Approach to the Patient

Sixteen seminars that start from the presenting problem rather than the diagnosis — jaundice, anasarca, chest pain, dyspnoea, headache, arthritis, chronic cough, hypertensive emergencies, PUO, coma, venous thromboembolism, shock, chronic diarrhoea, ascites, dementia and acute poisoning.

Seminars 1–16 · 18,015 words

How Part II Differs

In Part I you were told the diagnosis in the title and the case taught you how to confirm and manage it. **That is not how patients arrive.** A patient arrives with a symptom, and the diagnosis is the thing you are trying to produce.

These cases therefore run in a different order. **The differential comes first, before any history is taken**, because the differential is what the history is for. A student who takes a history before generating a differential is collecting facts without knowing which ones matter.

Section	What it does
1. The presentation	The patient as they actually appear — usually incomplete and sometimes misleading.
2. Think first	The differential, organised by mechanism or anatomy rather than as a list. Generated before the history.
3. What could kill in the next hour	The small number of diagnoses that must be excluded or treated immediately, regardless of how likely they are.
4. The history that discriminates	Only the questions that move you between the possibilities. Not a systems review.
5. The examination that discriminates	The same principle applied to the physical examination.
6. Investigations, in tiers	Bedside, then first-line, then targeted — with the pivotal test identified.
7. Management while you are still deciding	What to do before the diagnosis is secure, which is most of the time.
8. Teaching points and viva questions	As in Part I.

Case	Seminar it serves
Seminar 1 — Jaundice	Approach to the patient with jaundice
Seminar 2 — Generalised anasarca	Approach to the patient with generalised anasarca
Seminar 3 — Acute chest pain	Approach to the patient with acute chest pain
Seminar 4 — Acute dyspnoea	Approach to the patient with acute dyspnoea; respiratory failure and hypoxaemia
Seminar 5 — Headache	Approach to the patient with headache
Seminar 6 — Arthritis	Approach to the patient with arthritis; polyarthritis
Seminar 7 — Chronic cough	Approach to the patient with chronic cough
Seminar 8 — Hypertensive urgencies and emergencies	Hypertensive urgencies and emergencies
Seminar 9 — Pyrexia of unknown origin	Approach to the patient with pyrexia of unknown origin
Seminar 10 — Coma	Approach to the patient with coma
Seminar 11 — Venous thromboembolism	Approach to the patient with venous thromboembolism; pulmonary thromboembolism
Seminar 12 — Shock	Shock, its types and management
Seminar 13 — Chronic diarrhoea	Approach to the patient with chronic diarrhoea
Seminar 14 — Ascites	Approach to the patient with ascites
Seminar 15 — Dementia	Approach to the patient with dementia
Seminar 16 — Acute poisoning	Approach to the patient with acute poisoning

Seminar 1 · Approach to the Patient with Jaundice

THE PRESENTATION

A **58-year-old man** has become progressively yellow over three weeks. He itches intolerably, particularly at night. His stools have become pale and his urine dark. He has lost 8 kg without trying. **He has had no pain and no fever.**

On examination: deeply jaundiced with scratch marks over the trunk. **The gallbladder is palpable and non-tender.** The liver edge is firm. There are **no spider naevi, no palmar erythema and no ascites**. He is fully alert with no asterixis.

Bilirubin 280 µmol/L, predominantly conjugated · alkaline phosphatase 640 · gamma-glutamyl transferase 780 · alanine aminotransferase 92 · albumin 34 g/L · INR 1.1.

1. Think First — Three Compartments, Three Questions

Compartment	Bilirubin	Causes
Pre-hepatic	Unconjugated	Haemolysis of any cause, ineffective erythropoiesis, Gilbert syndrome. Liver enzymes are normal, the stools are normal in colour, and there is no bilirubin in the urine because unconjugated bilirubin is not water-soluble.
Hepatic	Mixed, hepatitic enzyme pattern	Viral hepatitis, drugs and toxins, alcohol, autoimmune hepatitis, ischaemic hepatitis, Wilson disease, haemochromatosis, cirrhosis, infiltration, severe sepsis.
Post-hepatic	Conjugated, cholestatic pattern	Gallstones, carcinoma of the head of the pancreas, cholangiocarcinoma, benign stricture, primary biliary cholangitis, primary sclerosing cholangitis, drug-induced cholestasis, nodal compression.

Answer three questions in order, and the diagnosis narrows at each step:

- **Is the bilirubin conjugated or unconjugated?** This separates haemolysis and Gilbert syndrome from everything else, and it costs one extra box on the request form.
- **Is the enzyme pattern hepatitic or cholestatic?** Aminotransferases out of proportion means the hepatocytes; alkaline phosphatase and gamma-glutamyl transferase out of proportion means the biliary tree.
- **Are the bile ducts dilated?** This is what the ultrasound is for, and it is the pivotal investigation in the whole assessment.

2. What Could Kill in the Next Hour

ASCENDING CHOLANGITIS

Charcot triad: fever, right upper quadrant pain and jaundice. Add hypotension and confusion and it becomes **Reynolds pentad**, which carries a high mortality.

An obstructed, infected biliary tree is a closed-space infection under pressure. **Antibiotics alone do not treat it — it must be drained**, urgently by endoscopic retrograde cholangiopancreatography or percutaneously, within hours in a septic patient.

This patient is afebrile and painless, which is precisely why he is **not** in this category — but you establish that deliberately rather than by default.

- **Acute liver failure** — jaundice with encephalopathy and a rising INR. See Case 35.
- **Paracetamol toxicity** — check a level in every jaundiced patient regardless of the history.

- **Severe haemolysis**, and **sepsis** with jaundice as a manifestation rather than a cause.

3. The History That Discriminates

Ask	What the answer tells you
Is there pain, and what kind?	Colicky right upper quadrant pain radiating to the back or shoulder suggests stones. Painless progressive jaundice with weight loss is malignant obstruction until proved otherwise.
Pale stools, dark urine and itch?	Cholestasis — bile is not reaching the gut. Their absence with dark urine but normal stools suggests haemolysis.
Fever or rigors?	Cholangitis, or hepatitis with a systemic illness.
Every drug, herbal and traditional preparation, and paracetamol	Drug-induced liver injury is common and under-recognised, and the culprit is often not prescribed.
Alcohol quantified; travel; unpasteurised dairy; transfusion; tattoos; sexual history	Alcoholic liver disease, viral hepatitis, brucellosis.
Previous biliary surgery, pancreatitis, inflammatory bowel disease	Stricture; primary sclerosing cholangitis, which is associated with ulcerative colitis.
Family history of jaundice, anaemia or liver disease; age under 40	Gilbert syndrome, hereditary haemolytic anaemia, Wilson disease.

4. The Examination That Discriminates

COURVOISIER'S SIGN

A palpable, non-tender gallbladder in a jaundiced patient makes gallstones an unlikely cause.

The reasoning is sound: a gallbladder chronically diseased by stones is fibrotic and cannot distend. A gallbladder that distends is therefore obstructed by something **downstream and recent** — characteristically a carcinoma of the head of the pancreas.

It is not infallible, but in this patient it points the assessment firmly towards malignant obstruction.

- **The presence or absence of chronic liver disease stigmata** — spider naevi, palmar erythema, gynaecomastia, ascites. Their **absence**, as here, argues against cirrhosis and for an acute obstructive process in a previously normal liver.
- **Mental state and asterixis**, which convert this from a diagnostic problem into an emergency.
- Liver edge — smooth and tender in hepatitis, hard and irregular in malignancy or cirrhosis; splenomegaly (portal hypertension or haemolysis); **left supraclavicular node**; abdominal masses; ascites.
- Pallor in haemolysis; **Kayser–Fleischer rings** in any patient under 40; scratch marks; xanthelasma in chronic cholestasis.

5. Investigations, in Tiers

- **Bedside:** urinalysis — **bilirubin in the urine means it is conjugated**; capillary glucose.
- **First line:** split bilirubin, full liver enzyme profile, **INR and albumin as the measures of function**, complete blood count with reticulocytes and blood film, haptoglobin and lactate dehydrogenase, direct

antiglobulin test if unconjugated, amylase, renal function, **paracetamol level**, viral hepatitis serology, autoimmune screen with immunoglobulins, and — under 40 — caeruloplasmin and iron studies.

THE PIVOTAL TEST

Ultrasound of the abdomen is the investigation that divides the problem in two, and it should be obtained early.

Ducts dilated → the obstruction is mechanical. Proceed to magnetic resonance cholangiopancreatography or endoscopic ultrasound to define the level and cause, and computed tomography for staging.

Ducts not dilated → the problem is within the hepatocytes or the small intrahepatic ducts. Pursue serology, autoantibodies and, where necessary, liver biopsy.

- **Magnetic resonance cholangiopancreatography is diagnostic; endoscopic retrograde cholangiopancreatography is therapeutic.** Do not use the latter as a purely diagnostic test — it carries a real risk of post-procedure pancreatitis and should be reserved for when you intend to intervene.
- CA 19-9 rises in biliary obstruction of any cause, including benign, so **interpret it only after the obstruction is relieved** and never as a screening test.

6. Management While You Are Still Deciding

- **Vitamin K** for a prolonged prothrombin time — in obstructive jaundice the deficiency is of absorption, and it corrects.
- **Cholangitis: broad-spectrum antibiotics and urgent biliary drainage.** Do not wait for the fever to settle before referring for drainage.
- **Maintain hydration and monitor renal function.** Jaundiced patients are prone to acute kidney injury, particularly around contrast and procedures.
- **Treat the itch** — it is genuinely disabling and is often ignored. Cholestyramine, with other agents where that fails.
- Nutrition, including fat-soluble vitamins; stop hepatotoxic drugs; and refer to the multidisciplinary team where malignancy is suspected, for staging and for **biliary stenting**, which relieves symptoms whether or not resection is possible.

7. Teaching Points and Viva Questions

- Split the bilirubin, read the enzyme pattern, then ask the ultrasound whether the ducts are dilated.
- Painless jaundice with weight loss is malignancy until proved otherwise.
- A palpable non-tender gallbladder points away from stones.
- Fever plus pain plus jaundice means the biliary tree needs draining, not just antibiotics.
- The INR and albumin tell you how the liver is working. The bilirubin does not.
- Endoscopic retrograde cholangiopancreatography is a treatment, not an investigation.

Questions you should be able to answer:

- His urine contains bilirubin. What does that exclude?
- Explain the reasoning behind Courvoisier's sign.
- The ultrasound shows no duct dilatation. How does your plan change?
- A different patient has jaundice, fever and rigors with a blood pressure of 84/50. What are your first three actions?
- A 24-year-old has an isolated unconjugated bilirubin of 45 with normal enzymes after a period of fasting. What is the diagnosis?

Seminar 2 · Approach to the Patient with Generalised Anasarca

THE PRESENTATION

A **42-year-old man** describes six weeks of progressive swelling. He first noticed **puffiness around his eyes on waking**; over the following weeks his legs, scrotum and abdomen have swollen. His urine has been **frothy**. He is breathless lying flat and has gained 12 kg.

On examination: periorbital oedema, pitting oedema to the sacrum, scrotal oedema, and ascites with shifting dullness. Blood pressure 152/94 mmHg. **The jugular venous pressure is not raised.** There are no spider naevi and no palmar erythema. Chest: dullness at both bases.

Urine dipstick: protein ++++, no blood. Urine albumin-to-creatinine ratio grossly raised · serum albumin 18 g/L · cholesterol markedly raised · creatinine normal.

1. Think First — Four Mechanisms

Oedema forms when the Starling forces are disturbed. There are only four ways to do that, and every cause sits in one of them.

Mechanism	Causes
Raised hydrostatic pressure	Heart failure, constrictive pericarditis, venous obstruction, renal failure with fluid overload, drugs — calcium channel blockers, non-steroidal anti-inflammatory drugs, thiazolidinediones
Reduced oncotic pressure	Nephrotic syndrome, cirrhosis, protein-losing enteropathy, severe malnutrition
Increased capillary permeability	Sepsis, anaphylaxis and angioedema, burns, systemic inflammatory states
Lymphatic obstruction	Malignancy, post-surgical or post-radiotherapy lymphoedema, filariasis

Add two that fit awkwardly: **hypothyroid myxoedema**, which is non-pitting, and pregnancy.

THE JUGULAR VENOUS PRESSURE IS THE SINGLE MOST USEFUL DISCRIMINATOR

Look at the neck before you look at the ankles.

Raised → the problem is on the venous side: heart failure, constrictive pericarditis, fluid overload from renal failure, or tamponade.

Normal or low → the problem is oncotic or permeability: **nephrotic syndrome, cirrhosis, malnutrition, protein-losing enteropathy**.

Then add the timing: **periorbital oedema worst on waking suggests a renal cause**; ankle oedema worst by evening suggests a cardiac or venous one; and unilateral swelling is local until proved otherwise.

2. What Could Kill in the Next Hour

- **Pulmonary oedema** with hypoxia; **cardiac tamponade**; **massive pulmonary embolism** — for which the nephrotic patient is at high risk; **severe hyponatraemia**; **sepsis**; and anasarca with **acute kidney injury and hyperkalaemia**.

3. The History That Discriminates

- **Where did it start and when is it worst?** Face on waking versus ankles by evening. This one question separates the renal from the cardiac patient more efficiently than any test.

- **Frothy urine, haematuria, reduced urine output** — renal. **Orthopnoea, paroxysmal nocturnal dyspnoea, chest pain, palpitations** — cardiac.
- **Jaundice, alcohol, known liver disease, abdominal swelling preceding leg swelling** — hepatic.
- **Diarrhoea, weight loss, malabsorption** — protein-losing enteropathy or malnutrition.
- **Drugs** — calcium channel blockers are a very common and entirely reversible cause of ankle oedema, and anti-inflammatory drugs cause fluid retention and worsen every other cause.
- **Underlying disease that causes nephrotic syndrome:** diabetes, hepatitis B and C, human immunodeficiency virus, systemic lupus erythematosus, amyloidosis, myeloma, malignancy, and recent infection.

4. The Examination That Discriminates

- **Jugular venous pressure**, weight, blood pressure, and **the urine dipstick — which you perform yourself rather than waiting for**.
- Distribution of oedema: periorbital, sacral in a bedbound patient, scrotal, and whether it pits.
- Ascites and shifting dullness; pleural effusions; third heart sound and murmurs; pulsatile hepatomegaly; **Kussmaul sign** for constrictive pericarditis.
- Stigmata of chronic liver disease; thyroid examination; lymphadenopathy; and xanthelasma, which accompanies the hyperlipidaemia of nephrotic syndrome.

5. Investigations, in Tiers

- **Bedside: urine dipstick, and a urine protein-to-creatinine or albumin-to-creatinine ratio.** This single test immediately separates the renal cause from all the others, and it is available within minutes.
- **First line:** serum albumin and total protein, liver and renal function, lipids, complete blood count, C-reactive protein, thyroid function, glucose, **natriuretic peptide**, chest radiograph, electrocardiogram.
- **Targeted — if nephrotic:** hepatitis B and C, human immunodeficiency virus, antinuclear antibody, anti-double-stranded DNA and complement, antineutrophil cytoplasmic antibodies, **serum immunoglobulins with protein electrophoresis and serum free light chains** to exclude myeloma and amyloidosis, and **renal biopsy — which in an adult determines the treatment** and is not optional.
- **Targeted — otherwise:** echocardiography, abdominal ultrasound, and faecal alpha-1 antitrypsin clearance for protein-losing enteropathy.

NEPHROTIC SYNDROME — DEFINITION AND CONSEQUENCES

Proteinuria above 3.5 g in 24 hours · serum albumin below 30 g/L · oedema · usually with hyperlipidaemia.

The complications are what harm the patient, and they are predictable:

Venous thromboembolism, including **renal vein thrombosis** — loss of antithrombin in the urine makes these patients strikingly prothrombotic · **infection**, particularly pneumococcal, from urinary loss of immunoglobulins · acute kidney injury · hyperlipidaemia · vitamin D deficiency.

6. Management While You Are Still Deciding

- **Salt restriction and fluid restriction**, with **daily weights** as the measure of response.
- **Loop diuretic — and expect to need more than usual.** Gut wall oedema impairs absorption of oral diuretics, so intravenous administration or a higher dose is frequently required, sometimes combined with a thiazide for sequential blockade.
- **An angiotensin-converting enzyme inhibitor or receptor blocker to reduce proteinuria**, with monitoring of potassium and creatinine.
- **Statin** for the hyperlipidaemia; **thromboprophylaxis, and formal anticoagulation where the albumin is very low** or there is another risk factor.
- **Vaccination**, including pneumococcal; treat infection promptly and with a low threshold.
- **Then treat the cause**, which in an adult means acting on the biopsy — immunosuppression for the primary glomerular diseases, and treatment of the underlying disorder in secondary causes.

7. Teaching Points and Viva Questions

- Look at the neck before the ankles. The jugular venous pressure divides the differential in two.
- Puffy eyes in the morning is a renal history; swollen ankles in the evening is a cardiac one.
- Dipstick the urine yourself, in the first five minutes.
- Nephrotic patients clot, and they get infected. Anticipate both.
- Oral diuretics are poorly absorbed through an oedematous gut.
- In an adult, the biopsy determines the treatment.

Questions you should be able to answer:

- His jugular venous pressure is normal. Which half of your differential does that remove?
- Why is he at risk of thrombosis, and what would you do about it?
- He is not responding to 40 mg of oral furosemide. Give two reasons and your next step.
- What must be excluded before you attribute this to a primary glomerular disease?
- A different patient has ankle oedema, a normal albumin and a normal jugular venous pressure, and takes amlodipine. What is your first move?

Seminar 3 · Approach to the Patient with Acute Chest Pain

THE PRESENTATION

A **62-year-old woman** presents with two hours of central chest discomfort which she describes as a heaviness radiating to her left arm. She is sweaty and nauseated. She also reports that it is worse on deep inspiration, and she has felt breathless since it began.

She had a knee replacement three weeks ago and flew home last week.

On examination: heart rate 108/min, blood pressure 132/84 mmHg in the right arm and 128/82 mmHg in the left, respiratory rate 24/min, **oxygen saturation 93% on air**. Chest is clear. Heart sounds normal. The right calf is slightly swollen.

Electrocardiogram: sinus tachycardia with T wave inversion in V1–V4. High-sensitivity troponin mildly raised.

1. Think First — The Six That Kill, Then Everything Else

Diagnosis	The story and the sign	The test
Acute coronary syndrome	Heavy, exertional, radiating to arm or jaw, with sweating and nausea	Electrocardiogram and serial troponin
Aortic dissection	Tearing pain, maximal at the very onset, radiating to the back; pulse or blood pressure differential; new aortic regurgitation; widened mediastinum	Computed tomographic aortogram
Pulmonary embolism	Pleuritic pain with breathlessness; hypoxia with a clear chest; risk factors; tachycardia	Computed tomographic pulmonary angiography
Tension pneumothorax	Sudden, unilateral hyper-resonance with absent breath sounds, tracheal deviation, shock	None — this is a clinical diagnosis. Decompress first
Oesophageal rupture	Severe pain after forceful vomiting; surgical emphysema; pleural effusion	Computed tomography with contrast
Cardiac tamponade	Raised jugular venous pressure, muffled heart sounds, hypotension, pulsus paradoxus	Echocardiography

Then the rest: pericarditis, myocarditis, pneumonia and pleurisy, gastro-oesophageal reflux and oesophageal spasm, musculoskeletal and costochondral pain, **herpes zoster before the rash appears**, biliary and pancreatic pain referred to the chest, and anxiety — which is a diagnosis of exclusion, not a first impression.

2. What Could Kill in the Next Hour

- **Electrocardiogram within 10 minutes**, repeated if the first is unremarkable and the pain continues.
- Oxygen only if hypoxic; intravenous access; continuous monitoring beside a defibrillator; analgesia.

BEFORE THE ASPIRIN

Take the blood pressure in both arms and feel all the peripheral pulses before you give aspirin, an antiplatelet or an anticoagulant.

Giving antithrombotic therapy to a patient with an aortic dissection is among the most damaging errors in acute medicine, and dissection presents as "chest pain radiating down the arm" often enough to catch people out.

Features that should stop you: tearing pain that was maximal at onset, radiation to the back, a pulse or pressure differential, a new early diastolic murmur, neurological signs, or a widened mediastinum.

3. The History That Discriminates

- **How quickly did it reach its worst?** Instantaneous and maximal at onset suggests dissection or pneumothorax. Crescendo over minutes suggests ischaemia.
- **Character and radiation.** Heaviness to the arm or jaw suggests ischaemia; tearing to the back suggests dissection; sharp and pleuritic suggests pleura, pericardium or embolism.
- **What changes it?** Worse on inspiration — pleuritic. Relieved by sitting forward — pericarditis. Reproduced by movement or palpation — musculoskeletal, though this does **not** exclude a serious cause. Related to food or lying flat — oesophageal.
- **What came with it?** Sweating, nausea and vomiting favour ischaemia. Syncope raises dissection, embolism and arrhythmia. **Vomiting immediately before the pain began** raises oesophageal rupture.
- **Risk factors for each of the six**, taken deliberately — including **recent surgery, immobility and long-haul travel** for embolism, as in this patient, and cocaine use in a younger one.

4. The Examination That Discriminates

- **Both arms, all pulses, respiratory rate and oxygen saturation**, perfusion and conscious level.
- **Jugular venous pressure** — raised in tamponade, right ventricular infarction and massive embolism.
- Heart sounds: a **new early diastolic murmur** suggests dissection into the aortic root; a **rub** suggests pericarditis; muffled sounds suggest tamponade. Test for pulsus paradoxus.
- Chest: tracheal position, asymmetry of expansion, percussion note, breath sounds, and **surgical emphysema**.
- Calves; abdomen; chest wall palpation; and the skin over the dermatomes for early zoster.

5. Investigations, in Tiers

- **Bedside:** electrocardiogram within 10 minutes and repeated; oxygen saturation; glucose.
- **First line:** high-sensitivity troponin using the local pathway with a repeat sample; complete blood count, renal function, coagulation; **chest radiograph**.
- **Targeted: D-dimer only where pulmonary embolism is being considered and the pre-test probability is low or intermediate** — a positive result in a high-probability patient adds nothing and a negative one cannot be relied on. Then computed tomographic pulmonary angiography, or computed tomographic aortogram, or echocardiography, as the differential dictates.

THE TRAP IN THIS CASE

A raised troponin means myocardial injury. It does not mean myocardial infarction, and it certainly does not mean coronary occlusion.

Troponin rises in **pulmonary embolism**, sepsis, heart failure, renal impairment, myocarditis, tachyarrhythmia, and after stroke or major exertion.

In this patient, a mildly raised troponin with **anterior T wave inversion, hypoxia, a clear chest, a swollen calf and recent surgery and air travel** is right heart strain from pulmonary embolism — not an anterior myocardial infarction. Anchoring on the first plausible diagnosis is the error the case is built to demonstrate.

6. Management While You Are Still Deciding

- Monitored area, oxygen to target, analgesia, and treatment of the specific diagnosis once identified — thrombolysis or thrombectomy for infarction, anticoagulation for embolism, urgent surgical referral for dissection or rupture, needle decompression for tension pneumothorax.
- **Where the diagnosis remains uncertain, the disposition decision is the clinical act.** Use a validated pathway, and if discharging, provide explicit safety-netting and arranged follow-up rather than an instruction to return if worse.
- **Remember that a patient may have two diagnoses at once** — an embolism and demand ischaemia, or pneumonia and an arrhythmia. Do not stop looking once the first explanation is found.

7. Teaching Points and Viva Questions

- Electrocardiogram in ten minutes, and repeat it if the pain persists.
- Both arms before the aspirin.
- Hypoxia with a clear chest is pulmonary embolism until proved otherwise.
- Tension pneumothorax is treated before it is imaged.
- Troponin is a marker of injury, not of a diagnosis.
- Reproducible chest wall tenderness lowers the probability of a dangerous cause but never excludes it.

Questions you should be able to answer:

- Give the six immediately life-threatening causes and one discriminating feature of each.
- Why is her troponin raised, and what does the T wave inversion represent?
- When is a D-dimer useful, and when is it worse than useless?
- What features in a history would make you withhold aspirin?
- A patient collapses with chest pain, a blood pressure of 70/40, a raised jugular venous pressure and a clear chest. Give three diagnoses.

Seminar 4 · Approach to the Patient with Acute Dyspnoea

THE PRESENTATION

A **74-year-old man** has become progressively breathless over three days and is now breathless at rest. He sleeps on four pillows and his ankles have swollen. He has also had a productive cough with green sputum and has felt feverish.

He has documented chronic obstructive pulmonary disease and documented heart failure, and takes inhalers, furosemide and bisoprolol.

On examination: speaking in short phrases. Respiratory rate 30/min, **oxygen saturation 86% on air**, heart rate 118/min and **irregularly irregular**, blood pressure 108/68 mmHg, temperature 38.1 °C. **The jugular venous pressure is raised.** There are widespread crackles **and** expiratory wheeze. Pitting oedema to both knees.

1. Think First — Follow the Oxygen

Level	Causes
Airway	Foreign body, anaphylaxis and angioedema, epiglottitis, laryngeal tumour — stridor is the sign
Lungs	Asthma, chronic obstructive pulmonary disease, pneumonia, pneumothorax, pleural effusion, acute respiratory distress syndrome, interstitial lung disease
Circulation	Pulmonary oedema, acute coronary syndrome, arrhythmia, pulmonary embolism, tamponade, valve disease
Blood	Anaemia, carbon monoxide poisoning, methaemoglobinaemia
Metabolic and neuromuscular	Metabolic acidosis — ketoacidosis, sepsis, renal failure, salicylate; neuromuscular weakness; chest wall restriction
Psychogenic	Hyperventilation — a diagnosis of exclusion and never a first impression

Four questions sort most patients within a minute of walking to the bedside:

- **Is there stridor, or wheeze, or neither?** Stridor is upper airway and is an emergency; wheeze is lower airway; neither points elsewhere.
- **Is the chest clear despite hypoxia?** Then think pulmonary embolism, anaemia, acidosis, or shunt.
- **Is the jugular venous pressure raised?** Cardiac, embolic or tamponade.
- **Is it unilateral?** Pneumothorax, effusion or consolidation.

2. What Could Kill in the Next Hour

- **Sit the patient up. Give oxygen to an explicit target** — 94–98%, or **88–92% where there is a risk of carbon dioxide retention**, written on the chart. Monitoring, intravenous access, and senior help early.
- **Look immediately for the causes that are reversible within minutes:** tension pneumothorax, anaphylaxis, upper airway obstruction, and opioid toxicity.

3. The History That Discriminates

- **Speed of onset is the most informative single item.** Seconds to minutes — pneumothorax, embolism, aspiration, anaphylaxis, arrhythmia. Hours — asthma, pulmonary oedema, pneumonia. Days to weeks — effusion, anaemia, interstitial disease, malignancy.

- **Orthopnoea and paroxysmal nocturnal dyspnoea** favour a cardiac cause; **fever and purulent sputum** an infective one; **pleuritic pain and unilateral leg swelling** an embolic one; **wheeze with a known diagnosis** an airways one.
- **Weight gain, ankle swelling, and any recent change to diuretic dose or adherence** — a very common and entirely correctable precipitant.
- Exposure history, occupational history, drugs, and the patient's **baseline exercise tolerance**, without which "breathless" cannot be quantified.

4. The Examination That Discriminates

- Respiratory rate counted, saturation **on air** with the device and flow recorded, ability to complete a sentence, accessory muscle use, cyanosis, conscious level.
- **Jugular venous pressure**; tracheal position; percussion and auscultation comparing sides; heart sounds, third sound and murmurs; peripheral oedema and calves; peak flow; temperature; capillary glucose.

5. Investigations, in Tiers

- **Bedside:** arterial or venous blood gas — which tells you the **type of respiratory failure, whether there is acidosis, the lactate, and the bicarbonate as a marker of chronicity**; electrocardiogram; glucose; peak flow.
- **First line:** chest radiograph, complete blood count, renal function, C-reactive protein, **natriuretic peptide**, troponin.
- **Targeted:** computed tomographic pulmonary angiography where embolism is suspected; echocardiography; sputum and blood cultures; carboxyhaemoglobin.
- **Bedside ultrasound** is increasingly the fastest discriminator available — B-lines of pulmonary oedema, pleural effusion, absent lung sliding in pneumothorax, gross cardiac function and inferior vena cava filling, all in a few minutes at the trolley.

THE POINT OF THIS CASE

This man has **at least three simultaneous problems**: an infective exacerbation of his airways disease, decompensated heart failure, and new atrial fibrillation — each worsening the others.

The commonest error in the breathless elderly patient is to find one diagnosis and stop. **Treat the contributors in parallel** — controlled oxygen, bronchodilators, antibiotics, diuresis, rate control — and reassess frequently, rather than debating which single label is correct.

Decide and **document the ceiling of care early**, while the patient can still participate in the conversation.

6. Management While You Are Still Deciding

- Controlled oxygen to a written target, with a repeat gas after 30–60 minutes.
- Treat the most likely and the most dangerous simultaneously, then **subtract** treatments as the picture clarifies rather than adding indefinitely.

- Continuous positive airway pressure for cardiogenic pulmonary oedema, or non-invasive ventilation for hypercapnic acidosis, with early critical care involvement.
- Reassess after every intervention. Improvement or its absence is itself diagnostic information.

7. Teaching Points and Viva Questions

- Write the target saturation range on the chart. "Give oxygen" is not a prescription.
- Speed of onset narrows the differential faster than any investigation.
- Hypoxia with a clear chest, and the jugular venous pressure, are the two highest-yield observations.
- Elderly patients are allowed more than one diagnosis, and usually have them.
- Hyperventilation is what you conclude at the end, never what you assume at the start.

Questions you should be able to answer:

- What oxygen target would you set for this man, and why?
- Name the three processes contributing to his breathlessness and the treatment for each.
- His gas shows pH 7.28, carbon dioxide 8.4 kPa, bicarbonate 31. Interpret it.
- A 24-year-old is acutely breathless with a clear chest and normal saturations. What must you exclude before diagnosing hyperventilation?
- Which four bedside ultrasound findings would you look for, and what would each tell you?

Seminar 5 · Approach to the Patient with Headache

THE PRESENTATION

A **48-year-old woman** developed a severe occipital headache while lifting a heavy box. **It reached its maximum intensity within seconds** and she describes it as the worst headache of her life. She vomited twice and now dislikes the light. Six hours later it has eased somewhat but has not gone.

On examination: Glasgow Coma Scale 15, no focal neurological deficit. There is neck stiffness. Blood pressure 168/96 mmHg, temperature 36.9 °C. Fundoscopy shows no papilloedema.

THE SINGLE MOST IMPORTANT QUESTION IN THIS CONSULTATION

"How long did it take to reach its worst?" is the question that matters — not "how bad is it?"

Almost everybody with a severe headache says it is the worst they have had. **Thunderclap headache — maximum intensity within about one minute — is the feature that demands investigation**, and it means subarachnoid haemorrhage until proved otherwise.

Partial improvement over hours is entirely compatible with subarachnoid haemorrhage and provides no reassurance at all.

1. Think First — Primary and Secondary

- **Primary headache disorders:** migraine with and without aura, tension-type headache, and the trigeminal autonomic cephalalgias including cluster headache.
- **Secondary headache** is where the danger lies:

Cause	The pattern that suggests it
Subarachnoid haemorrhage	Thunderclap onset, vomiting, neck stiffness, may have no focal signs at all
Meningitis or encephalitis	Fever, neck stiffness, rash, altered consciousness, seizures, personality change
Giant cell arteritis	Age over 50, new headache, scalp tenderness, jaw claudication, visual symptoms, raised inflammatory markers
Raised intracranial pressure or a mass lesion	Worse on waking, worse on coughing, bending or straining; vomiting; papilloedema; focal signs; new seizure
Cerebral venous sinus thrombosis	Subacute progressive headache, pregnancy or the puerperium, prothrombotic state, seizures, papilloedema
Arterial dissection	Neck pain, Horner syndrome, after trauma or neck manipulation
Acute angle-closure glaucoma	Painful red eye, haloes around lights, fixed mid-dilated pupil, vomiting
Carbon monoxide poisoning	Other members of the household also unwell; heaters and poor ventilation
Medication overuse headache	Daily analgesia for more than 10–15 days per month
	THE RED FLAGS — SNOOP **S** — **S**ystemic features: fever, weight loss, malignancy, immunosuppression, pregnancy **N** — **N**eurological deficit, seizure, or altered consciousness **O** — **O**nset sudden: thunderclap **O** — **O**lder: a new headache over the age of 50 **P** — **P**attern change or **P**rogression; **P**ositional variation; **P**recipitated by Valsalva; **P**apilloedema

2. What Could Kill in the Next Hour

- **Bacterial meningitis** — antibiotics immediately, before imaging and before lumbar puncture (Case 44).
- **Subarachnoid haemorrhage** with rebleeding or hydrocephalus.
- **Giant cell arteritis with visual symptoms — start high-dose corticosteroids on suspicion, before the biopsy**, because the visual loss is sudden, painless and irreversible.
- Raised intracranial pressure with impending herniation; carbon monoxide poisoning; hypertensive emergency with encephalopathy.

3. The History That Discriminates

- **Time to maximum intensity**, as above. Then duration, frequency and whether the pattern has changed.
- **Positional variation:** worse lying flat and on waking suggests raised pressure; worse on standing and relieved by lying suggests low cerebrospinal fluid pressure, as after a dural puncture.
- **Triggers:** exertion, coughing, straining and sexual activity all raise the concern for haemorrhage on a first occurrence.
- **Associated features:** fever, rash, visual disturbance, diplopia, weakness, seizure, jaw pain on chewing, lacrimation and nasal congestion with a strictly unilateral orbital pain (cluster), and aura.
- **Analgesic use — how many days per month**, which identifies medication overuse headache, a very common and entirely reversible cause of daily headache.
- **Pregnancy or recent delivery; anticoagulation; immunosuppression; malignancy; recent trauma or neck manipulation; and whether anyone else at home has a headache.**

4. The Examination That Discriminates

- **Temperature and blood pressure**, conscious level, and a full neurological examination.
- **Fundoscopy for papilloedema** — omitted far more often than it is performed, and the finding changes the plan entirely.
- Neck stiffness; the skin for a non-blanching rash; **temporal artery tenderness, thickening and absent pulsation in anyone over 50**; the eye itself for redness and a mid-dilated pupil; visual acuity, fields and colour vision; and pupillary responses including Horner syndrome.

5. Investigations, in Tiers

SUSPECTED SUBARACHNOID HAEMORRHAGE — THE PATHWAY

Non-contrast computed tomography of the head, as soon as possible. Its sensitivity is very high when performed **within 6 hours** of onset and falls progressively thereafter as the blood degrades.

If the computed tomogram is negative and more than 6 hours have elapsed since onset, proceed to lumbar puncture — performed at least 12 hours after the headache began, so that **xanthochromia** has had time to develop. It is measured by spectrophotometry, not by eye.

A negative scan alone does not exclude the diagnosis in a patient presenting late, and this is the point at which the diagnosis is most often missed.

Where haemorrhage is confirmed, or suspicion persists, proceed to **computed tomographic or catheter angiography** to find the aneurysm.

- **Giant cell arteritis:** erythrocyte sedimentation rate and C-reactive protein **urgently**, then temporal artery ultrasound or biopsy within days — **the biopsy remains informative for a week or two after starting steroids, so treatment is never delayed for it.** Same-day ophthalmology assessment if there are visual symptoms.
- **Meningitis:** blood cultures, polymerase chain reaction, and lumbar puncture — with antibiotics given first.
- **Cerebral venous sinus thrombosis:** computed tomographic or magnetic resonance venography — plain imaging is frequently normal.
- **Dissection:** angiography with fat-suppressed sequences. **Glaucoma:** intraocular pressure and immediate ophthalmology referral. **Carbon monoxide:** carboxyhaemoglobin on a blood gas.
- **Magnetic resonance imaging** for a suspected mass lesion or where the picture is progressive.

6. Management While You Are Still Deciding

- Analgesia and antiemetics while investigation proceeds — treating the pain does not obscure the diagnosis.
- **Avoid opioids in primary headache disorders**, which promote medication overuse headache and rarely work well.
- Treat the specific cause: neurosurgical referral and nimodipine for subarachnoid haemorrhage; antibiotics for meningitis; corticosteroids for arteritis; anticoagulation for venous thrombosis.
- **For primary headache:** acute treatment with a triptan or an anti-inflammatory drug, consideration of prophylaxis where attacks are frequent, a **headache diary**, attention to sleep, hydration and caffeine, and explicit review of analgesic frequency.

7. Teaching Points and Viva Questions

- Ask how long it took to peak, not how severe it is.
- Computed tomography sensitivity falls after six hours; xanthochromia needs twelve.
- Start steroids in suspected giant cell arteritis before the biopsy, not after.

- Do fundoscopy. Papilloedema changes everything that follows.
- Take the temperature and the blood pressure in every headache.
- Ask how many days a month she takes painkillers, and whether anyone else at home has a headache.

Questions you should be able to answer:

- Her computed tomogram at 7 hours is normal. What do you do, and when?
- What is xanthochromia and why must you wait 12 hours?
- A 72-year-old has a new headache and pain in her jaw when chewing. What do you do, in what order?
- Give five red flags from the SNOOP list and the diagnosis each suggests.
- A patient has daily headache and takes codeine most days. What is the likely diagnosis and the treatment?

Seminar 6 · Approach to the Patient with Arthritis

THE PRESENTATION

A **58-year-old man** presents with two days of a hot, swollen, exquisitely painful right knee. He cannot bear weight and will not let you move the joint at all. He has had three previous attacks of sudden severe pain and swelling in his left great toe over the past two years, each settling within a week.

He has type 2 diabetes and chronic kidney disease, and takes a thiazide diuretic.

On examination: temperature 37.9 °C. The right knee is hot, red, and holds a tense effusion. Movement is restricted **in every direction**. No other joint is involved. There is no rash and no psoriasis.

C-reactive protein 180 · white cells 14.2 × 10^9/L · serum urate 340 µmol/L (within the normal range).

BEFORE ANYTHING ELSE

A single hot, swollen joint is septic arthritis until proved otherwise, and the joint must be aspirated. An infected joint can be destroyed within days, and the cartilage does not come back.

Two errors this case is built to prevent:

1. A history of gout does not protect against sepsis. **Crystals and infection coexist**, and finding crystals in the fluid does not exclude a co-existing septic arthritis.

2. A normal serum urate does not exclude gout. Urate levels commonly fall during an acute attack, so a normal value taken at presentation is uninformative — measure it again several weeks later.

1. Think First — By Number of Joints and Tempo

Pattern	Causes
Acute monoarthritis	Septic arthritis · crystal arthritis — gout and calcium pyrophosphate disease · haemarthrosis and trauma · reactive arthritis · the first joint of what will become a polyarthritis · avascular necrosis
Oligoarthritis (2–4 joints)	The spondyloarthritides — psoriatic, reactive, inflammatory bowel-associated, axial · crystal disease · sarcoidosis · Behçet disease · brucellosis · disseminated gonococcal infection
Acute polyarthritis	Viral — parvovirus B19, hepatitis B and C, chikungunya, dengue, rubella · reactive · early rheumatoid arthritis · systemic lupus erythematosus · drug-induced
Chronic polyarthritis	Rheumatoid arthritis · psoriatic arthritis · connective tissue disease · osteoarthritis (non-inflammatory) · chronic tophaceous gout

Then refine by **symmetry, joint size, axial involvement, and whether the distal interphalangeal joints are affected** — as set out in the rheumatology system opener.

2. What Could Destroy or Kill

- **Septic arthritis** — including of a prosthetic joint, which has a different microbiology and needs orthopaedic involvement immediately.
- **Disseminated gonococcal infection** — migratory arthralgia, tenosynovitis and pustular skin lesions in a sexually active patient.
- **Necrotising fasciitis** — pain out of proportion, rapid spread, systemic toxicity. Not a joint problem, but it presents as one.
- **Giant cell arteritis** in an older patient with a polymyalgic presentation — because untreated it blinds.

3. The History That Discriminates

- **Speed of onset and number of joints**, and whether previous episodes followed the same pattern.
- **Risk factors for sepsis:** diabetes, immunosuppression, chronic kidney disease, a **prosthetic joint**, intravenous drug use, a skin breach or ulcer, and **recent joint injection or aspiration**.
- **Risk factors for gout:** diuretics, alcohol, chronic kidney disease, obesity, previous podagra, family history, and a diet high in purines.
- **Preceding infection** — gastrointestinal or genitourinary, one to four weeks earlier, for reactive arthritis. **Sexual history** for gonococcal disease.
- **Unpasteurised milk and animal contact — brucellosis causes sacroiliitis and large joint arthritis and is a realistic cause here.** Recent travel for chikungunya and dengue.
- Psoriasis, inflammatory bowel disease, red eye, urethral discharge, oral and genital ulcers, rash, and every drug.

4. The Examination That Discriminates

- **Fever — but its absence does not exclude sepsis**, particularly in the elderly and the immunosuppressed.
- **Restriction of movement in every direction with severe pain** indicates the joint itself. **Bursitis and cellulitis over a joint preserve most passive movement** — this distinction is made at the bedside in thirty seconds and prevents an unnecessary aspiration or, worse, a missed one.
- Examine **all** the other joints, the entheses, and the spine; look for psoriasis in the scalp, natal cleft, umbilicus and nails; look for tophi, pustules, erythema nodosum and conjunctivitis.

5. Investigations, in Tiers

THE PIVOTAL INVESTIGATION

Aspirate the joint, and do it before antibiotics are given. One sample answers the question.

Send: cell count with differential · Gram stain · culture · **polarised light microscopy for crystals**.

Interpretation: over 50,000 white cells per mm^3 with a neutrophil predominance strongly suggests sepsis — but **lower counts do not exclude it**, especially early or in the immunosuppressed. 2,000–50,000 indicates inflammation; under 2,000 is non-inflammatory; frank blood suggests trauma, a bleeding disorder, or pseudogout.

Negatively birefringent needle-shaped crystals are urate; **positively birefringent rhomboid crystals** are calcium pyrophosphate.

- **Blood cultures before antibiotics**; complete blood count, C-reactive protein and erythrocyte sedimentation rate, renal and liver function, glucose, and serum urate — interpreted with the caution above.
- **Radiograph** as a baseline, and to look for chondrocalcinosis, erosions or avascular necrosis. **Ultrasound** to confirm an effusion and guide aspiration; **magnetic resonance imaging** where osteomyelitis or spondylodiscitis is suspected.

- **Targeted:** rheumatoid factor and anti-cyclic citrullinated peptide antibody, antinuclear antibody, **brucella serology**, parvovirus and hepatitis serology, and nucleic acid amplification testing for gonorrhoea and chlamydia from the appropriate sites.

6. Management While You Are Still Deciding

- **Aspirate, culture, then start empirical antibiotics** covering staphylococci according to local resistance patterns, adjusted once cultures return. **Do not wait for the culture result to start treatment, and do not start treatment before taking the sample.**
- **Refer to orthopaedics for washout or arthroscopic lavage**, with repeated aspiration where surgery is delayed. Splint briefly for comfort, then mobilise early to preserve function.
- **If it proves to be crystal disease:** an anti-inflammatory drug, colchicine, or corticosteroid — oral or intra-articular — chosen according to renal function and comorbidity. In this patient, with chronic kidney disease, anti-inflammatory drugs are a poor choice.
- **Do not start urate-lowering therapy during an acute attack — but do not stop it if the patient is already taking it.** Begin allopurinol or febuxostat several weeks later, with colchicine or anti-inflammatory cover for the first months, and treat to a target urate level.
- Review the thiazide, which is contributing, and address alcohol, weight and renal function.

7. Teaching Points and Viva Questions

- Aspirate every hot joint. It is the investigation, and it takes minutes.
- Crystals in the fluid do not exclude infection in the same joint.
- A normal urate during an attack tells you nothing.
- Bursitis and cellulitis preserve passive movement; septic arthritis does not.
- Ask about milk, animals, sexual history and recent joint injections.
- Never start urate-lowering therapy in an acute attack, and never stop it either.

Questions you should be able to answer:

- The aspirate shows 30,000 white cells and urate crystals. Have you excluded sepsis?
- Why is his urate normal, and when should you measure it?
- He has chronic kidney disease. How does that change your choice of anti-inflammatory treatment?
- Distinguish septic arthritis from prepatellar bursitis at the bedside.
- A 26-year-old has migratory arthralgia, tenosynovitis and pustules. What is the diagnosis and what do you send?

Seminar 7 · Approach to the Patient with Chronic Cough

THE PRESENTATION

A **52-year-old woman** has coughed for four months. The cough is dry, worse at night and when she lies down, and it interrupts conversation and has twice caused stress incontinence. She is not breathless. She has never smoked.

She was started on **ramipril five months ago** for hypertension. She also describes a sensation of mucus dripping at the back of her throat and frequent throat clearing, and occasional heartburn.

On examination: entirely normal. **Chest radiograph normal. Spirometry normal with no reversibility.**

1. Think First

Chronic cough means more than eight weeks. Under three weeks is acute, and three to eight weeks subacute — most often post-infectious and self-limiting.

Category	Causes
The three commonest in a non-smoker with a normal chest radiograph	Upper airway cough syndrome (post-nasal drip, rhinosinusitis) · asthma, including cough-variant asthma and non-asthmatic eosinophilic bronchitis · gastro-oesophageal reflux
Drugs	Angiotensin-converting enzyme inhibitors — in up to one in seven patients, beginning anywhere from days to months after starting
Must not be missed	Lung cancer · tuberculosis · bronchiectasis · interstitial lung disease · heart failure · inhaled foreign body · chronic infection
Others	Smoking and chronic obstructive pulmonary disease · obstructive sleep apnoea · chronic aspiration · cough hypersensitivity syndrome · habit cough
	RED FLAGS — INVESTIGATE RATHER THAN TREAT EMPIRICALLY **Haemoptysis · weight loss · fever or night sweats · hoarseness · dysphagia · breathlessness · chest pain · recurrent pneumonia · an abnormal examination or chest radiograph · a smoker over 40 · immunosuppression.** **And the standing regional rule: any cough lasting more than two to three weeks, particularly with weight loss or night sweats, means send three sputum samples for tuberculosis** before you begin an empirical trial for reflux. The empirical pathway below applies only once tuberculosis and malignancy have been considered and excluded.

2. The History That Discriminates

- **Check the drug chart before you take the history.** An angiotensin-converting enzyme inhibitor explains a large number of chronic coughs, and **the temporal link is often missed because the cough may begin months after the drug was started.**
- **Dry or productive, and how much.** Large daily volumes of purulent sputum means bronchiectasis until proved otherwise.
- **Timing and triggers:** worse at night suggests asthma, reflux or heart failure; worse on lying flat suggests reflux; provoked by cold air, exercise or laughing suggests airway hyper-responsiveness; provoked by eating or talking suggests reflux or aspiration.
- **Nasal symptoms and throat clearing** for upper airway cough syndrome; heartburn and regurgitation for reflux — **although reflux-related cough frequently occurs with no heartburn at all**.

- **Exposures:** smoking including waterpipe, occupational dusts, **biomass fuel and incense smoke**, birds and moulds, and tuberculosis contact.

3. The Examination That Discriminates

- **Expect it to be normal, and record that it is.** A normal examination in chronic cough is the usual finding, not a failure to examine properly.
- Examine the nose and post-nasal space, the throat, and the **ears** — impacted wax against the drum stimulates the auricular branch of the vagus and causes cough.
- Cervical lymph nodes, clubbing, the chest, the heart, and — importantly — **watch the patient use her inhaler** if she has one.

4. Investigations, in Tiers

- **First line for everyone: chest radiograph**, spirometry with reversibility testing, and a complete blood count looking for eosinophilia.
- **Where tuberculosis is possible — which is often, here: three sputum specimens for smear, nucleic acid amplification testing and culture.**
- **Targeted:** fractional exhaled nitric oxide or bronchial provocation testing for cough-variant asthma; **computed tomography of the chest** for suspected bronchiectasis, malignancy or interstitial disease; ear, nose and throat assessment with nasendoscopy; oesophageal pH and impedance monitoring where reflux is suspected and empirical treatment has failed; bronchoscopy for a suspected foreign body or endobronchial lesion; echocardiography.

5. Management While You Are Still Deciding

> THE FIRST THERAPEUTIC STEP IN THIS PATIENT
>
> **Stop the ramipril and substitute an angiotensin receptor blocker** — and tell the patient that **resolution takes up to four weeks and occasionally longer**.
>
> The commonest error is to stop the drug, review at one week, find the cough unchanged, and conclude that the drug was not responsible. **Do not judge it before four weeks have passed.**

- **Then treat the likely causes one at a time, each for long enough to be a fair trial.** Treating three things simultaneously means that when the cough settles you will not know why, and when it does not you will not know what to change.
- **Upper airway cough syndrome:** intranasal corticosteroid, with an antihistamine where there is an allergic component. **Asthma or eosinophilic bronchitis:** a trial of inhaled corticosteroid. **Reflux:** a proton pump inhibitor with dietary and postural measures, recognising that the evidence for acid suppression in cough is modest.
- **Smoking cessation** where relevant, which alone resolves a substantial proportion of coughs within weeks.
- **Refractory chronic cough:** speech and language therapy with cough suppression techniques, and neuromodulator therapy such as gabapentin or low-dose morphine, under specialist supervision.

- **Acknowledge the impact.** Chronic cough causes urinary incontinence, sleep loss, social embarrassment, exhaustion and occasionally rib fractures. Patients are frequently told it is nothing.

6. Teaching Points and Viva Questions

- Eight weeks defines chronic cough. Read the drug chart before the history.
- Angiotensin-converting enzyme inhibitor cough can start months after the drug and takes weeks to settle after stopping.
- A normal examination and a normal film are expected — but red flags and regional tuberculosis risk override the empirical pathway.
- Reflux cough often occurs without heartburn.
- Treat one cause at a time, for long enough to know whether it worked.

Questions you should be able to answer:

- Name the three commonest causes in a non-smoker with a normal film.
- She stopped the ramipril a week ago and is no better. What do you tell her?
- Which findings in this history would make you send sputum before starting any trial?
- Why should you not start a nasal steroid, an inhaler and a proton pump inhibitor together?
- A 63-year-old smoker has coughed for three months and has lost 5 kg. What is your first investigation?

Seminar 8 · Hypertensive Urgencies and Emergencies

THE PRESENTATION

A **54-year-old man** attends after a pharmacy reading of 226/128 mmHg. For two days he has had a persistent headache and blurred vision. He has no chest pain and no breathlessness. He was prescribed antihypertensives two years ago and **stopped taking them several months ago** because he felt well.

On examination: blood pressure 224/126 mmHg in both arms, correctly measured and repeated. **Fundoscopy shows flame haemorrhages, cotton wool spots and bilateral papilloedema.** No focal neurological deficit. Chest clear, no murmur, no radio-femoral delay.

Creatinine 168 µmol/L · urine dipstick: blood and protein · haemoglobin 112 g/L · platelets 96 × 10^9/L · blood film: red cell fragments · lactate dehydrogenase raised.

THE DISTINCTION THAT GOVERNS EVERYTHING THAT FOLLOWS

The blood pressure number does not tell you which of these you are dealing with. The end-organ assessment does.

Hypertensive emergency — severe hypertension **with acute end-organ damage**. Requires immediate, **controlled** parenteral treatment in a monitored bed.

Hypertensive urgency — severe hypertension **without** acute end-organ damage. Treated with oral therapy and gradual reduction over days, usually as an outpatient. **Rapid lowering in this group causes harm and no benefit.**

This patient has retinopathy with papilloedema, acute kidney injury and a thrombotic microangiopathy on the film. **That is an emergency.**

1. Think First — Where Is the End-Organ Damage?

Organ	What to look for
Brain	Hypertensive encephalopathy — headache, confusion, seizures, visual loss; ischaemic stroke; intracerebral haemorrhage; posterior reversible encephalopathy syndrome
Eye	Flame haemorrhages, cotton wool spots and papilloedema — which defines accelerated or malignant hypertension
Heart and aorta	Acute coronary syndrome, acute pulmonary oedema, aortic dissection
Kidney	Acute kidney injury, haematuria and proteinuria, and thrombotic microangiopathy with fragments and thrombocytopenia
Pregnancy	Pre-eclampsia and eclampsia — a separate pathway entirely, and to be considered in any pregnant or recently delivered woman

2. How Fast, and With What — the Targets Differ by Situation

WHY YOU DO NOT SIMPLY NORMALISE THE BLOOD PRESSURE

In chronic hypertension the cerebral autoregulatory curve **shifts to the right**. The brain, kidney and retina are perfused adequately at pressures that would be dangerous in a normotensive person — and they lose perfusion at pressures that appear entirely acceptable on the chart.

Dropping the pressure too quickly therefore causes watershed cerebral infarction, blindness, acute kidney injury and myocardial ischaemia. More harm has been done by treating the number aggressively than by treating it gradually.

Situation	Target and agent
General hypertensive emergency	Reduce mean arterial pressure by no more than 20–25% in the first hour, then to around 160/100 over the next 2–6 hours, then normalise over 24–48 hours. Intravenous labetalol or nicardipine, titrated.
Aortic dissection	The exception — lower fast and hard: systolic below 120 mmHg and heart rate below 60 within about 20 minutes. Give the beta blocker first, then the vasodilator, so that reflex tachycardia does not increase aortic wall shear stress.
Acute ischaemic stroke	Do not lower unless above about 220/120, or thrombolysis is planned — in which case below 185/110. See Case 64.
Intracerebral haemorrhage	Early lowering towards a systolic of about 140 mmHg.
Acute pulmonary oedema	Intravenous nitrate with a loop diuretic; non-invasive ventilation as needed.
Phaeochromocytoma, cocaine or amphetamine	Alpha blockade first. Never a beta blocker alone — unopposed alpha stimulation raises the pressure further.
Pre-eclampsia and eclampsia	Labetalol, nifedipine or hydralazine, plus magnesium sulfate, with obstetric involvement and delivery as the definitive treatment.

Sublingual short-acting nifedipine has been abandoned because it produces an uncontrolled and unpredictable fall in pressure. It should not be used.

3. The History That Discriminates

- **Ask what medication was stopped, and when.** Non-adherence, or an abrupt withdrawal — particularly of clonidine or a beta blocker — is among the commonest precipitants, and it is entirely correctable.
- Symptoms of each organ system: headache, visual change, confusion, seizure, chest pain, breathlessness, oliguria, frothy or bloody urine.
- **Drugs:** anti-inflammatory drugs, decongestants, corticosteroids, the combined oral contraceptive, ciclosporin, erythropoietin, liquorice — and **cocaine and amphetamines**, which change the treatment.
- **Features of a secondary cause**, as in Case 14 — because severe hypertension in a young patient, or hypertension that suddenly becomes uncontrolled, is far more likely to be secondary.
- **Pregnancy or recent delivery** in any woman of childbearing age.

4. The Examination That Discriminates

- **Blood pressure measured properly, in both arms, and repeated** — with the correct cuff size, after rest. Severe readings are frequently artefactual.
- **Fundoscopy in every patient. This is the examination that most often converts an urgency into an emergency**, and it is the one most often skipped.
- Full neurological examination; heart and lungs; **radio-femoral delay and peripheral pulses**; abdominal and renal bruits; volume status.

5. Investigations, in Tiers

- **Bedside: urinalysis** — blood and protein indicate renal involvement and are available in a minute; electrocardiogram.
- **First line:** urea, electrolytes and creatinine, **complete blood count with a blood film for fragments**, lactate dehydrogenase, troponin, natriuretic peptide, glucose, chest radiograph, **pregnancy test**.
- **Targeted:** computed tomography of the head where there are neurological features; **computed tomographic aortogram where dissection is suspected**; echocardiography; renal imaging.
- **Once stabilised, screen for a secondary cause** — aldosterone-to-renin ratio, plasma metanephrines, renal artery imaging, and a sleep study.

6. Management While You Are Still Deciding

- **Monitored bed with continuous blood pressure monitoring**, ideally intra-arterial, and an intravenous agent titrated to a defined target that is written down.
- **Identify and remove the precipitant**, and restart oral therapy early so that the infusion can be weaned.
- Expect the **creatinine to rise transiently** as the pressure falls — this is haemodynamic and is not a reason to stop treating, provided the fall is controlled.
- **In a hypertensive urgency:** do not treat in the emergency department with repeated intravenous boluses. Restart or adjust oral therapy, arrange review within days, and address why the medication stopped.

7. Teaching Points and Viva Questions

- The organs define the emergency, not the number.
- Do fundoscopy. Papilloedema changes the diagnosis and the disposition.
- Twenty to twenty-five per cent in the first hour — except in dissection, and except in acute ischaemic stroke where you do not lower it at all.
- Beta blocker before vasodilator in dissection; alpha before beta in cocaine and phaeochromocytoma.
- Always ask which tablets were stopped and why.

Questions you should be able to answer:

- Is this an urgency or an emergency, and what exactly makes it so?
- What is your target pressure at one hour, and why not lower?
- Why do the fragments on his blood film matter?
- A patient with the same pressure has tearing interscapular pain. How does your plan change, and in what order do you give the drugs?

- A 22-year-old with a pressure of 230/130 has taken cocaine. What must you avoid?

Seminar 9 · Approach to the Patient with Pyrexia of Unknown Origin

THE PRESENTATION

A **47-year-old man** has had intermittent fever for six weeks, reaching 38.8 °C most evenings, with **drenching night sweats** and 9 kg of weight loss. He has had two courses of antibiotics from different doctors, each followed by a few days of apparent improvement.

He keeps sheep and goats and drinks fresh unboiled milk.

On examination: thin and tired. Temperature 38.4 °C with a pulse of 82/min. There is **mild hepatosplenomegaly** and a soft systolic murmur that has not previously been documented.

Erythrocyte sedimentation rate 88 · normocytic anaemia · white cell count normal with relative lymphocytosis · mildly raised transaminases · chest radiograph, urine culture and first blood cultures all negative.

1. Think First — Four Categories

Pyrexia of unknown origin is conventionally fever above 38.3 °C on several occasions for more than three weeks, remaining undiagnosed after appropriate initial investigation. The causes fall into four groups, and the proportions differ substantially by region.

Category	The causes that matter here
Infection	Tuberculosis — especially extrapulmonary: miliary, abdominal, spinal, nodal · brucellosis · infective endocarditis, including culture-negative from *Coxiella*, *Brucella* and *Bartonella* · occult abscess — dental, hepatic, subphrenic, psoas, pelvic · osteomyelitis and spondylodiscitis · human immunodeficiency virus · typhoid · visceral leishmaniasis · malaria
Malignancy	Lymphoma above all · leukaemia · renal cell and hepatocellular carcinoma · atrial myxoma · metastatic disease
Non-infectious inflammatory	Adult-onset Still disease · giant cell arteritis and polymyalgia rheumatica — a major and treatable cause over the age of 50 · systemic lupus erythematosus and vasculitis · sarcoidosis · inflammatory bowel disease · Behçet disease · familial Mediterranean fever and other periodic fever syndromes, worth considering where there is consanguinity and a lifelong history
Miscellaneous	Drug fever · venous thromboembolism · subacute thyroiditis · adrenal insufficiency · haematoma · factitious fever · and a substantial group that resolve without ever being diagnosed

2. The Method — Which Matters More Than the List

HOW TO ACTUALLY APPROACH IT

1. Confirm the fever is real. Documented, measured by staff, with a chart. Suspect factitious fever if there is no tachycardia, no diurnal variation, and normal inflammatory markers.

2. Stop every non-essential drug. Drug fever occurs with almost any agent, typically one to three weeks after starting, in a patient who looks surprisingly well, and it settles within 72 hours of withdrawal. It costs nothing to test.

3. Repeat the history and the full examination every single day. In pyrexia of unknown origin the diagnosis is found more often by re-examining than by ordering another test. A new murmur, a new node, a rash, a tender vertebra, a joint — these appear over days.

4. Take the exposure history again, from the beginning. Travel, animals, **unpasteurised milk**, contacts, occupation, sexual history, dental work, prostheses, previous surgery.

5. Stop the antibiotics and re-culture. Prior antibiotic exposure is the commonest reason blood cultures are sterile in endocarditis — and this patient has had two courses.

3. What Could Kill

- Infective endocarditis; disseminated tuberculosis; an occult abscess progressing to sepsis; aggressive lymphoma; **giant cell arteritis with impending visual loss**; and adrenal crisis.

4. Investigations, in Tiers

- **First tier:** complete blood count **with a blood film**, erythrocyte sedimentation rate and C-reactive protein, renal and liver function, lactate dehydrogenase, **three sets of blood cultures taken off antibiotics**, urinalysis and culture, chest radiograph, human immunodeficiency virus and hepatitis serology, antinuclear antibody and rheumatoid factor, protein electrophoresis, thyroid function, creatine kinase, and **ferritin**.

ONE TEST WORTH ADDING EARLY

A markedly raised ferritin — many times the upper limit — in a febrile patient points to adult-onset Still disease or to haemophagocytic lymphohistiocytosis. The latter is rapidly fatal if missed and is diagnosed by a specific combination of fever, cytopenias, splenomegaly, hypertriglyceridaemia, hypofibrinogenaemia and very high ferritin.

It is a cheap test that is frequently omitted from the standard panel.

- **Regionally targeted: brucella serology with prolonged blood cultures** — and tell the laboratory; **tuberculin or interferon-gamma release assay with sputum and tissue for tuberculosis**; *Coxiella* and *Bartonella* serology; malaria films; leishmania serology.
- **Imaging: echocardiography, proceeding to transoesophageal study** where endocarditis is possible; computed tomography of chest, abdomen and pelvis; **magnetic resonance imaging of the spine** for spondylodiscitis; dental panoramic radiograph; and **fluorodeoxyglucose positron emission tomography where available**, which has a high diagnostic yield in this setting.
- **Tissue, when imaging is unrevealing:** lymph node biopsy; **bone marrow aspirate and trephine sent for culture as well as histology**, which has a good yield for tuberculosis, lymphoma and leishmaniasis; liver biopsy; and **temporal artery biopsy or ultrasound in anyone over 50 with a raised erythrocyte sedimentation rate**.

5. Management While You Are Still Deciding

THE HARDEST DISCIPLINE IN THIS PRESENTATION

Avoid empirical antibiotics and empirical corticosteroids. Both blunt the fever, obscure the picture, sterilise the cultures, and can make a definitive diagnosis impossible — sometimes permanently.

The exceptions, where you treat before you know: suspected endocarditis in a haemodynamically compromised patient · suspected giant cell arteritis with visual symptoms · suspected tuberculous meningitis · neutropenic sepsis · any patient who is deteriorating.

Otherwise: supportive care, daily review, daily examination, and patience. A proportion of patients defervesce spontaneously without a diagnosis ever being made, and that is an acceptable outcome once the dangerous causes have been excluded.

6. Teaching Points and Viva Questions

- Re-examine the patient every day. That is where the diagnosis usually comes from.
- Stop the drugs. Drug fever is common and free to exclude.

- Three sets of blood cultures, off antibiotics.
- In this region, tuberculosis and brucellosis head the list — and both need the laboratory to be told.
- Check the ferritin.
- Empirical treatment feels active and usually destroys the diagnosis.

Questions you should be able to answer:

- Define pyrexia of unknown origin and give the four diagnostic categories.
- Which two diagnoses does this man's occupation and diet put at the top of your list?
- He has had two courses of antibiotics. How does that affect your investigation?
- A colleague wants to start empirical antituberculous therapy. Argue both sides.
- Which single investigation would you request if a positron emission tomogram were unavailable and the computed tomogram was normal?

Seminar 10 · Approach to the Patient in Coma

THE PRESENTATION

A **62-year-old woman** is brought in by ambulance having been found unresponsive at home by her son, who last spoke to her the previous evening. She has type 2 diabetes treated with gliclazide, and takes an antidepressant.

On arrival: Glasgow Coma Scale 7 — **eyes 1, verbal 2, motor 4**. Pupils 2 mm and reactive **bilaterally**. No lateralising signs; tone symmetrical; plantars flexor. Temperature 35.8 °C, blood pressure 148/86 mmHg, heart rate 62/min, respiratory rate 10/min and shallow.

Capillary glucose 3.1 mmol/L.

BEFORE THE DIFFERENTIAL

Airway first. A Glasgow Coma Scale of 8 or less does not protect the airway. Position, suction, adjunct, and call for anaesthetic help.

Then, in this order, and within the first two minutes:

Glucose — check it and correct it. **In anyone malnourished or alcohol-dependent, give thiamine before or with the glucose**, to avoid precipitating Wernicke encephalopathy.

Oxygen and ventilation — hypoxia and hypercapnia are both causes and consequences.

Naloxone if opioid toxicity is conceivable — look at the pupils and the respiratory rate.

Everything else can wait a few minutes. These cannot.

1. Think First — Two Mechanisms

Consciousness requires **both cerebral hemispheres** and the **reticular activating system in the brainstem**. Coma therefore means either diffuse bilateral hemispheric dysfunction or a brainstem lesion — and the examination tells you which.

	Diffuse / metabolic-toxic	Structural
Findings	Symmetrical, with preserved pupillary reflexes and preserved brainstem reflexes; may have tremor, myoclonus, asterixis	Asymmetry, focal signs, cranial nerve and brainstem abnormalities, abnormal pupils, gaze deviation
Causes	Hypoglycaemia and hyperglycaemia · hyponatraemia, hypercalcaemia · uraemia, hepatic encephalopathy · hypoxia, hypercapnia · hypothermia and hyperthermia · sepsis · myxoedema, adrenal crisis · thiamine deficiency · drugs and toxins — opioids, benzodiazepines, alcohol, antidepressants, salicylate, carbon monoxide · post-ictal state and non-convulsive status epilepticus	Intracerebral and subarachnoid haemorrhage · large infarct with oedema · subdural and extradural haematoma · tumour · abscess · hydrocephalus · basilar artery occlusion · herniation

Infection sits across both: meningitis, encephalitis and cerebral malaria can produce either picture, and they are treated before imaging.

2. The History — From Everybody Except the Patient

- **The circumstances in which she was found**, and **when she was last known to be well** — which determines the treatment window if this proves to be a stroke.
- **Access to drugs: what is in the house, and what is missing.** Ask the ambulance crew about empty packets, bottles and syringes, and about the state of the room.

- Preceding headache, fever, confusion, focal symptoms, seizures, vomiting or trauma — including a fall days earlier, which raises subdural haematoma.
- Comorbidity: diabetes, liver disease, kidney disease, epilepsy, psychiatric illness, malignancy, and alcohol use.
- **Whether anyone else in the household is unwell or has a headache** — carbon monoxide poisoning affects the whole house, and is repeatedly missed.

3. The Coma Examination

- **Record the Glasgow Coma Scale as its three components**, not as a total. "GCS 7" hides whether the patient is deteriorating in eye opening or in motor response, and the motor score is the most prognostically important.
- **Respiratory pattern and temperature**, both of which are diagnostic clues and both of which are frequently omitted.

Pupils	Suggests
Pinpoint and reactive	Opioids, or a pontine lesion
Mid-position and fixed	Midbrain lesion
Unilateral fixed and dilated	Third nerve compression — uncal herniation. A neurosurgical emergency
Bilateral fixed and dilated	Severe hypoxic-ischaemic injury, profound drug toxicity, or hypothermia
Equal and reactive	Favours a metabolic or toxic cause — pupillary reflexes are remarkably resistant to metabolic insult

- **Eye movements:** spontaneous movement, gaze deviation (**towards the lesion in a hemispheric stroke, away from it in a seizure focus**), corneal reflexes, and oculocephalic testing — **only once the cervical spine has been cleared**.
- **Fundoscopy** for papilloedema and for the subhyaloid haemorrhage of subarachnoid bleeding.
- Motor response to a standardised stimulus, tone, reflexes, plantars, **asymmetry**, and abnormal posturing.
- Neck stiffness — with caution if trauma is possible. Skin for rash, needle marks, jaundice, cyanosis, bruising, and **signs of head injury including periorbital and mastoid bruising and cerebrospinal fluid leak**. Smell the breath. Look for a medical alert bracelet.

4. Investigations, in Tiers

- **Bedside:** glucose, arterial blood gas **including carboxyhaemoglobin and lactate**, electrocardiogram, temperature.
- **First line:** urea and electrolytes, calcium, magnesium, liver function, ammonia, complete blood count, C-reactive protein, coagulation, creatine kinase, thyroid function, **cortisol**, blood and urine cultures, **paracetamol and salicylate levels**, toxicology screen, and **serum osmolality with an osmolar gap** where a toxic alcohol is possible.

- **Computed tomography of the head** in all but the most obvious metabolic cases; **lumbar puncture after imaging** where infection is suspected — with antibiotics given first.

THE CAUSE THAT IS INVISIBLE WITHOUT ASKING FOR IT

Consider non-convulsive status epilepticus in any patient whose coma is unexplained, particularly after a witnessed seizure, in a known epileptic, or where there are subtle twitching movements of the face or eyes.

It is invisible without an **electroencephalogram**, it is treatable, and it is one of the commonest missed causes of persistent unexplained coma. If the imaging and the metabolic screen are normal, ask for an electroencephalogram rather than waiting.

5. Management While You Are Still Deciding

- **Secure the airway** — intubate for a score of 8 or less, an absent gag, or a rising carbon dioxide. Maintain oxygenation and normocapnia.
- **Treat the reversible causes empirically in sequence:** glucose, thiamine, naloxone; then antibiotics and aciclovir if infection is possible, before imaging.
- Nurse head-up at 30 degrees; maintain normothermia, normoglycaemia and normal sodium; treat seizures; **treat raised intracranial pressure** and involve neurosurgery urgently for a mass lesion or herniation.
- Eye care, pressure area care, nutrition, thromboprophylaxis, and a urinary catheter.
- **Talk to the family early and honestly**, and establish what the patient would have wanted before decisions become urgent.

6. Teaching Points and Viva Questions

- Airway, glucose, thiamine, naloxone — in that order, in the first two minutes.
- Record the coma score in its three components.
- Symmetrical signs with reactive pupils point to a metabolic cause; asymmetry points to a structural one.
- A unilateral fixed dilated pupil is a neurosurgical emergency.
- Ask whether anyone else at home is unwell.
- If nothing explains it, ask for an electroencephalogram.

Questions you should be able to answer:

- What is the likely cause here, and what would you give first — and in what order?
- Why does she need thiamine considered before glucose?
- Her pupils are equal and reactive. What does that make more likely?
- The glucose is corrected but she does not wake. What are your next three steps?

- A patient has a right pupil of 6 mm that does not react. What has happened and whom do you call?

Seminar 11 · Approach to the Patient with Venous Thromboembolism

THE PRESENTATION

A **34-year-old woman** presents with three days of pain and swelling in the left calf. Since this morning she has had right-sided pleuritic chest pain and has felt breathless climbing stairs.

She takes the combined oral contraceptive pill. She returned four days ago from a 14-hour car journey. Her mother had a deep vein thrombosis at the age of 40.

On examination: heart rate 104/min, blood pressure 118/74 mmHg, respiratory rate 22/min, **oxygen saturation 94% on air**. The left calf is **4 cm greater in circumference** than the right, with tenderness along the deep venous system and pitting oedema. Chest is clear. Heart sounds normal.

1. Think First

Deep vein thrombosis and pulmonary embolism are one disease presenting in two places. This patient has both, and finding one is sufficient reason to treat.

- **Differential for the leg:** cellulitis, ruptured Baker cyst, calf muscle tear or haematoma, superficial thrombophlebitis, chronic venous insufficiency, lymphoedema, compartment syndrome.
- **Differential for the chest:** as in Seminar 3.

Generate the risk factors from Virchow's triad rather than memorising a list:

Element	Risk factors
Stasis	Immobility, long-distance travel, surgery, plaster cast, paralysis, heart failure, obesity, pregnancy
Endothelial injury	Surgery, trauma, central venous catheter, previous venous thromboembolism
Hypercoagulability	Malignancy · pregnancy and the puerperium · oestrogen — combined oral contraceptive and hormone replacement · inherited thrombophilia · antiphospholipid syndrome · nephrotic syndrome · inflammatory disease · acute infection · dehydration

2. What Could Kill in the Next Hour

HIGH-RISK PULMONARY EMBOLISM

A patient with pulmonary embolism who is hypotensive, shocked or arresting does not go down a diagnostic pathway.

Persistent hypotension or shock with right ventricular strain defines **high-risk pulmonary embolism**, and it requires **immediate reperfusion — systemic thrombolysis**, or catheter-directed therapy or surgical embolectomy where thrombolysis is contraindicated.

Bedside echocardiography showing a dilated, poorly functioning right ventricle is enough to act on in a peri-arrest patient. Waiting for a computed tomogram in this situation costs lives.

3. Score Before You Test

THE CENTRAL PRINCIPLE OF THIS CASE

Calculate the pre-test probability first, using a validated score. The correct next test depends entirely on the answer.

D-dimer is useful only where the probability is low or intermediate, and its value lies **entirely in its negative predictive value** — it excludes, it never confirms. It is raised by age, pregnancy, infection, malignancy, surgery, trauma and inflammation, so a positive result on its own means almost nothing.

In a high-probability patient, a negative D-dimer does not exclude the diagnosis. Go straight to imaging. Requesting a D-dimer in this patient, whose probability is high, would be an error whichever way it came back.

Age-adjusted thresholds improve specificity in older patients without losing sensitivity.

4. Investigations

- **Suspected deep vein thrombosis: proximal leg vein compression ultrasound.** If negative but the probability is high, repeat it after about a week, or proceed to further imaging.
- **Suspected pulmonary embolism: computed tomographic pulmonary angiography.**

THE IMAGING CHOICE THAT IS REGULARLY GOT WRONG

Use **ventilation–perfusion scanning** in preference to computed tomography where the chest radiograph is normal and the patient is **pregnant or a young woman**, because it delivers substantially less radiation to breast tissue. It is also preferred in significant renal impairment and in contrast allergy.

In pregnancy, start with bilateral leg ultrasound. If a deep vein thrombosis is confirmed, the treatment is the same and chest imaging can be avoided altogether — sparing both mother and fetus.

- **Supporting tests, none of which diagnose:** electrocardiogram — sinus tachycardia is the commonest finding, and the classical S1Q3T3 pattern is uncommon; chest radiograph, whose main role is to exclude alternatives; blood gas; and **troponin and natriuretic peptide, which are used for risk stratification and not for diagnosis**.
- **Echocardiography** for right ventricular strain in the haemodynamically unstable patient.

5. Management

- **Start anticoagulation on clinical suspicion while awaiting imaging** where the probability is high and the bleeding risk is acceptable. Do not wait several hours for a scan with the patient untreated.

Situation	Anticoagulant
Most patients	A direct oral anticoagulant, which is now first line and needs no monitoring
Antiphospholipid syndrome, particularly triple-positive	Warfarin — direct oral anticoagulants are inferior and are avoided
Pregnancy	Low-molecular-weight heparin. Warfarin and direct oral anticoagulants are contraindicated
Severe renal impairment	Low-molecular-weight heparin or warfarin, with dose adjustment
Cancer-associated thrombosis	A direct oral anticoagulant or low-molecular-weight heparin, weighing bleeding risk — particularly with gastrointestinal and genitourinary tumours

- **Duration:** three months where the event was provoked by a major transient risk factor that has now resolved; at least three to six months if unprovoked, with a considered decision about extended therapy weighing recurrence risk against bleeding risk; indefinite therapy in active cancer, antiphospholipid syndrome, and recurrent unprovoked events.
- **Low-risk pulmonary embolism can be managed as an outpatient** using a validated severity score, which is safe and avoids admission.

TWO INVESTIGATIONS THAT ARE DONE TOO OFTEN

Do not perform a thrombophilia screen acutely, and do not perform one while the patient is anticoagulated — the results are unreliable and, in most cases, would not change management. The duration of anticoagulation is decided by whether the event was provoked, not by a genetic result.

Antiphospholipid antibodies are the exception, and are worth testing in a young patient with unprovoked thrombosis, recurrent pregnancy loss, or both arterial and venous events — because a positive result **does** change the drug.

And do not scan the whole body looking for cancer after an unprovoked event. A careful history and examination, basic bloods, and **age-appropriate screening only** — extensive occult malignancy screening finds little and improves nothing.

- **Stop the combined oral contraceptive** and counsel on alternative contraception — a progestogen-only method or a non-hormonal method — and on future pregnancy, which will require thromboprophylaxis.
- Graduated compression for post-thrombotic symptoms; explain the risk of recurrence and the symptoms that should bring her back; and **follow up persisting breathlessness at three to six months for chronic thromboembolic pulmonary hypertension**, which is treatable and easily missed.

6. Teaching Points and Viva Questions

- Score the probability before you choose the test.
- D-dimer excludes; it never confirms — and it is useless in a high-probability patient.
- Ventilation–perfusion scanning in pregnancy and in young women; leg ultrasound first in pregnancy.
- Anticoagulate on suspicion when the probability is high.
- The exceptions to direct oral anticoagulants are antiphospholipid syndrome, pregnancy and severe renal impairment.
- No thrombophilia screen, and no whole-body cancer hunt.

Questions you should be able to answer:

- Would you send a D-dimer on this patient? Justify your answer.
- Which imaging would you choose for her, and why not computed tomography?
- She is 8 weeks pregnant. How does everything change?
- When is a thrombophilia screen actually useful?
- Six months later she remains breathless on exertion. What must you exclude?

Seminar 12 · Approach to the Patient in Shock

THE PRESENTATION

A **58-year-old man** is brought in drowsy. His wife reports two days of vomiting and diarrhoea, and that he has passed no urine today. He takes ramipril and metformin.

On arrival: heart rate 128/min, blood pressure 74/44 mmHg, respiratory rate 28/min, oxygen saturation 96% on air, temperature 36.2 °C. **The peripheries are cold with a capillary refill of five seconds. The jugular venous pressure is not visible.** He is rousable but confused.

Lactate 5.8 mmol/L · glucose 6.4 · no rash · abdomen soft.

THE DEFINITION THAT MATTERS

Shock is inadequate tissue perfusion, not a blood pressure reading.

A patient with a "normal" blood pressure, cold peripheries, oliguria, confusion and a lactate of 5 is shocked. A young person compensates by vasoconstriction and tachycardia and maintains their pressure until they decompensate suddenly.

Judge perfusion by: mental state · capillary refill and peripheral temperature · urine output · and the lactate and its trend — which is the single most useful marker of both severity and response.

1. Think First — Four Mechanisms

Mechanism	Causes
Hypovolaemic	Haemorrhage — gastrointestinal, ruptured aortic aneurysm, ectopic pregnancy, trauma, retroperitoneal · fluid loss — vomiting and diarrhoea, burns, ketoacidosis, pancreatitis, third-space losses
Distributive	Sepsis — the commonest · anaphylaxis · neurogenic after spinal cord injury · adrenal crisis · liver failure · vasodilating drugs and toxins
Cardiogenic	Myocardial infarction · arrhythmia · myocarditis · acute valve failure or papillary muscle rupture · cardiomyopathy · beta blocker and calcium channel blocker overdose
Obstructive	Massive pulmonary embolism · tension pneumothorax · cardiac tamponade · dynamic hyperinflation in severe asthma

	Jugular venous pressure	Peripheries	Other clues
Hypovolaemic	Low	Cold	Obvious loss; collapsed inferior vena cava on ultrasound. This patient
Distributive / septic	Low or normal	Warm early, cold late	Fever, bounding pulse, wide pulse pressure, a source
Cardiogenic	Raised	Cold	Crackles, third heart sound, murmur, abnormal electrocardiogram
Obstructive	Raised	Cold	Tamponade — muffled sounds, pulsus paradoxus. Tension pneumothorax — unilateral hyper-resonance, tracheal shift. Embolism — hypoxia with a clear chest

The jugular venous pressure and the temperature of the hands divide the four mechanisms in about ten seconds, and they are available before any test.

2. What Could Kill in the Next Minutes

THE REVERSIBLE CAUSES

Act on these **before** the diagnosis is complete:

Tension pneumothorax — needle decompression, not imaging · **anaphylaxis** — intramuscular adrenaline · **haemorrhage** — stop the bleeding and give blood · **tamponade** — pericardiocentesis · **arrhythmia** — cardioversion or pacing · **adrenal crisis** — intravenous hydrocortisone · **sepsis** — antibiotics within the hour · **hypoglycaemia** — glucose.

Immediate actions in every shocked patient: airway and high-flow oxygen · two large-bore cannulae · continuous monitoring · bloods including **lactate and cross-match** · urinary catheter · **an early call to a senior and to critical care** — made while you are still resuscitating, not after.

3. The History and Examination That Discriminate

- **From whoever is available:** onset and course; bleeding of any kind; fever; chest pain; breathlessness; vomiting and diarrhoea; **drugs — antihypertensives, beta blockers, insulin, anticoagulants, and recently stopped corticosteroids**; allergies and exposures; recent surgery or procedure; immobility; and the possibility of pregnancy.

- **Examination:** full vital signs **including the respiratory rate**, capillary refill, peripheral temperature, conscious level; **jugular venous pressure**; heart sounds, murmurs and pulsus paradoxus; chest; abdomen for peritonism, a pulsatile mass or distension; **rectal examination**; calves; **the whole skin for a non-blanching rash, urticaria or cellulitis**; every line and wound; and **a pregnancy test in any woman of childbearing age**.

4. Investigations, in Tiers

- **Bedside: lactate, blood gas, glucose, electrocardiogram, pregnancy test — and bedside ultrasound**, which in a few minutes gives cardiac function, inferior vena cava filling, free intraperitoneal fluid, pneumothorax and the aorta.

- **First line:** complete blood count, coagulation, **cross-match**, urea and electrolytes, liver function, amylase, C-reactive protein, blood cultures, troponin, urinalysis, chest radiograph.

- **Targeted:** echocardiography, computed tomography of the aorta, chest or abdomen, random cortisol, and toxicology.

5. Management While You Are Still Deciding

HOW TO GIVE FLUID

Give balanced crystalloid in boluses of 250 to 500 mL and reassess after each one. Prescribing a litre over an hour and walking away is not resuscitation.

Reassess with: blood pressure, heart rate, capillary refill, urine output, mental state and the lactate.

Three important exceptions:

In haemorrhage, give blood rather than crystalloid — activate the major haemorrhage protocol, give tranexamic acid where indicated, and **accept permissive hypotension until surgical control** in trauma.

In cardiogenic shock, fluid may make things worse — small cautious challenges only, with inotropic support and treatment of the cause.

In obstructive shock, no amount of fluid will work until the obstruction is relieved.

- **Vasopressors once fluid responsiveness is exhausted** — noradrenaline first line. A central line is preferable, but **peripheral administration while one is being placed is better than delay**.
- **Sepsis: antibiotics within one hour, and source control** — remembering that no antibiotic treats an undrained collection (Cases 32 and 48).
- **Anaphylaxis: intramuscular adrenaline, repeated as needed**, plus fluid, and observation for a biphasic reaction.
- Correct calcium, potassium and severe acidosis; **give hydrocortisone if adrenal crisis is conceivable, including in any patient on long-term steroids** (Case 26); and actively rewarm the hypothermic.
- **Document the escalation plan and ceiling of care early**, while the patient or family can still participate.

6. Teaching Points and Viva Questions

- Shock is a perfusion diagnosis. A normal blood pressure does not exclude it.
- The jugular venous pressure and the hands divide the four mechanisms.
- Treat the reversible causes before the diagnosis is complete.
- Fluid in boluses with reassessment after each — never a bag prescribed and forgotten.
- Blood, not crystalloid, in haemorrhage; caution with fluid in cardiogenic shock.
- Follow the lactate.

Questions you should be able to answer:

- Which mechanism is this, and which two bedside findings told you?
- A shocked patient has a raised jugular venous pressure and a clear chest. Give three diagnoses.
- Why is a lactate of 5.8 more useful to you than the blood pressure?
- A trauma patient is hypotensive from abdominal bleeding. How does your fluid strategy differ?
- He does not respond to two litres. What do you do next, and where?

Seminar 13 · Approach to the Patient with Chronic Diarrhoea

THE PRESENTATION

A **34-year-old woman** describes eight months of loose stool four to six times daily, with bloating, and 7 kg of weight loss. **In the last two months she has begun waking at night to open her bowels.** She feels persistently exhausted.

On examination: pale, body mass index 19 kg/m^2, angular stomatitis, mild abdominal distension, no mass, normal perianal examination.

Haemoglobin 96 g/L microcytic · ferritin 4 µg/L · folate low · albumin 34 g/L · C-reactive protein normal · faecal calprotectin normal.

1. Think First — Define It, Then Classify by Mechanism

- **Chronic means more than four weeks.** First distinguish it from **faecal incontinence** and from **overflow diarrhoea with underlying constipation** — patients call all three "diarrhoea", and the management of each is entirely different.

Mechanism	Causes and clues
Osmotic	Lactose intolerance, sugar alcohols, laxatives, magnesium salts. Improves with fasting.
Secretory	Bile acid diarrhoea, microscopic colitis, hormone-secreting tumours (VIPoma, carcinoid, gastrinoma), some infections. Persists with fasting and occurs at night.
Inflammatory	Inflammatory bowel disease, coeliac disease, infection, radiation, ischaemic colitis. Blood, fever, raised inflammatory markers or calprotectin.
Malabsorptive	Coeliac disease, pancreatic exocrine insufficiency, small intestinal bacterial overgrowth, giardiasis, short bowel. Steatorrhoea and nutritional deficiency.
Dysmotility	Irritable bowel syndrome, diabetic autonomic neuropathy, thyrotoxicosis, drugs.

- **Regionally: giardiasis, amoebiasis, intestinal tuberculosis, schistosomiasis, tropical sprue and brucellosis.** Chronic diarrhoea with weight loss here requires stool parasitology and active consideration of tuberculosis (Case 39).

RED FLAGS — INVESTIGATE RATHER THAN LABEL

Weight loss · nocturnal diarrhoea · blood in the stool · anaemia · raised inflammatory markers · onset after the age of 50 · a family history of colorectal cancer or inflammatory bowel disease · fever · steatorrhoea · a short history in an older patient.

This patient has three of them, which is why she needs investigating rather than reassuring.

And irritable bowel syndrome is a positive diagnosis, made on symptom criteria in the absence of red flags — not a label applied when the tests come back normal.

2. The History That Discriminates

- Frequency, consistency, volume, **blood, mucus, urgency, tenesmus, incontinence**, and **nocturnal symptoms**.
- **Steatorrhoea** — pale, bulky, offensive stool that floats and is difficult to flush.
- **Does it stop when she does not eat?** This single question separates osmotic from secretory diarrhoea.

- **The drug chart, which is a common and entirely reversible cause: metformin, magnesium-containing antacids, proton pump inhibitors, antibiotics, laxatives — including covert use — non-steroidal anti-inflammatory drugs, colchicine, selective serotonin reuptake inhibitors, orlistat and chemotherapy.**
- **Previous surgery: cholecystectomy or ileal resection causes bile acid diarrhoea**; gastrectomy and bariatric surgery cause malabsorption. Also radiotherapy.
- **Extraintestinal clues:** an itchy vesicular rash on the elbows and knees (**dermatitis herpetiformis** — Case 76), arthritis, mouth and eye lesions, thyroid symptoms, flushing, diabetes.
- Travel, water source, unpasteurised dairy, contacts, and human immunodeficiency virus risk. Family history of coeliac disease, inflammatory bowel disease and colorectal cancer.

3. Examination

- Weight and nutritional state; pallor; **angular stomatitis and glossitis**; clubbing; mouth ulcers; thyroid; abdominal masses and tenderness; **perianal inspection and rectal examination**; skin, joints and lymph nodes.

4. Investigations, in Tiers

- **First line:** complete blood count, ferritin, B12, folate, urea and electrolytes, liver function, albumin, calcium, C-reactive protein, **thyroid function**, **faecal calprotectin**, **stool culture with ova and parasites on three samples and *Clostridioides difficile* toxin**, and a human immunodeficiency virus test.

COELIAC SEROLOGY, DONE PROPERLY

Send tissue transglutaminase antibody with a total IgA level, in a patient who is still eating gluten.

Two ways this test is wasted: selective IgA deficiency — present in a small but significant minority, and it makes the antibody falsely negative, which is why the total IgA must be requested alongside it; and **a patient who has already started a gluten-free diet**, in whom both the serology and the biopsy may normalise.

Confirm with duodenal biopsy while the patient is still on gluten, then start the diet — not the other way round.

- **Second line, by suspicion: faecal elastase** for pancreatic insufficiency; **bile acid testing or a therapeutic trial of a sequestrant**; breath testing for bacterial overgrowth and lactose intolerance; gut hormones and urinary 5-hydroxyindoleacetic acid; **duodenal biopsy for coeliac disease and giardia**; **colonoscopy with ileal intubation**; computed tomography or magnetic resonance enterography; and a **faecal laxative screen** where surreptitious use is suspected.

TWO DIAGNOSES MISSED BECAUSE THE COLON LOOKS NORMAL

Microscopic colitis — watery secretory diarrhoea, often nocturnal, in a middle-aged or older patient, with **a completely normal-looking colon at endoscopy.** It is diagnosed only on **random biopsies of normal mucosa**, so the biopsies must be taken. It is frequently **drug-induced — proton pump inhibitors, non-steroidal anti-inflammatory drugs, selective serotonin reuptake inhibitors** — and responds to budesonide.

Bile acid diarrhoea — after cholecystectomy, ileal resection or Crohn disease, and often idiopathic. Urgent watery diarrhoea, frequently postprandial. **It responds dramatically to a bile acid sequestrant**, and a therapeutic trial is a reasonable diagnostic test.

Both are common, both are treatable, and both are routinely labelled as irritable bowel syndrome.

5. Management While You Are Still Deciding

- Rehydrate, correct electrolytes, and replace deficiencies — **iron, B12, folate, calcium, vitamin D, magnesium and zinc**.
- **Stop the culprit drug**, which may be the entire treatment.
- **Avoid antidiarrhoeal agents until infection and inflammatory colitis have been excluded** (Case 39).
- **Then treat the cause:** a **gluten-free diet with dietetic support, coeliac follow-up, pneumococcal vaccination and bone density assessment** in coeliac disease; pancreatic enzyme replacement; antimicrobials for giardiasis or overgrowth; a sequestrant for bile acid diarrhoea; budesonide for microscopic colitis; and the therapy of Case 39 for inflammatory bowel disease.
- **In coeliac disease, explain the timescale:** symptoms improve over weeks, the serology falls over many months, and adherence must be lifelong — with first-degree relatives offered testing.

6. Teaching Points and Viva Questions

- Four weeks defines chronic. Distinguish diarrhoea from incontinence and from overflow.
- Nocturnal diarrhoea, weight loss, blood and anaemia are red flags, not features of irritable bowel syndrome.
- Read the drug chart before ordering anything.
- Send total IgA with the coeliac serology, and biopsy before starting the diet.
- Biopsy a normal-looking colon — microscopic colitis is invisible to the eye.
- Think of bile acid diarrhoea after a cholecystectomy, and try a sequestrant.

Questions you should be able to answer:

- Which three red flags does she have, and what do they exclude?
- Her tissue transglutaminase is negative. What must you check before accepting that?
- A 62-year-old has watery nocturnal diarrhoea and a normal colonoscopy. What was not done?
- A patient develops urgent watery diarrhoea after a cholecystectomy. What is the diagnosis and how would you test it?
- Why should the biopsy come before the gluten-free diet?

Seminar 14 · Approach to the Patient with Ascites

THE PRESENTATION

A **46-year-old woman** describes six weeks of increasing abdominal distension with early satiety and weight gain, and is now breathless lying flat. She drinks no alcohol and has no risk factors for liver disease. For three months she has had vague pelvic discomfort and irregular vaginal bleeding.

On examination: tense ascites with shifting dullness. **There are no spider naevi, no palmar erythema and no jaundice. The jugular venous pressure is normal.** There is a firm irregular mass in the pelvis, and **a hard nodule at the umbilicus**.

Ascitic tap: serum-ascites albumin gradient 6 g/L · ascitic protein 42 g/L · neutrophils 40/mm³ · cytology: malignant cells. CA-125 markedly raised. Computed tomography: ovarian mass with peritoneal deposits.

THE POINT OF THIS CASE

Not all ascites is cirrhosis — and the assumption that it is delays the diagnoses that are treatable.

This patient has **no stigmata of chronic liver disease, a normal jugular venous pressure, a low albumin gradient and malignant cytology.** Started on spironolactone and furosemide as "decompensated liver disease", she would have lost months.

Tap the fluid. It is safe under ultrasound, it takes ten minutes, and it answers the question.

1. Think First — the Albumin Gradient Divides It in Two

Serum-ascites albumin gradient = serum albumin minus ascitic albumin, on samples taken at the same time.

Gradient 11 g/L or more — portal hypertension	Gradient below 11 g/L — not portal hypertension
Cirrhosis — the commonest cause overall	Peritoneal malignancy — ovarian, gastric, colorectal, pancreatic, mesothelioma
Alcoholic hepatitis; acute liver failure	Tuberculous peritonitis — important in this region
Heart failure and constrictive pericarditis	Pancreatic ascites
Budd–Chiari syndrome; portal vein thrombosis	Nephrotic syndrome (Seminar 2)
Massive hepatic metastases	Serositis in lupus; chylous ascites
	A REFINEMENT WORTH KNOWING **Cirrhosis: high gradient, LOW ascitic protein.** **Heart failure: high gradient, HIGH ascitic protein.** Both raise the gradient, because both raise sinusoidal pressure — so the **protein** is what separates them. A patient with a high gradient, a high ascitic protein and a raised jugular venous pressure has a cardiac problem, and diuretics will work.

2. What Must Not Be Missed

- **Spontaneous bacterial peritonitis** — **ascitic neutrophils of 250/mm³ or more**, with or without fever, pain or a positive culture. **Every patient admitted with ascites is tapped** (Case 37).

- **Tense ascites causing respiratory compromise**; **hepatorenal syndrome**; a **surgical abdomen** — perforation or ischaemia — presenting as distension; and **malignancy or tuberculosis mistaken for cirrhosis**.

3. The History and Examination That Discriminate

- Speed of onset; abdominal pain and fever; **alcohol quantified and hepatitis risk**; **cardiac symptoms — orthopnoea, oedema, known heart disease**; **weight loss and night sweats, pointing to malignancy or tuberculosis**; **gynaecological history — pelvic pain, irregular bleeding, and family history of ovarian or breast cancer**; frothy urine; diarrhoea; previous abdominal surgery or peritoneal dialysis; tuberculosis contact and unpasteurised milk.

- **First confirm it is ascites:** shifting dullness requires about 1.5 litres, and a fluid thrill more. Distinguish it from obesity, a large ovarian cyst, a distended bladder, pregnancy and gross organomegaly — **ultrasound settles it in a minute**.

- **Then: the presence or absence of chronic liver disease stigmata · the jugular venous pressure · a pelvic and rectal examination, and in women a vaginal examination · the umbilicus for a Sister Mary Joseph nodule · the left supraclavicular fossa · the breasts · peripheral oedema · and the mental state for encephalopathy.**

4. Investigations, in Tiers

- **Diagnostic paracentesis, under ultrasound. Send: cell count and differential; culture in blood culture bottles inoculated at the bedside; albumin and total protein with a paired serum albumin; glucose; lactate dehydrogenase; cytology — and where indicated adenosine deaminase and mycobacterial culture, amylase, and triglycerides.**

- **Interpretation:** the gradient as above; **neutrophils ≥250 means peritonitis**; **a lymphocyte-predominant, high-protein fluid with a high adenosine deaminase suggests tuberculous peritonitis** — where the culture yield is low, so **laparoscopic peritoneal biopsy is often needed and empirical treatment is sometimes justified**; and **a negative cytology does not exclude malignancy**, since sensitivity is moderate at best.

- **Blood:** liver function, INR, albumin, renal function, complete blood count, C-reactive protein, natriuretic peptide, alpha-fetoprotein, and **CA-125 — which is raised in cirrhotic ascites as well, so interpret it with the imaging rather than alone**.

- **Imaging: ultrasound with Doppler** for liver texture, portal and hepatic vein patency, nodules and the pelvis; echocardiography where a cardiac cause is possible; computed tomography of chest, abdomen and pelvis; and **laparoscopy where the cause remains unexplained**.

5. Management While You Are Still Deciding

THE THERAPEUTIC ERROR

Do not diurese an undiagnosed ascites.

Malignant and tuberculous ascites respond poorly or not at all to diuretics, so treating them as cirrhosis produces months of ineffective treatment, electrolyte disturbance and renal impairment while the actual disease progresses. Establish the mechanism first — which takes one tap and one ultrasound.

- **Cirrhotic ascites:** sodium restriction, spironolactone with furosemide, daily weights, and large-volume paracentesis with albumin replacement — Case 37.
- **Malignant ascites:** treat the cancer; **repeated therapeutic paracentesis or an indwelling drain for symptom control**; diuretics help only where the mechanism is massive hepatic metastasis.
- **Tuberculous peritonitis:** standard antituberculous therapy.
- **Cardiac ascites:** treat the heart failure — here diuretics are the treatment (Case 12).
- Nutrition and symptom control; and where malignancy is confirmed, **early palliative care involvement** and an honest conversation.

6. Teaching Points and Viva Questions

- Tap every ascites, and tap it on admission.
- The albumin gradient divides the differential in two; the ascitic protein then separates cirrhosis from heart failure.
- Neutrophils of 250 or more is spontaneous bacterial peritonitis.
- A negative cytology does not exclude malignancy.
- Lymphocytic, high-protein fluid with a raised adenosine deaminase is tuberculosis.
- Examine the pelvis and the umbilicus, and do not diurese an undiagnosed ascites.

Questions you should be able to answer:

- What does her albumin gradient of 6 tell you, and what does it exclude?
- A patient has a gradient of 14 with an ascitic protein of 45 and a raised jugular venous pressure. What is the diagnosis?
- The cytology is negative but you still suspect malignancy. What next?
- What would make you suspect tuberculous peritonitis, and how would you prove it?
- Why is starting spironolactone before the tap a mistake?

Seminar 15 · Approach to the Patient with Dementia

THE PRESENTATION

A **74-year-old man** is brought by his daughter. Over two years he has become forgetful — repeating questions, losing possessions, missing appointments, and recently **becoming lost while driving a familiar route**. He struggles to find words. He has withdrawn from the mosque and from friends. In the last six months bills have gone unpaid and **he has twice left the gas on**.

There is no fluctuation, no hallucination, no falls and no tremor.

On examination: cognitive testing shows impaired recall and orientation with preserved attention. No focal neurological signs, normal gait. Blood pressure 148/86 mmHg. **Hearing is poor and he has no hearing aid.**

Thyroid function normal · B12 low-normal · calcium normal. Computed tomography: generalised atrophy with medial temporal predominance, no vascular lesions.

1. Think First — Three Questions in Order

	Delirium	Dementia	Depression
	QUESTION ONE: IS THIS DEMENTIA, DELIRIUM OR DEPRESSION? **This is the single most important step, and it is where the errors are made.**		
Onset	Hours to days	Months to years	Weeks to months
Course	Fluctuating, worse at night	Progressive	Persistent low mood
Attention	Impaired — the cardinal feature	Preserved until late	Variable, effort-dependent
Consciousness	Clouded	Clear	Clear
Hallucinations	Common	Late, or in Lewy body disease	Uncommon
Answers	Muddled	Confabulated or approximate	"I don't know"

- **Delirium is frequently superimposed on dementia. So a sudden change in someone with established dementia is delirium, and it needs a cause found** — infection, pain, constipation, urinary retention, drugs, metabolic disturbance — not an increase in the antipsychotic.
- **Depression can present as pseudodementia**, with prominent "don't know" answers, and it is reversible with treatment.

QUESTION TWO: IS ANY OF IT REVERSIBLE?

Reversible or contributory causes to exclude in every patient:

Drugs — and this is the highest-yield item on the list: anticholinergics, benzodiazepines, opioids, antipsychotics, antiepileptics, and polypharmacy generally.

Metabolic and deficiency: B12 and folate deficiency, hypothyroidism, hypercalcaemia, hyponatraemia, uraemia, liver disease, recurrent hypoglycaemia.

Structural: chronic subdural haematoma, **normal pressure hydrocephalus**, cerebral tumour.

Infective: human immunodeficiency virus, neurosyphilis.

Other: alcohol and Wernicke–Korsakoff syndrome, obstructive sleep apnoea, depression.

And sensory impairment: untreated deafness and poor vision both mimic and worsen cognitive impairment. In this patient a hearing aid may be part of the treatment, and it is the cheapest intervention available.

Question three: which dementia?

Type	What identifies it
Alzheimer disease	Insidious, episodic memory affected first, then language and visuospatial function; medial temporal atrophy. This patient.
Vascular dementia	Stepwise decline or focal neurological signs, vascular risk factors, imaging burden. Frequently mixed with Alzheimer disease.
Dementia with Lewy bodies	Fluctuating cognition, visual hallucinations, dream enactment, parkinsonism — and severe, sometimes fatal, neuroleptic sensitivity (Case 69).
Frontotemporal dementia	Younger patient, with personality and behavioural change or progressive language failure, and relatively preserved memory early. Frequently misdiagnosed as psychiatric illness.
Others	Parkinson disease dementia, progressive supranuclear palsy, Huntington disease, prion disease (rapid progression), alcohol-related, HIV-associated, normal pressure hydrocephalus.

2. The History — Which Must Come From an Informant

- **Always take a collateral history.** The patient's own account is unreliable by the nature of the condition, and a consultation conducted with the patient alone will underestimate the problem.
- **Which domain failed first** — memory, language, behaviour or visuospatial function — because that is what distinguishes the subtypes.
- **Function, in specifics rather than generalities: finances, medication, cooking, driving, appliances, getting lost, washing and dressing.** This is what determines risk and the support required, and it is more useful than any score.
- Behavioural and psychological symptoms; hallucinations and fluctuation; **dream enactment**; falls; continence; mood and sleep; alcohol; **the complete drug history**; vascular risk factors; family history.
- **Safety, asked explicitly: driving, gas and fire, wandering, and financial vulnerability and exploitation.** This patient has already left the gas on twice and become lost while driving.

3. Examination

- A validated cognitive assessment, noting **which domains** are impaired; **test attention specifically**, to exclude delirium; assess mood.
- **Hearing and vision.**
- Full neurological examination — focal signs, gait, parkinsonism; **postural blood pressure**; cardiovascular examination.
- **Evidence of self-neglect or of neglect by others:** weight loss, dehydration, poor dentition, unexplained bruising, pressure damage.

4. Investigations, in Tiers

- **First line:** complete blood count, urea and electrolytes, **calcium**, glucose or glycated haemoglobin, liver function, **thyroid function, B12 and folate**, C-reactive protein — and **human immunodeficiency virus**

and syphilis serology where there is risk or the picture is atypical.

- **Structural imaging — computed tomography, or preferably magnetic resonance imaging — to exclude a subdural haematoma, tumour or hydrocephalus, to assess vascular burden, and to look at the pattern of atrophy. It is not performed to "confirm dementia".**
- **Specialist:** neuropsychometry; electroencephalography where prion disease or non-convulsive status is suspected; dopamine transporter imaging in suspected Lewy body disease; cerebrospinal fluid or amyloid biomarkers in young-onset or atypical cases; genetic testing in familial disease.
- **Refer urgently rather than routinely if: the onset is before 65, progression is rapid, behaviour or language change dominates, or there are focal neurological signs.**

5. Management While You Are Still Deciding

- **Treat what is treatable first. A medication review — stopping anticholinergic and sedative drugs — is among the highest-yield interventions in the whole assessment.** Correct deficiencies, treat depression and sleep apnoea, **fit hearing aids and correct vision**, and manage vascular risk.
- **Cholinesterase inhibitors** — donepezil, rivastigmine or galantamine — in Alzheimer, Lewy body and Parkinson disease dementia; **memantine** in moderate to severe Alzheimer disease or where inhibitors are not tolerated. **The benefit is modest and symptomatic: say so, or the family will expect reversal.**

BEHAVIOURAL AND PSYCHOLOGICAL SYMPTOMS

Look for a cause before you prescribe. Agitation, aggression and shouting are usually communication, not psychosis.

The usual causes: pain · constipation · urinary retention · infection · hunger or thirst · fear · boredom · too much noise · an unfamiliar place · a new carer. Look for each, and use non-pharmacological approaches first.

Antipsychotics increase the risk of stroke and death in dementia. They are a last resort, at the lowest dose, for a defined period, with a documented review date — and they are dangerous in dementia with Lewy bodies, where sensitivity can be fatal.

WHERE THE REAL WORK IS

Occupational therapy and a home assessment · a care package and day services · dementia-friendly environment · nutrition, dental and foot care · vaccination.

Carer health is a clinical issue. Carer strain predicts institutionalisation and carer illness; ask about it directly and offer support and respite.

Do the legal and financial planning while capacity remains — lasting power of attorney, advance decisions, a will, and an advance care plan. **Capacity is decision-specific and fluctuates: assess it for the decision in front of you.**

Driving must be discussed and documented, with the legal duty to notify the licensing authority explained.

Then an honest prognosis, a palliative approach in advanced disease, and an agreed plan that avoids futile hospital admission.

6. Teaching Points and Viva Questions

- Dementia, delirium or depression — decide that first, and test attention to do it.
- A sudden change in someone with dementia is delirium, and needs a cause.
- Always take an informant history, and ask about function in specifics.

- Review the drugs — it is the highest-yield intervention.
- Fit the hearing aid.
- Find the cause of agitation before prescribing an antipsychotic, and never use one lightly in Lewy body disease.
- Do the legal planning while capacity remains.

Questions you should be able to answer:

- How would you distinguish this from delirium at the bedside?
- Name six reversible contributors you would exclude, and the test for each.
- Why does his hearing matter to his cognition?
- His daughter reports he has become aggressive at bath time. What do you do before prescribing anything?
- What must be arranged while he still has capacity?

Seminar 16 · Approach to the Patient with Acute Poisoning

THE PRESENTATION

A **22-year-old woman** is brought in six hours after taking "a lot of tablets" following an argument. Empty packets of paracetamol and ibuprofen were found beside her, and she also drank vodka. She will not say how many she took.

On examination: drowsy but rousable, Glasgow Coma Scale 14. She has vomited twice. Respiratory rate 22/min, heart rate 104/min, blood pressure 112/70 mmHg, temperature 36.6 °C, pupils 3 mm and reactive, glucose 5.2 mmol/L.

Alanine aminotransferase normal · INR 1.1 · lactate 2.4 · salicylate not detected · paracetamol level pending.

1. Think First — Four Questions

- **What, how much, when, and with what else?** — and **the history in poisoning is frequently wrong**, whether through confusion, shame or intent. **Assume the worst plausible scenario and treat it.**
- **Is there a specific antidote or elimination strategy, and is there a time window?**
- **Does the toxidrome fit the story?** If it does not, believe the patient's physiology rather than their account.
- **Why did this happen, and what is the risk of it happening again?** The psychosocial assessment is part of the medical management, not an addendum to it.

Toxidrome	Features and immediate action
Opioid	Pinpoint pupils, respiratory depression, reduced consciousness → naloxone, by infusion for long-acting agents
Sedative — benzodiazepine, alcohol	Reduced consciousness with relatively preserved respiration early, normal pupils. Flumazenil is rarely used — it precipitates seizures
Anticholinergic	"Mad, hot, dry, red, blind and full" — delirium, dry skin, dilated pupils, urinary retention, tachycardia
Cholinergic — organophosphate	Salivation, lacrimation, urination, defaecation, bronchorrhoea, bradycardia, small pupils, fasciculation → atropine in large repeated doses titrated to drying of secretions, plus pralidoxime. Decontaminate, and protect yourself and the staff
Sympathomimetic — cocaine, amphetamine	Agitation, dilated pupils, tachycardia, hypertension, hyperthermia and sweating (which distinguishes it from anticholinergic) → benzodiazepines first; avoid a beta blocker alone
Serotonin syndrome	Clonus and hyperreflexia, agitation, hyperthermia, diarrhoea, rapid onset after a serotonergic drug → benzodiazepines, cooling, cyproheptadine
Salicylate	Tinnitus, hyperventilation, vomiting, sweating, a mixed respiratory alkalosis with metabolic acidosis

2. What Could Kill in the Next Hour

- **Airway and breathing** — the commonest cause of death in poisoning is airway obstruction and aspiration, not the drug itself.
- **Hypoglycaemia**; **arrhythmia** — check the electrocardiogram for **a wide QRS from sodium channel blockade, which is treated with sodium bicarbonate**, and for a prolonged QT interval; **seizures**; **hyperthermia**, which requires active cooling.

- **And the patient who looks well.** Paracetamol produces no symptoms for 24 hours and then destroys the liver. **The level, not the appearance, decides the treatment.**
- **Use the poisons information service or database in every significant case.** It is a core clinical resource, not a last resort, and it should be consulted early.

3. Decontamination and Elimination

- **Activated charcoal within about one hour of ingestion, in a patient who can protect their airway.** It is of limited value later, and **useless for iron, lithium, alcohols and corrosives**.
- **Gastric lavage and ipecac are obsolete.** Do not use them.
- **Whole bowel irrigation** for modified-release or enteric-coated preparations in selected cases.
- **Urinary alkalinisation** for salicylate; **haemodialysis for lithium, salicylate, toxic alcohols, metformin-associated lactic acidosis, valproate and theophylline**; **intravenous lipid emulsion** in refractory local anaesthetic and some lipophilic drug toxicity.

Poison	Antidote
Paracetamol	N-acetylcysteine
Opioid · benzodiazepine	Naloxone · flumazenil (rarely)
Tricyclic antidepressant	Sodium bicarbonate for a wide QRS
Beta blocker · calcium channel blocker	Glucagon · calcium; high-dose insulin with euglycaemia for both
Digoxin	Digoxin-specific antibody fragments
Iron · methanol and ethylene glycol	Desferrioxamine · fomepizole or ethanol, with haemodialysis
Organophosphate · warfarin	Atropine with pralidoxime · vitamin K and prothrombin complex concentrate
Isoniazid · sulfonylurea	Pyridoxine · glucose with octreotide (Case 21)
Carbon monoxide · cyanide · methaemoglobinaemia	High-flow oxygen · hydroxocobalamin · methylene blue — with caution in glucose-6-phosphate dehydrogenase deficiency
	PARACETAMOL — THE ONE TO GET RIGHT **Asymptomatic for the first 24 hours**, then right upper quadrant pain and vomiting, then hepatic failure at three to four days. **The patient who feels fine is the one who needs the level.** **Measure the level at four hours or later after a single acute ingestion and treat according to the nomogram.** **Start N-acetylcysteine without waiting for the level if: the overdose was staggered or the timing is unknown · the patient presents more than eight hours after ingestion · or the patient is unwell.** The antidote loses efficacy after eight hours, so the balance shifts firmly towards treating. **Monitor INR, alanine aminotransferase, creatinine, pH and lactate — and note that the INR and the pH are the markers of severity, not the aminotransferase.** Use the King's College criteria and refer to a liver unit early (Case 35). **Anaphylactoid reactions to N-acetylcysteine are common: slow the infusion, treat the reaction, and continue — do not abandon the antidote.**

4. Management While You Are Still Deciding

- Airway, breathing and circulation; oxygen; glucose; monitoring; intravenous access; **electrocardiogram with attention to the QRS width and the QT interval**; temperature; and a **pregnancy test**.
- Supportive care is the mainstay for most poisons: **most patients survive because of good airway and circulatory management, not because of an antidote.**
- Correct electrolytes and acid–base disturbance; treat seizures with benzodiazepines; cool the hyperthermic patient actively; and observe for the delayed effects of modified-release preparations.

THE HALF OF THE ASSESSMENT THAT IS NOT THE DRUG

Every patient presenting with self-poisoning requires a risk and mental health assessment before discharge, by someone competent to perform it.

Establish: intent and planning · precautions taken against discovery · ongoing suicidal ideation · the means available and the access to them · previous attempts · psychiatric and substance misuse history · **and the social circumstances, including abuse and coercion, which may be the reason for the presentation.**

Treat the physical and the psychiatric problem together. A patient discharged "medically fit" without an assessment has been half treated — and the risk of repetition and of completed suicide is highest in the weeks that follow.

Also consider the non-suicidal explanations: accidental ingestion, therapeutic error, drug interaction, recreational use, a child, an older person with cognitive impairment, and **deliberate poisoning by another person.**

Arrange safety planning, discuss means restriction with the family, and **arrange follow-up before discharge rather than after it.**

5. Teaching Points and Viva Questions

- Treat the patient, not the poison — supportive care saves more lives than antidotes.
- The history is often wrong. Let the toxidrome and the tests guide you.
- Use the poisons information service early.
- Charcoal only within the hour and with a protected airway; lavage is obsolete.
- Paracetamol is silent then lethal — treat on the level, or blind if staggered, late or unwell.
- The INR and the pH, not the aminotransferase, tell you how sick the liver is.
- Check the QRS width on the electrocardiogram.
- No self-poisoning is discharged without a mental health assessment.

Questions you should be able to answer:

- She cannot say how much she took or when. How does that change your management?
- Give three situations in which you would start N-acetylcysteine without a level.
- Her aminotransferase is normal at eight hours. Is that reassuring?
- A patient arrives drowsy with a QRS of 140 ms. What is the likely poison and the treatment?

- She is medically fit at 24 hours and wants to go home. What must happen first?