematous skin, **pale smooth skin**, loss of axillary and pubic hair, **bitemporal hemianopia**, signs of hypogonadism and hypoadrenalism

Billewicz diagnostic index — bedside signs scored

- Symptoms: diminished sweating, dry skin, cold intolerance, weight gain, constipation, hoarseness, paraesthesiae, deafness
- Signs: **slow movements**, **coarse skin**, **cold skin**, **periorbital puffiness**, **slow pulse**, **delayed ankle reflex**

Myxoedema coma — the emergency

RECOGNISE IT

Elderly patient (often in winter, after infection, sedatives or stopping thyroxine) with **hypothermia**, drowsiness or coma, bradycardia, hypotension, hypoventilation (CO_2 retention), **hyponatraemia**, hypoglycaemia. Mortality is high — IV levothyroxine (or liothyronine), **IV hydrocortisone first** (until adrenal insufficiency is excluded), passive rewarming, ventilatory support, treat the precipitant.

Myxoedema and hypothyroidism — note the terms

- **Myxoedema** strictly means the non-pitting skin infiltration with glycosaminoglycans seen in severe hypothyroidism; **pretibial myxoedema** is a feature of **Graves' disease**, not of hypothyroidism

Differential diagnosis of a puffy face

Condition	Distinguishing points
Hypothyroidism	Dry coarse skin, loss of lateral eyebrows, hoarse voice, slow pulse, delayed ankle jerk
Nephrotic syndrome	Pitting oedema, periorbital puffiness in the morning, frothy urine, normal skin
SVC obstruction	Plethora, dilated neck and chest veins, non-pulsatile raised JVP, Pemberton's sign
Cushing's syndrome	Moon face, plethora, acne, hirsutism, striae, hypertension
Angioedema / allergy	Sudden, itchy, lips and tongue, urticaria
Acromegaly	Coarse features, large nose, lips and jaw, spaced teeth, large hands

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How to present

EXAMPLE

"This 52-year-old woman has a puffy, pale, expressionless face with periorbital puffiness, loss of the lateral third of the eyebrows and dry coarse hair. Her voice is hoarse and slow. The skin is cold, dry and rough. Pulse is 54/min, regular, BP 140/96 mmHg. There is a diffuse firm goitre that moves with deglutition. Ankle jerks show delayed relaxation. There are signs of bilateral carpal tunnel syndrome. My diagnosis is primary hypothyroidism, probably due to Hashimoto's thyroiditis."

Causes of hypothyroidism

Type	Causes
Primary (goitre present)	**Hashimoto's thyroiditis** (commonest in iodine-sufficient areas), iodine deficiency, drugs (amiodarone, lithium, antithyroid drugs), dyshormonogenesis
Primary (no goitre)	**Post-thyroidectomy**, post-radioactive iodine, external neck irradiation, atrophic thyroiditis, congenital agenesis
Transient	Postpartum thyroiditis, subacute (de Quervain's) thyroiditis — recovery phase
Secondary / tertiary	Pituitary tumour, Sheehan's syndrome (postpartum haemorrhage), pituitary surgery or irradiation, hypothalamic disease

Clinical associations to check

- **Anaemia** — normocytic, macrocytic (pernicious anaemia), microcytic (menorrhagia)
- **Hyperlipidaemia**, hyponatraemia, raised CK, hyperprolactinaemia, raised homocysteine
- Other autoimmune: vitiligo, type 1 diabetes, Addison's (Schmidt syndrome), coeliac disease

Investigations

Test	Interpretation
TSH	Raised in primary hypothyroidism (best screening test); low / inappropriately normal in secondary
Free T4	Low (overt); normal with high TSH = **subclinical** hypothyroidism
Anti-TPO antibodies	Positive in Hashimoto's thyroiditis
Others	CBC, lipids, sodium, CK, ECG (low voltage, bradycardia, flat T), echo (pericardial effusion), USG neck, pituitary MRI if secondary

Treatment in one glance

- **Levothyroxine** 1.6 µg/kg/day on an empty stomach; in the elderly or with ischaemic heart disease start low (**12.5–25 µg**) and increase slowly
- Recheck TSH after 6–8 weeks; increase dose in pregnancy (by ~30%); in secondary hypothyroidism monitor free T4, not TSH, and replace cortisol first

SUBCLINICAL HYPOTHYROIDISM

High TSH with normal free T4 — treat if TSH >10 mIU/L, in pregnancy or when planning pregnancy, with goitre or positive anti-TPO and symptoms; otherwise repeat TSH in 6–12 months.

EXAMINER ASKS

Why is the ankle jerk delayed? · Why carpal tunnel syndrome? · Causes of hoarseness in hypothyroidism · Subclinical hypothyroidism — when to treat? · Myxoedema coma · Why give hydrocortisone before thyroxine in pituitary failure?

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CHAPTER 26 · SHORT CASE · ENDOCRINE

Thyrotoxic Features

Restless, sweaty and thin with a fast pulse — confirm thyrotoxicosis, examine the eyes and the gland, and name the cause

THE INSTRUCTION

"Look at this patient", "Examine the eyes" or "Examine the hands". Start with the hands and pulse, then the eyes, then the neck — and name the cause (Graves' disease, toxic MNG, toxic adenoma, thyroiditis).

Bio data

Name ..... **Age / Sex** .....

- Graves' disease — young to middle-aged women; toxic multinodular goitre — elderly (often with AF, few eye signs)

Quick focused history (if permitted)

- **Weight loss despite increased appetite**, **heat intolerance**, excessive **sweating**, **palpitation**, **tremor**, anxiety, irritability, insomnia
- Frequent loose stools, oligomenorrhoea, **proximal muscle weakness** (difficulty climbing stairs, getting up from squatting)
- Eye symptoms: grittiness, watering, **protruding eyes**, double vision, pain, blurred or reduced vision (optic nerve compression)
- Breathlessness, ankle swelling — heart failure; episodes of sudden paralysis after a heavy carbohydrate meal — **thyrotoxic periodic paralysis** (Asian men)
- Neck pain with fever after a viral illness — **subacute thyroiditis**; recent delivery — postpartum thyroiditis; iodine contrast, amiodarone, excess thyroxine intake
- Family history of thyroid disease; other autoimmune diseases (vitiligo, type 1 diabetes, myasthenia)

General inspection

- Thin, anxious, restless, fidgety; warm flushed skin; staring eyes; **exophthalmos**; visible goitre

Pulse (rate, rhythm) ..... **BP** .....

Temperature ..... **Weight** .....

The hands

- **Warm, moist** palms; **palmar erythema**; **fine tremor** of outstretched hands (place a sheet of paper on them)
- **Onycholysis** (Plummer's nails — separation of the nail from the bed, ring finger); **thyroid acropachy** (clubbing, Graves')
- **Pulse**: tachycardia (sleeping pulse >90), **atrial fibrillation** (elderly, toxic MNG), collapsing quality; wide pulse pressure (systolic hypertension)

Past and treatment history

- Antithyroid drugs (dose, duration, relapses, side effects — rash, agranulocytosis, hepatitis), β-blockers, radioactive iodine, surgery
- Smoking (worsens eye disease), pregnancy and lactation plans, cardiac disease, diabetes

APATHETIC THYROTOXICOSIS

In the elderly, thyrotoxicosis may present only with weight loss, AF, heart failure, depression or apathy — no tremor, no eye signs. Always check TSH in new AF.

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The eyes — step by step

Sign	How to elicit / what you see
Lid retraction	Sclera visible **above** the upper limbus with the eye in the primary position (Dalrymple's sign)
Lid lag (von Graefe's)	Ask the patient to follow your finger downwards slowly — the upper lid lags behind the globe, exposing sclera
Exophthalmos (proptosis)	Sclera visible **below** the lower limbus; view from **above and behind** the patient's head (Naffziger's) and from the side; measure with an exophthalmometer (>20 mm abnormal)
Joffroy's sign	Absence of wrinkling of the forehead when the patient looks up
Stellwag's sign	Infrequent blinking
Möbius sign	Weakness of convergence
Ophthalmoplegia	Restricted eye movements, especially **upward gaze** (inferior rectus fibrosis); diplopia
Chemosis, conjunctival injection	Active inflammation (congestive ophthalmopathy)
Optic neuropathy	Reduced acuity, colour vision loss (red desaturation), RAPD — **sight-threatening, urgent**

CLINICAL PEARL

Lid retraction and lid lag occur in any thyrotoxicosis (sympathetic overactivity of Müller's muscle); **exophthalmos, ophthalmoplegia and pretibial myxoedema are specific to Graves' disease** (autoimmune).

The neck

- **Diffuse goitre** with a **bruit** and thrill over the upper poles — Graves'; **multiple nodules** — toxic MNG; **single nodule** with the rest impalpable — toxic adenoma; **tender** gland — subacute thyroiditis; no goitre — exogenous thyroxine, early Graves', painless thyroiditis

The rest of the examination

- **Proximal myopathy** — ask the patient to rise from a squatting position or a low chair without using hands; shoulder abduction
- Reflexes — **brisk** with fast relaxation
- **Pretibial myxoedema** — raised, firm, non-pitting, pink-brown plaques with **peau d'orange** surface on the shins (Graves')
- Heart — tachycardia, AF, flow murmur, signs of high-output failure; gynaecomastia, splenomegaly (Graves'), lymphadenopathy

Causes of exophthalmos other than Graves'

- **Unilateral**: orbital tumour, haemangioma, lymphoma, metastasis, orbital cellulitis, **cavernous sinus thrombosis**, carotid–cavernous fistula (pulsating, with bruit) — though Graves' is still the commonest cause of unilateral proptosis
- **Pseudo-exophthalmos**: high myopia, lid retraction, contralateral enophthalmos

Wayne's index (clinical score for hyperthyroidism)

- Scores symptoms (palpitation, heat intolerance, sweating, tiredness, appetite, weight change, nervousness) and signs (goitre, bruit, exophthalmos, lid retraction, lid lag, hyperkinesis, fine tremor, warm moist hands, pulse rate, AF) — **>19 toxic**, 11–19 equivocal, **<11 euthyroid**

Thyroid storm — the emergency

RECOGNISE IT

High fever, severe tachycardia / AF, heart failure, agitation, delirium, vomiting, diarrhoea, jaundice — after surgery, infection, radioactive iodine, stopping antithyroid drugs or iodine load. Treat with **propylthiouracil**, iodine 1 hour later, **propranolol**, hydrocortisone, cooling, fluids and treatment of the trigger (Burch–Wartofsky score aids diagnosis).

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How to present

EXAMPLE

"This 28-year-old woman is thin, anxious and restless. Her palms are warm and moist with a fine tremor of the outstretched hands. Pulse is 120/min, regular, with a wide pulse pressure. She has bilateral lid retraction, lid lag and exophthalmos with weakness of convergence; eye movements and visual acuity are normal. There is a diffuse, smooth, firm goitre with a bruit over both upper poles. There is proximal myopathy and pretibial myxoedema over both shins. My diagnosis is thyrotoxicosis due to Graves' disease with ophthalmopathy and dermopathy."

Causes of thyrotoxicosis

Cause	Clues
Graves' disease	Diffuse goitre with bruit, **eye signs**, pretibial myxoedema, acropachy, TRAb positive, diffuse uptake on scan
Toxic multinodular goitre	Elderly, nodular goitre, AF, heart failure, no ophthalmopathy, patchy uptake
Toxic adenoma	Single nodule, "hot" on scan with suppression of the rest
Thyroiditis (subacute, painless, postpartum)	Tender or painless gland, transient, **low uptake** on scan, high ESR (subacute)
Exogenous	Excess thyroxine, iodine (contrast, amiodarone) — low uptake, low thyroglobulin (thyroxine)
Rare	TSH-secreting pituitary adenoma, struma ovarii, hydatidiform mole / choriocarcinoma (hCG)

Thyroid eye disease — severity (EUGOGO)

- Mild (lid retraction, mild proptosis), moderate–severe (significant proptosis, diplopia, soft-tissue involvement), **sight-threatening** (optic neuropathy, corneal breakdown) — the last needs urgent steroids / decompression; smoking worsens eye disease and must stop

Investigations

- **TSH** (suppressed), **free T4 and T3** (raised; T3 toxicosis — only T3 high); **TSH receptor antibody** (TRAb)
- Radionuclide uptake scan (99mTc / I-131) — high diffuse (Graves'), hot nodule, low uptake (thyroiditis, exogenous); USG with Doppler; ESR (subacute thyroiditis); ECG (AF), CBC (neutropenia before and on antithyroid drugs), LFT

Treatment in one glance

- **Propranolol** for symptoms; **carbimazole / methimazole** (PTU in first trimester and thyroid storm) — warn about **agranulocytosis** (sore throat, fever — stop and check WBC)
- Definitive: **radioactive iodine** (avoid in pregnancy and active eye disease) or **thyroidectomy** (large goitre, compressive, suspicious nodule, pregnancy failing drugs)

Special situations

- **Pregnancy**: **PTU** in the first trimester, then carbimazole; lowest effective dose; check fetal heart rate and growth; hCG-mediated transient thyrotoxicosis in hyperemesis
- **Thyrotoxic periodic paralysis**: hypokalaemia with flaccid weakness — careful potassium, non-selective β-blocker, treat thyrotoxicosis
- **Amiodarone-induced thyrotoxicosis**: type 1 (iodine-induced, high uptake) vs type 2 (destructive thyroiditis, low uptake — steroids)

EXAMINER ASKS

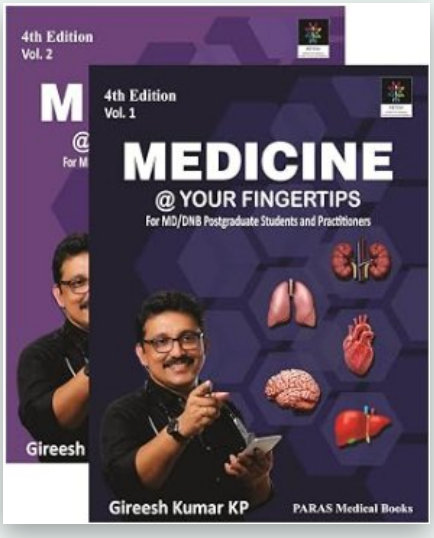
Mechanism of lid lag and lid retraction · Signs specific to Graves' disease · Toxic MNG vs Graves' · Why AF is common in the elderly · Agranulocytosis with carbimazole · Thyroid storm treatment.

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CHAPTER 27 · SHORT CASE · GENERAL

Anaemia Workup

"This patient is pale — examine him" — confirm pallor, grade it, find the type from the signs, and look for the cause

THE INSTRUCTION

"Look at this patient", "Examine for pallor" or "This patient is anaemic — examine". Pallor plus a few targeted signs — koilonychia, glossitis, icterus, nodes, spleen, bleeding spots — tell you the type and the cause.

Bio data

Name ..... **Age / Sex** .....

Diet .....

Vegetarian (B12 deficiency), poor iron intake, tea with meals

Occupation / native place .....

Barefoot farm work (hookworm), malaria and thalassaemia-prevalent areas, tribal communities (sickle cell)

Quick focused history (if permitted)

Symptoms of anaemia

- Fatigue, weakness, **breathlessness on exertion**, palpitation, giddiness, headache, tinnitus, angina or claudication (elderly), swelling of feet (high-output failure)
- **Pica** (eating mud, chalk, ice — iron deficiency), burning tongue, mouth ulcers

Symptoms that point to the cause

- **Blood loss**: menorrhagia, number of pregnancies and spacing, haematemesis, **melaena**, bleeding piles, haematuria, epistaxis; NSAID / aspirin use
- **Poor intake / absorption**: vegetarian diet, alcohol, chronic diarrhoea, gastric surgery, worm infestation, coeliac disease
- **Haemolysis**: yellow eyes, dark urine, gallstones, family history, transfusions since childhood (thalassaemia), drug-induced (G6PD — primaquine, dapsone)
- **Marrow failure / infiltration**: fever, bleeding gums, petechiae, recurrent infections, bone pain, weight loss, lumps (leukaemia, lymphoma, myeloma, aplastic anaemia)
- **B12 deficiency**: tingling and numbness of feet, unsteady gait, memory loss, vitiligo
- **Chronic disease**: kidney disease, rheumatoid arthritis, TB, hypothyroidism, chronic infections
- Previous blood transfusions, iron or B12 injections, response to treatment

General examination — confirm and grade pallor

- Look at the **lower palpebral conjunctiva**, **tongue**, **nail beds**, **palmar creases** (pale when Hb <7 g/dL), soft palate, lips

Grade (WHO, adults)	Haemoglobin
Mild	11–12.9 g/dL (men) · 11–11.9 (non-pregnant women) · 10–10.9 (pregnancy)
Moderate	8–10.9 g/dL (7–9.9 in pregnancy)
Severe	<8 g/dL (<7 in pregnancy)

Pulse ..... **BP** .....

Respiratory rate ..... **Temperature** .....

- **Hyperdynamic circulation**: tachycardia, bounding pulse, wide pulse pressure, warm peripheries, haemic (flow) murmur, raised JVP and oedema (high-output failure)

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Signs that tell the type and cause — examine systematically

Sign	Significance
Koilonychia (spoon-shaped nails), platynychia, brittle nails	**Iron deficiency**
Angular stomatitis, atrophic glossitis (smooth, pale tongue)	Iron, B12, folate deficiency
Beefy red, raw tongue	B12 / folate deficiency
Lemon-yellow tint (pallor + mild icterus), vitiligo, premature greying	Pernicious anaemia (B12 deficiency)
Icterus	Haemolytic anaemia, megaloblastic anaemia (ineffective erythropoiesis), liver disease
Hyperpigmentation of knuckles and palms	B12 deficiency (in dark-skinned individuals)
Frontal bossing, malar prominence, depressed nasal bridge (chipmunk facies)	**Thalassaemia major** (extramedullary haematopoiesis)
Leg ulcers	Sickle cell disease, spherocytosis
Petechiae, purpura, gum bleeding, gum hypertrophy	Aplastic anaemia, acute leukaemia (gum hypertrophy — AML M4/M5)
Generalised lymphadenopathy	Leukaemia, lymphoma, TB, HIV
Sternal tenderness	Acute leukaemia, CML, myeloma (marrow expansion)
Splenomegaly ± hepatomegaly	Haemolysis, thalassaemia, malaria, kala-azar, leukaemia, lymphoma, myelofibrosis, portal hypertension
Bony tenderness, fractures	Myeloma, metastasis
Signs of CKD, liver disease, hypothyroidism, rheumatoid arthritis	Anaemia of chronic disease
Dental caries, poor oral hygiene, malnutrition	Nutritional anaemia

Systemic examination

Cardiovascular

- **Haemic (flow) murmur** — soft ejection systolic at the pulmonary / mitral area; cardiomegaly, S3, signs of failure in severe anaemia

Abdomen

- **Spleen and liver** (see above), epigastric tenderness or mass (gastric carcinoma), **per rectal examination** (haemorrhoids, carcinoma rectum — melaena on the glove)

Nervous system — essential in macrocytic anaemia

- **Subacute combined degeneration of the cord** (B12): loss of **vibration and position sense** in the legs, **positive Romberg's sign**, absent ankle jerks with **extensor plantars**, peripheral neuropathy, cognitive impairment, optic atrophy

Fundus

- Retinal haemorrhages (severe anaemia, leukaemia), Roth spots (endocarditis), salmon patches (sickle cell)

Others

- Pelvic examination in menorrhagia (fibroid), breast, thyroid, joints (rheumatoid arthritis)

CLINICAL PEARL

Pallor + koilonychia + no organomegaly in a woman = iron deficiency (menorrhagia, pregnancy, hookworm). Pallor + icterus + splenomegaly = haemolysis. Pallor + bleeding + fever + sternal tenderness = leukaemia. Pallor + glossitis + posterior column signs = B12 deficiency.

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How to present

EXAMPLE

"This 32-year-old woman with menorrhagia has severe pallor, koilonychia, angular stomatitis and atrophic glossitis. Pulse is 108/min, high volume, BP 110/60 mmHg. There is no icterus, lymphadenopathy, bleeding manifestation, sternal tenderness or hepatosplenomegaly. A soft ejection systolic flow murmur is heard in the pulmonary area. My diagnosis is severe anaemia, probably iron-deficiency anaemia due to chronic blood loss from menorrhagia."

Morphological classification and causes

Type (MCV)	Causes
Microcytic hypochromic (MCV <80 fL)	**Iron deficiency**, thalassaemia trait / major, anaemia of chronic disease (some), sideroblastic anaemia, lead poisoning
Normocytic normochromic (80–100 fL)	Acute blood loss, **anaemia of chronic disease**, **CKD**, haemolysis, marrow failure (aplastic), infiltration (leukaemia, myeloma), hypothyroidism
Macrocytic (MCV >100 fL)	**B12 / folate deficiency** (megaloblastic), alcohol, liver disease, hypothyroidism, reticulocytosis, myelodysplasia, drugs (methotrexate, hydroxyurea, zidovudine)

Iron deficiency vs thalassaemia trait vs chronic disease

Test	Iron deficiency	Thalassaemia trait	Chronic disease
Serum ferritin	**Low**	Normal	Normal / **high**
Serum iron / TIBC	Low / **high**	Normal / normal	Low / **low**
RBC count	Low	**Normal or high**	Low
Mentzer index (MCV ÷ RBC)	>13	**<13**	—
HbA2 (HPLC)	Normal / low	**Raised (>3.5%)**	Normal

Investigations — step by step

1. **CBC** — Hb, RBC indices (MCV, MCH, MCHC, RDW), WBC and platelets (pancytopenia — aplastic, megaloblastic, hypersplenism, infiltration)
2. **Peripheral smear** — pencil cells, target cells, macro-ovalocytes with **hypersegmented neutrophils**, spherocytes, sickle cells, fragmented cells, blasts, parasites
3. **Reticulocyte count** — high in haemolysis and bleeding; low in deficiency and marrow failure
4. By type: iron studies; B12, folate; haemolysis screen (bilirubin, LDH, haptoglobin, Coombs test, G6PD, HPLC); renal, liver, thyroid function
5. For the source: stool occult blood and **ova / hookworm**, upper GI endoscopy and colonoscopy (men and post-menopausal women with iron deficiency), USG pelvis; **bone marrow** examination for unexplained cytopenias

Treatment in one glance

- Treat the cause; **oral iron** (60–120 mg elemental iron daily or alternate days, continue 3 months after Hb normalises); IV iron (ferric carboxymaltose) for intolerance, malabsorption, late pregnancy, CKD
- **B12** — IM hydroxocobalamin / cyanocobalamin (never folate alone in B12 deficiency — worsens neuropathy); folic acid 5 mg; transfusion only for severe symptomatic anaemia or heart failure (slowly, with diuretic)

EXAMINER ASKS

Where do you look for pallor? · Mechanism of koilonychia · Causes of macrocytosis without megaloblastosis · Why is icterus seen in megaloblastic anaemia? · Mentzer index · Anaemia Mukt Bharat strategy.

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CHAPTER 28 · SHORT CASE · GENERAL

Generalised Oedema

Pitting or non-pitting, where it began, and whether heart, kidney, liver, gut or nutrition is to blame

THE INSTRUCTION

"Examine this patient's legs" or "Look at this swollen patient". Demonstrate pitting, find the extent, then examine the JVP, heart, abdomen, urine and nutrition to name the cause. Bilateral oedema is systemic until proved otherwise.

Bio data

Name ..... **Age / Sex** .....

Quick focused history (if permitted)

The swelling

- **Site of onset**: face / eyelids in the morning — **renal** (nephrotic, nephritic); feet and ankles, worse in the evening — **cardiac**, venous, drugs; abdomen first, then legs — **liver** (cirrhosis)
- Progression, extent (sacrum, scrotum, abdomen, chest), pitting, pain

Clues to the system

- **Heart**: breathlessness on exertion, **orthopnoea, PND**, palpitation, chest pain, rheumatic fever
- **Kidney**: frothy urine, smoky or red urine, reduced urine output, hypertension, diabetes, nausea, itching
- **Liver**: jaundice, abdominal distension, haematemesis, melaena, alcohol, hepatitis
- **Gut / nutrition**: chronic diarrhoea (protein-losing enteropathy, malabsorption), poor diet, alcohol (wet beriberi)
- **Thyroid**: cold intolerance, weight gain, constipation, hoarse voice (myxoedema)
- **Drugs**: **amlodipine** and other calcium-channel blockers, pioglitazone, NSAIDs, steroids, oestrogens, minoxidil, gabapentin / pregabalin
- **Pregnancy** (with hypertension — pre-eclampsia); premenstrual (cyclical oedema); prolonged standing
- Filarial area: recurrent fever with painful limb swelling, scrotal swelling — lymphatic filariasis (usually asymmetrical)

General examination

Pulse ..... **BP** .....

Respiratory rate ..... **Weight** .....

- **JVP** — raised in heart failure, constrictive pericarditis, fluid overload (renal); normal or low in liver disease and hypoproteinaemia
- Pallor (CKD, nutritional), icterus and signs of liver cell failure, **facial puffiness** (renal), **anasarca**
- Signs of malnutrition: wasting, angular stomatitis, glossitis, flaky-paint dermatosis, sparse hair, muscle wasting
- Signs of hypothyroidism; thyroid swelling; Cushingoid features (steroid use)

Past and treatment history

- Heart disease, kidney disease, liver disease, diabetes, hypertension, TB (constrictive pericarditis), thyroid disease
- Diuretics (dose, response), salt and fluid intake, recent IV fluids; dialysis

Bedside approach in one line

ASK

Where did it start? Is it pitting? Is the JVP raised? Is there protein in the urine? Is the albumin low? — five questions that locate almost every case of generalised oedema.

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Examination of oedema — step by step

1. **Inspection**: puffiness of face, eyelids, legs, sacrum (bedridden patients), scrotum, abdominal wall; skin shiny, stretched; blisters, cellulitis
2. **Pitting**: press **firmly with the thumb for at least 10–15 seconds** over a bony surface (dorsum of foot, medial malleolus, shin, sacrum) — a pit that slowly refills = **pitting oedema**
3. **Extent**: work upwards — ankle, mid-shin, knee, thigh, sacrum, abdominal wall, chest; grade it
4. **Sacral oedema**: always examine the sacrum in a lying patient — fluid gravitates there
5. Compare both sides — **asymmetry** suggests a local cause (DVT, cellulitis, lymphatic obstruction)
6. Temperature and tenderness of the skin; Stemmer's sign (cannot pinch the skin on the dorsum of the second toe — lymphoedema)

Grading of pitting oedema (commonly used scale)

Grade	Pit depth	Rebound
1+	2 mm	Disappears immediately
2+	4 mm	Disappears in 10–15 seconds
3+	6 mm	Lasts more than 1 minute
4+	8 mm	Lasts 2–5 minutes

Pitting vs non-pitting oedema

Type	Causes
Pitting	Low protein (nephrotic, cirrhosis, malnutrition, protein-losing enteropathy), raised venous pressure (heart failure, renal salt retention), drugs, pregnancy
Non-pitting	**Lymphoedema** (filariasis, malignancy, surgery, congenital — Milroy), **myxoedema** (hypothyroidism), **pretibial myxoedema** (Graves'), lipoedema (fat), chronic venous lymphoedema

Systemic examination — find the cause

Cardiovascular

- **Raised JVP**, hepatojugular reflux, **S3**, murmurs (valvular disease), cardiomegaly, basal crackles, pleural effusion — **congestive heart failure**
- Raised JVP with **Kussmaul's sign**, pericardial knock, quiet heart, ascites out of proportion to oedema — **constrictive pericarditis** (TB in India)

Abdomen

- **Ascites**, splenomegaly, dilated veins, shrunken liver — **cirrhosis**; tender pulsatile liver — TR / CCF
- Ballotable kidneys (ADPKD), renal angle tenderness; pelvic mass (venous / lymphatic compression)

Respiratory

- Bilateral pleural effusions (any cause of generalised oedema); signs of COPD with cor pulmonale

Urine (bedside)

- Dipstick: **3+/4+ protein** — nephrotic; blood + protein — nephritic; froth on shaking

CLINICAL PEARL

Raised JVP points to the heart or the kidney (or pericardium); a normal JVP with generalised oedema points to low albumin — kidney (nephrotic), liver or gut and nutrition.

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How to present

EXAMPLE

"This 55-year-old man has bilateral pitting pedal oedema extending up to the knees, grade 3, with sacral oedema. JVP is raised 8 cm above the sternal angle with a positive hepatojugular reflux. The apex is displaced to the 6th space in the anterior axillary line, there is an S3 gallop and bilateral basal crackles; the liver is tender and palpable 4 cm. Urine shows trace protein. My diagnosis is generalised oedema due to congestive cardiac failure, probably due to dilated / ischaemic cardiomyopathy."

Causes of generalised oedema — the systems approach

System	Causes	Key bedside clue
Heart	**Congestive heart failure**, constrictive pericarditis, cor pulmonale	**Raised JVP**, S3, orthopnoea
Kidney	**Nephrotic syndrome**, acute nephritic syndrome, CKD, AKI with overload	**Facial puffiness**, proteinuria, hypertension
Liver	**Cirrhosis**	Ascites first, liver cell failure signs, normal JVP
Gut / nutrition	Malnutrition (kwashiorkor), protein-losing enteropathy, malabsorption	Wasting, diarrhoea, low albumin, normal urine
Endocrine	Hypothyroidism (myxoedema), Cushing's	Non-pitting, slow reflexes; moon face
Drugs	Calcium-channel blockers, pioglitazone, NSAIDs, steroids	Drug history, pedal oedema only
Others	Pregnancy, pre-eclampsia, wet beriberi (thiamine), idiopathic cyclical oedema, capillary leak (sepsis, dengue)	Context

Unilateral (localised) oedema — causes

- **DVT**, cellulitis, ruptured Baker's cyst, venous insufficiency, **filarial lymphoedema**, lymphatic obstruction by tumour or surgery, trauma, hemiplegic limb, compartment syndrome

Mechanisms (Starling forces)

- **Raised capillary hydrostatic pressure** (heart failure, renal salt retention, venous obstruction)
- **Reduced plasma oncotic pressure** (albumin <2.5 g/dL — nephrotic, cirrhosis, malnutrition)
- **Increased capillary permeability** (sepsis, burns, allergy, dengue); **lymphatic obstruction**

Investigations

- **Urine** — protein (dipstick, 24-h / PCR), microscopy; **serum albumin**, total protein; **renal function**, electrolytes
- **LFT**, INR; **CBC**; **TSH**; **ECG, chest X-ray, echocardiography**, NT-proBNP (heart failure)
- USG abdomen (liver, kidneys, ascites), Doppler of leg veins (if asymmetrical), stool for fat / α1-antitrypsin (protein-losing enteropathy)

Treatment principle

- Treat the cause; **salt restriction**, loop diuretics (furosemide) ± spironolactone (cirrhosis, heart failure); albumin with diuretics in severe hypoalbuminaemia; leg elevation, compression for venous disease; stop the offending drug

EXAMINER ASKS

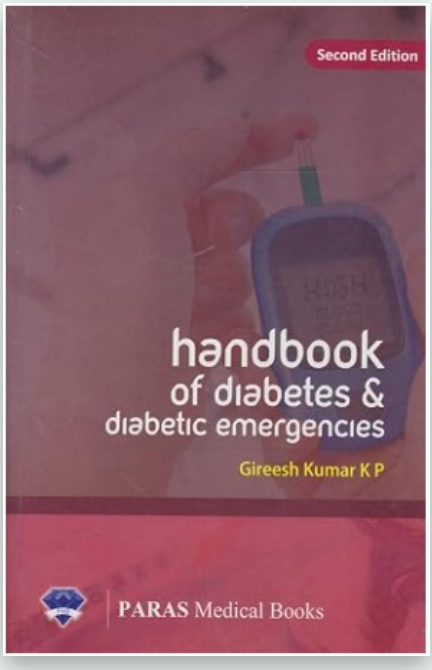
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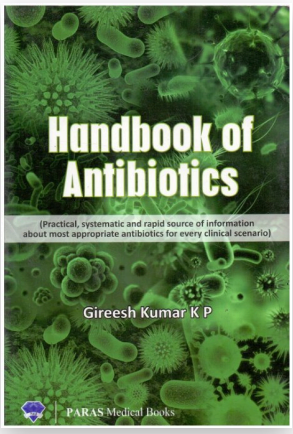
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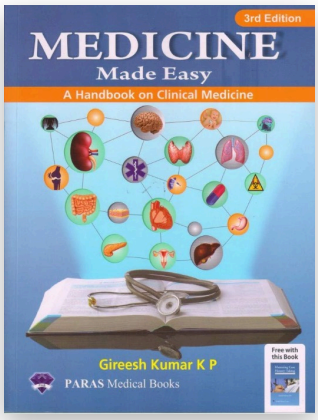
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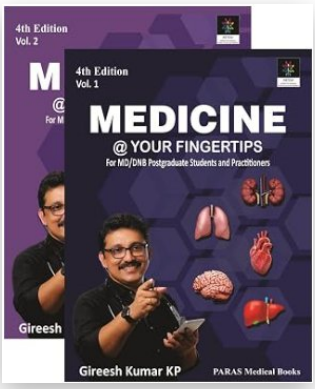
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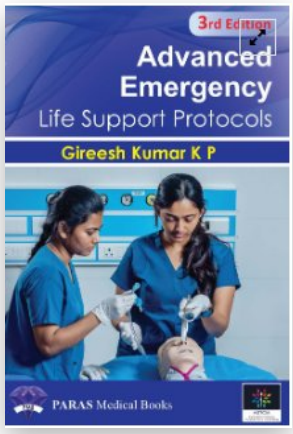
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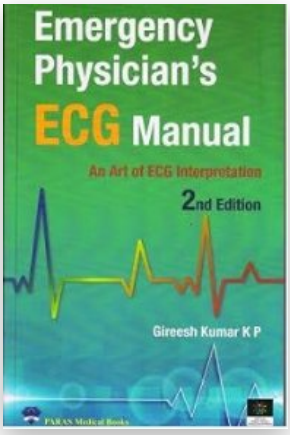
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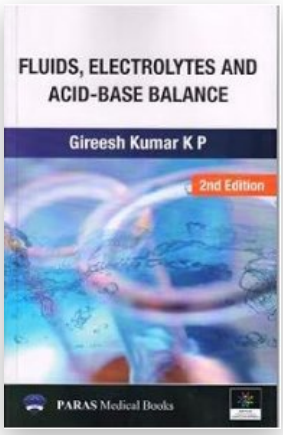
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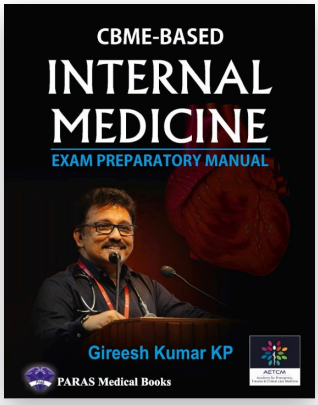
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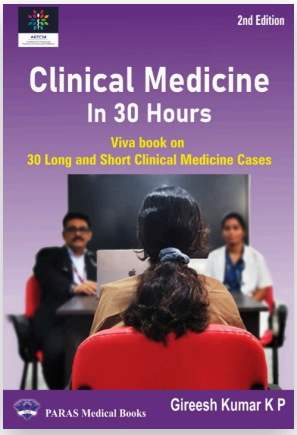
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