

General Instructions

- (i) This booklet contains 142 questions, each provided with a complete, step-by-step solution.
- (ii) It comprises 142 single-correct multiple-choice questions.
- (iii) Attempt each question on your own before reviewing the given solution.

1.

Which area is fractured in the given X-ray?



- (A) Tibial tuberosity
- (B) Medial epicondyle of tibia
- (C) Gerdy's tubercle

Correct Answer: (A) Tibial tuberosity

SOLUTION:

Step 1: Localise the lesion on the film.

The fracture sits on the front of the upper (proximal) end of the tibia, at the bony bump just below the kneecap where the patellar tendon pulls. This anterior projection is the landmark we must name.

Step 2: Match the landmark to its anatomy.

The patellar ligament carries the strong pull of the quadriceps through the patella down onto this anterior eminence. Because the tendon's force is concentrated here, avulsion-type and direct-impact fractures are typical — classically seen in adolescents (the Osgood-Schlatter spectrum) and after sudden forceful knee extension. That bump is the tibial tuberosity.

Step 3: Rule out the distractors by location.

- A medial epicondyle of the tibia is not a recognised proximal landmark in this anterior position, so it does not fit the imaged site.
- Gerdy's tubercle lies on the lateral (outer) surface of the proximal tibia and is the insertion of the iliotibial band; it is not the anterior midline bump shown and fractures there are uncommon.

Step 4: Conclude.

The anterior, tendon-loaded prominence below the knee that is fractured is the tibial tuberosity.

Final answer: Option 1 — Tibial tuberosity.

Quick Tip: Fractures of the tibial tuberosity are often seen in active adolescents and can result from trauma or repetitive stress.



2.

Which nerve will be commonly involved if the injury occurs at the marked levels?



- (A) L4
- (B) L5
- (C) L2
- (D) L3

Correct Answer: (B) L5

SOLUTION:

Step 1: Read what the marked level supplies.

The injury is flagged at a lower lumbar segment. To name the nerve, ask which root governs the motor sign that level is known for. The hallmark of this root is dorsiflexion of the foot (lifting the foot and great toe upward) plus sensation over the dorsum of the foot — that motor function points to L5.

Step 2: Build the lumbar root map for cross-checking.

- L2 — hip flexion.
- L3 — knee extension.
- L4 — quadriceps and the knee-jerk reflex.
- L5 — dorsiflexion (foot/great-toe lift), the function tied to the marked level.

Step 3: Apply clinical frequency.

The L5 root is one of the most commonly injured because of its weight-bearing position and the mechanical stress at the lower lumbar spine, so a lesion at the marked level characteristically disturbs the foot-lifting (extensor) muscles it controls.

Step 4: Conclude.

The root that matches both the segment marked and the dorsiflexion deficit is L5.

Final answer: Option 2 — L5.

Quick Tip: L5 nerve root lesions are often associated with foot drop and weakness in the dorsiflexion of the foot, and they are common in lumbar spine injuries.

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3.

What is the most likely diagnosis for the lesion shown here?



(A)

Sunburst appearance

(B)

Onion-skin appearance

(C)

Codman's triangle

(D)

Ground glass appearance

Correct Answer: (A)

Sunburst appearance

SOLUTION:

Step 1: Recognise the radiological pattern.

The image shows fine, radiating spicules of new bone streaming outward from the cortex, like rays from the sun. This perpendicular spiculated periosteal reaction is described as a sunburst (sunray) appearance and is a classic aggressive bone-lesion sign, most associated with osteosarcoma.

Step 2: Why this pattern arises.

When a fast-growing tumour breaks through the cortex, it lays down bone along the stretched, lifted periosteal vessels, producing the radiating spicules seen here. The aggressive, perpendicular pattern distinguishes it from slower benign reactions.

Step 3: Separate it from the other named reactions.

- Onion-skin appearance — concentric, multilayered periosteal lamellae (typical of Ewing sarcoma), not radiating rays, so it does not match.
- Codman's triangle — a triangular cuff of reactive bone where the elevated periosteum meets cortex; it is a localized angular lift, not the diffuse radiating spicules shown.
- Ground-glass appearance — a hazy, homogenous matrix (e.g., fibrous dysplasia), the opposite of the sharp radiating lines here.

Step 4: Conclude.

The radiating, sun-ray spicules identify the lesion as a sunburst appearance.

Final answer: Option 1 — Sunburst appearance.

Quick Tip: The sunburst pattern is often seen in aggressive bone tumors like osteosarcoma and is a key diagnostic feature on radiographs.

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4. A patient from a railway station is arrested by police and shows symptoms like dilated pupils, sweating, hot skin, and staggering gait. He is suspected to have intoxication from:

- (A) Datura
- (B) Cocaine
- (C) Alcohol
- (D) Morphine

Correct Answer: (A) Datura

SOLUTION:

Step 1: Group the symptoms into a toxidrome.

Dilated pupils (mydriasis), sweating yet hot, flushed skin, and an unsteady staggering gait point to an anticholinergic state — a substance blocking acetylcholine, producing the classic "hot as a hare, dry/flushed, blind as a bat, mad as a hatter" picture with ataxia.

Step 2: Match the toxidrome to the agent.

Datura (Datura) seeds contain tropane alkaloids — atropine, scopolamine (hyoscine) and hyoscyamine — which are potent muscarinic antagonists. They reproduce exactly this combination: mydriasis, hot flushed skin, agitation/delirium and an unsteady gait. Such poisonings are well documented in railway-station "road-side poisoning" robberies, fitting the scenario.

Step 3: Eliminate the other options.

- Cocaine — a sympathomimetic; it dilates pupils but typically gives tachycardia, hypertension and euphoria rather than this full anticholinergic gait/skin picture.
- Alcohol — causes a staggering gait but usually does not dilate pupils or give hot, dry skin.
- Morphine — an opioid producing pinpoint (constricted) pupils and CNS/respiratory depression, the reverse of mydriasis here.

Step 4: Conclude.

Only Dhatura explains every feature through its anticholinergic alkaloids.

Final answer: Option 1 — Dhatura.

Quick Tip: Dhatura poisoning is characterized by anticholinergic symptoms, including dilated pupils, dry skin, and altered mental status.

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5. In MTP at 28 weeks for congenital anomalies, whose presence is not required?

- (A) Obstetrician
- (B) Lawyer
- (C) Pediatrician
- (D) Sonologist

Correct Answer: (B) Lawyer

SOLUTION:

Step 1: Frame the question medically, not legally.

A medical termination of pregnancy at 28 weeks for a congenital anomaly is a clinical decision. The team needed is one that can diagnose the anomaly, confirm it on imaging, perform/manage the procedure and assess the fetus — i.e., medical and diagnostic specialists.

Step 2: Identify who each clinician contributes.

- Obstetrician — conducts and manages the termination procedure itself.
- Sonologist — performs detailed ultrasound to confirm and characterise the anomaly.
- Pediatrician (neonatologist) — assesses fetal/neonatal viability and condition.

These three are genuinely required for the medical assessment.

Step 3: Spot the non-medical role.

A lawyer adds no clinical input. The legal permissibility is handled by following the statutory framework and the opinion of the registered medical practitioners; a lawyer's physical presence is not part of the medical board needed for this decision.

Step 4: Conclude.

Of the four, the presence not required is the lawyer.

Final answer: Option 2 — Lawyer.

Quick Tip: MTP at 28 weeks due to congenital anomalies requires the involvement of medical professionals like obstetricians, pediatricians, and sonologists, but not necessarily a lawyer.

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6. A child was born after the death of his father, and some of them claimed that the child is not from his biological father. DNA fingerprinting was done to identify the father, and the deceased father was found to be the biological father. This child is called as:

- (A) Posthumous child
- (B) Suppositious child
- (C) Illegitimate child

Correct Answer: (A) Posthumous child

SOLUTION:

Step 1: Pin down the defining feature.

The key fact is the timing of birth relative to the father: the child was born after the father had already died. The term we need is the one that specifically denotes "born after the father's death."

Step 2: Define the candidate terms.

- Posthumous child — a child delivered after the biological father has died. This matches the scenario precisely.
- Suppositious child — a child fraudulently passed off as another woman's offspring; this concerns false maternity, not the father's death.
- Illegitimate child — a child born to parents not lawfully married; here paternity was confirmed, so this label does not apply.

Step 3: Use the DNA evidence.

DNA fingerprinting confirmed the deceased man as the true biological father, settling the dispute over parentage. With biological paternity established and birth occurring after his death, the medico-legal term is fixed.

Step 4: Conclude.

A child born after the death of the (confirmed) biological father is a posthumous child.

Final answer: Option 1 — Posthumous child.

Quick Tip: Posthumous children are conceived after the father's death and are legally considered as his children if paternity can be confirmed.



7. Which one is correct about the sequence of rigor mortis?

- (A) Centre to periphery
- (B) Head to foot
- (C) Foot to head
- (D) Simultaneously

Correct Answer: (B) Head to foot

SOLUTION:

Step 1: Recall the biochemical driver of rigor mortis.

After death, ATP is no longer regenerated, so actin and myosin lock together and muscles stiffen. The smaller the muscle mass, the sooner its ATP is exhausted — so the order of stiffening follows muscle size, smallest first.

Step 2: Translate that into a body sequence.

The smallest muscle groups are in the face, jaw and neck, so stiffness appears there first. It then spreads to the trunk and limbs and reaches the larger muscles of the legs and feet last. This gives a top-down progression: head to foot (the descending pattern; Nysten's rule).

Step 3: Reject the other patterns.

- Centre to periphery — not how rigor spreads; it is governed by muscle size, not radial distance.
- Foot to head — reverses the true order, since the large foot/leg muscles stiffen last, not first.
- Simultaneously — incorrect, because differing muscle masses deplete ATP at different rates, giving a sequence rather than a single moment.

Step 4: Conclude.

Rigor mortis classically advances from head to foot.

Final answer: Option 2 — Head to foot.

Quick Tip: Rigor mortis begins in the small muscles of the face and progresses down the body, ending in the lower extremities.

8. An 18-year-old male accused of rape claims he is 16 years old. Which joint X-ray should be done?

- (A) Elbow and ankle
- (B) Knee and wrist
- (C) Shoulder and head
- (D) Hip & knee

Correct Answer: (B) Knee and wrist

SOLUTION:

Step 1: Decide what we must prove.

The accused says he is 16; the question is whether he has actually crossed the threshold near 18. Age estimation in the late teens relies on which epiphyseal (growth) plates have fused, because each joint fuses at a known age window.

Step 2: Pick joints whose fusion brackets 16 to 18.

The wrist and knee are ideal for this age band:

- Wrist — the lower end of the radius fuses around 18–19 years; an unfused versus fusing radius helps separate 16 from 18.
- Knee — the lower femur and upper tibia epiphyses fuse around 17–19 years in males, again straddling the disputed age.

Together they give a reliable read on whether bone age is nearer 16 or 18.

Step 3: Why not the other pairings.

Elbow-and-ankle, shoulder-and-head, and hip-and-knee combinations either fuse too early or do not give the same tight 16–18 discrimination, so they are less useful for resolving this specific claim.

Step 4: Conclude.

To test the claimed age of 16 against ~18, radiograph the knee and wrist for epiphyseal fusion.

Final answer: Option 2 — Knee and wrist.

Quick Tip: Knee and wrist X-rays are standard for assessing skeletal maturity and estimating age, as these joints exhibit predictable patterns of growth plate closure.

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9.

What is the management of the endoscopic finding given in the image?



- (A) Stenting
- (B) Balloon dilatation
- (C) Fulguration
- (D) Internal urethrotomy

Correct Answer: (C) Fulguration

SOLUTION:

Step 1: Read the endoscopic picture.

The image is taken during cystourethroscopy and shows a small, flat, abnormal mucosal lesion within the lumen rather than a long fibrotic narrowing or a tight ring stricture. When the abnormality is a localised, superficial lesion (for example a small papillary growth, a posterior urethral valve leaflet, or focal abnormal mucosa) the goal of treatment is to **destroy the offending tissue**, not to widen a passage.

Step 2: Match the lesion to the right tool.

Tissue-destroying interventions delivered through the endoscope are electrosurgical. **Fulguration** uses a high-frequency electric current (electrocoagulation/diathermy) to char and ablate abnormal mucosa or a lesion under direct vision, sealing the base as it works.

Step 3: Eliminate the lumen-widening options.

- *Stenting* keeps a lumen patent (e.g. across an obstruction) but removes no tissue.
 - *Balloon dilatation* stretches a stricture radially; again no ablation.
 - *Internal (optical) urethrotomy* incises a urethral stricture to relieve narrowing.
- All three address obstruction or stricture, so none fits a finding that calls for destruction of a lesion.

Step 4: Conclusion.

Because the indicated treatment is ablation of abnormal tissue seen endoscopically, the appropriate management is electrosurgical fulguration.

Final answer: Option 3 — Fulguration.

Quick Tip: Fulguration is a useful treatment option for superficial lesions, where heat is applied to destroy the abnormal tissue.

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10.

Identify the image given in UGIE (Upper Gastrointestinal Endoscopy):



- (A) Barrett's esophagus
- (B) Esophageal varices
- (C) Gastric erosion
- (D)

Correct Answer: (B) Esophageal varices

SOLUTION:

Step 1: Set the clinical frame.

This is an upper GI endoscopy. The pattern recognition question asks which lesion the picture shows in the lower oesophagus / gastro-oesophageal region.

Step 2: Pin down the discriminating sign.

The classic finding for the correct entity is **dilated, tortuous, bluish, serpentine (snake-like) submucosal veins** bulging into the lower oesophageal lumen, running as longitudinal columns toward the gastro-oesophageal junction. These engorged collateral veins are the endoscopic signature of **oesophageal varices**, which form because portal hypertension forces blood through porto-systemic collaterals at the lower oesophagus.

Step 3: Contrast with the distractors so the match is unambiguous.

- *Barrett's oesophagus* would appear as salmon-pink columnar mucosa extending in tongues up from the GE junction — a colour/mucosal change, not raised vessels.
 - *Gastric erosion* is a shallow mucosal break or superficial defect on the stomach lining — a flat lesion, not dilated veins.
- Neither shows the bluish dilated venous columns in the picture.

Step 4: Conclusion.

The dilated bluish serpentine veins identify the lesion as varices arising from portal hypertension.

Final answer: Option 2 — Esophageal varices.

Quick Tip: Esophageal varices are a common finding in patients with liver cirrhosis and portal hypertension, and they require careful management to prevent life-threatening bleeding.

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11.

A 12-year-old child presented with sudden severe onset of scrotal pain after a history of trauma 6 hours before. Intraoperative image is given. What is the likely diagnosis?



- (A) Torsion testis with gangrene
- (B) Testicular hematoma
- (C) Torsion testis
- (D) Gangrene of testis

Correct Answer: (A) Torsion testis with gangrene

SOLUTION:

Step 1: Build the clinical timeline.

A 12-year-old boy — the peak age for testicular torsion — develops sudden, severe scrotal pain, and the operation is happening **about 6 hours after onset**. Torsion is a surgical emergency precisely because the spermatic cord twist throttles the blood supply.

Step 2: Apply the ischaemia clock.

Once the cord twists, the testis becomes ischaemic. Salvage is excellent if detorsion happens within roughly 6 hours; beyond that window the gland

progressively necroses, and by 6–12 hours frank gangrene is common. At the 6-hour mark, irreversible damage is already very likely.

Step 3: Read the intraoperative appearance against the clock.

At surgery a torted, non-salvageable testis looks dusky/black, congested and non-viable — i.e. it has progressed past simple torsion into **gangrene**. So the picture is not merely a twisted but viable testis; it is torsion that has already infarcted.

Step 4: Sort the options.

- *Torsion testis* alone describes the mechanism but understates the operative finding of necrosis.
- *Testicular haematoma* implies a blood collection from trauma, not the cord-twist ischaemia seen here.
- *Gangrene of testis* names the end-state but omits the causative torsion.

The complete diagnosis must capture both the cause (torsion) and the consequence (gangrene).

Step 5: Conclusion.

Sudden pain, peak age, a 6-hour ischaemic interval and a non-viable gland together indicate torsion that has gone on to gangrene.

Final answer: Option 1 — Torsion testis with gangrene.

Quick Tip: Testicular torsion is a surgical emergency, and prompt intervention within 6 hours is crucial to save the testis from gangrene.

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12. What is the negative pressure in VAC dressing?

- (A) 60-90 mmHg
- (B) 90-100 mmHg
- (C) 120-130 mmHg
- (D) 130-150 mmHg

Correct Answer: (C) 120-130 mmHg

SOLUTION:

Step 1: Recall what VAC therapy does.

Vacuum-Assisted Closure (VAC), also called Negative Pressure Wound Therapy (NPWT), applies controlled sub-atmospheric suction to a sealed foam dressing over a wound. The suction removes excess interstitial fluid and exudate, reduces oedema, draws the wound edges inward, improves local perfusion and stimulates granulation tissue.

Step 2: Recall the standard pressure setting.

The conventionally taught therapeutic negative pressure for continuous NPWT is around **125 mmHg** below atmospheric. Among the choices, the band that contains this value is **120–130 mmHg**.

Step 3: Reject the other ranges.

- 60–90 mmHg and 90–100 mmHg are below the standard effective setting (such low pressures are reserved for special situations, not the default).
- 130–150 mmHg overshoots the usual prescribed level.

Only the **120–130 mmHg** band brackets the standard **125 mmHg**.

Step 4: Conclusion.

The routine negative pressure used in VAC dressing centres on **125 mmHg**, i.e. the **120–130 mmHg** range.

Final answer: Option 3 — 120–130 mmHg.

Quick Tip: The optimal range for negative pressure in VAC dressing is 120-130 mmHg to effectively promote wound healing and tissue regeneration.



13. Identify the structure marked in the intraoperative image of congenital inguinal hernia:

- (A) Femoral vein
- (B) Obturator vein
- (C) Testicular vein
- (D) Inferior epigastric vein

Correct Answer: (C) Testicular vein

SOLUTION:

Step 1: Anchor on the operative field.

This is a congenital inguinal hernia repair, so the dissection is happening at the **spermatic cord within the inguinal canal**. The structure to identify must therefore be a vein that normally travels with the cord.

Step 2: List what runs in the spermatic cord.

The cord contains the vas deferens, the testicular artery, and the venous drainage of the testis — the pampiniform plexus, which coalesces to form the **testicular (gonadal) vein**. A vein marked alongside the cord structures must be this testicular vein.

Step 3: Exclude veins that do not belong to the cord.

- *Femoral vein* lies in the femoral sheath below the inguinal ligament, medial to the femoral artery — outside the cord.
- *Obturator vein* is a deep pelvic vein draining to the internal iliac vein — not encountered in routine cord dissection.
- *Inferior epigastric vein* runs in the anterior abdominal wall and is the medial boundary that separates direct from indirect hernias; it is a landmark, not a cord vein.

None of these accompanies the testis within the canal.

Step 4: Conclusion.

The only listed vein intrinsic to the spermatic cord/inguinal canal is the testicular vein, so that is the marked structure.

Final answer: Option 3 — Testicular vein.

Quick Tip: The testicular vein should be identified and preserved during the repair of a congenital inguinal hernia to prevent damage to the venous drainage of the testes.

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14. How does a skin graft derive its nutrition on day 3?

- (A) Plasma imbibition
- (B) Inosculation
- (C) Neovascularization
- (D) None

Correct Answer: (B) Inosculation

SOLUTION:

Step 1: Frame the question around the timeline of graft take.

A free skin graft has no blood supply of its own at the moment it is laid down, so it is nourished in three sequential phases. The question asks specifically what is supplying it on **day 3**.

Step 2: Walk through the three phases.

- *Phase 1 — Plasma imbibition (roughly days 1–2):* the graft passively soaks up plasma and nutrients from the recipient wound bed, keeping it hydrated and alive while no vessels are yet connected.
- *Phase 2 — Inosculation (around day 3):* cut capillary ends in the graft line up and physically anastomose with the host's capillaries at the wound bed, restoring an actual circulatory connection.
- *Phase 3 — Neovascularisation/revascularisation (later):* new vessels grow into the graft to establish a durable, mature blood supply.

Step 3: Map day 3 to the right phase.

By the third day, imbibition is handing over to the linking-up of capillaries, so the graft is now drawing nutrition through the newly connected vessels — that is inosculation. Plasma imbibition is too early, and neovascularisation comes too late, so neither fits day 3.

Step 4: Conclusion.

On day 3 the graft survives via direct capillary linkage between graft and host.

Final answer: Option 2 — Inosculation.

Quick Tip: Inosculation is critical in the early stages after skin grafting, as it establishes blood supply between the graft and the recipient tissue.

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15.

Identify the ulcer:



- (A) Arterial
- (B) Venous
- (C) Trophic
- (D) Diabetic foot ulcer

Correct Answer: (B) Venous

SOLUTION:

Step 1: Decide what features to look for in a leg ulcer.

Leg ulcers are sorted by their site, edge, base and surrounding skin. Reading those four cues off the picture lets us assign the underlying cause.

Step 2: Identify the signature pattern in the image.

The ulcer sits **over the lower leg, classically the medial malleolus (gaiter area)**; it is **shallow with an irregular, sloping border**; and the surrounding skin shows the stigmata of **venous insufficiency** — oedema, hyperpigmentation/haemosiderin staining and often varicose veins. This constellation is diagnostic of a **venous ulcer**.

Step 3: Differentiate from the other ulcer types.

- *Arterial ulcer*: on toes, heel or pressure points, painful, punched-out with a deep, well-defined edge and a poorly perfused base — not shallow or ankle-sited.
- *Trophic ulcer*: a painless, punched-out ulcer over a pressure/weight-bearing point from neuropathy — lacks the venous skin changes.
- *Diabetic foot ulcer*: over foot pressure points in a diabetic, mixed neuropathic-ischaemic, prone to infection — foot-located, not in the gaiter zone with venous staining.

Step 4: Conclusion.

A shallow, irregular ulcer at the lower leg/ankle with oedema and pigmentation points to chronic venous insufficiency.

Final answer: Option 2 — Venous.

Quick Tip: Venous ulcers are often seen in the lower legs and are caused by poor venous circulation, leading to stagnation and skin breakdown.

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16. A 30-year-old female presented with swelling in the neck. What is the next step in evaluation?

- (A) I131
- (B) FNAC
- (C) TSI & T4
- (D) FSH

Correct Answer: (C) TSI & T4

SOLUTION:

Step 1: Define the clinical problem.

A 30-year-old woman with neck swelling most often has a thyroid lesion. Before deciding whether the swelling is structural (a nodule needing tissue diagnosis) or functional/autoimmune, the logical first move is to learn how the gland is **working**.

Step 2: Establish the evaluation principle.

Thyroid work-up begins with biochemistry. Assessing function with **thyroid hormone (T4)** and, where autoimmune disease is suspected, **Thyroid-Stimulating Immunoglobulin (TSI)** tells you whether the patient is hyper-, hypo- or euthyroid and whether an autoimmune process (e.g. Graves' disease) is driving the swelling. This biochemical status then guides every subsequent test.

Step 3: Test each option against that principle.

- *I¹³¹I uptake/scan*: useful for characterising function/nodules, but it is not the first-line step and is generally deferred until biochemistry suggests it.
- *FNAC*: the cytology test of choice for a discrete suspicious nodule, but it answers a structural/malignancy question, not the initial functional screen.
- *FSH*: a reproductive hormone with no role in evaluating a thyroid swelling.
- *TSI & T4*: directly assesses gland activity and autoimmunity — the appropriate next step.

Step 4: Conclusion.

The next step in evaluating thyroid-origin neck swelling is to check function and autoimmune status with TSI and T4.

Final answer: Option 3 — TSI & T4.

Quick Tip: Measuring thyroid hormones (TSH, T3, T4) is crucial for diagnosing thyroid disorders in patients with neck swelling.

17. A 5-year-old girl was washing her doll with shampoo containing a chemical Rotenone. Her mother notices her in an unconscious state. Which of the following complex is inhibited by the above-mentioned chemical?

- (A) NADH Dehydrogenase
- (B) Succinate DH
- (C) Cytochrome C
- (D) Cytochrome oxidase

Correct Answer: (A) NADH Dehydrogenase

SOLUTION:

Step 1: Spot the toxicology trigger.

Rotenone is a classic mitochondrial poison used in pesticides and some shampoos. Sudden unconsciousness after exposure points to a sudden collapse of cellular energy (ATP) production, so the lesion must sit on the electron transport chain (ETC).

Step 2: Recall where Rotenone acts mechanistically.

Rotenone is a lipophilic molecule that plugs the ubiquinone (coenzyme Q) binding pocket of the very first ETC pump. It therefore blocks the handoff of electrons from NADH-fed iron-sulfur clusters to coenzyme Q. With this gate shut, the entire NADH-linked arm of respiration stalls.

Step 3: Match the block to a numbered complex.

The pump that accepts electrons from NADH and passes them to coenzyme Q is Complex I, also called NADH dehydrogenase (NADH:ubiquinone oxidoreductase). This is exactly the protein Rotenone disables.

Step 4: Confirm the other complexes are bystanders.

- Succinate dehydrogenase (Complex II) feeds electrons via FADH_2 and bypasses the Rotenone block, so it keeps working.
- Cytochrome c is only a mobile shuttle between Complexes III and IV, not a Rotenone target.

- Cytochrome oxidase (Complex IV) is the cyanide/CO target, not the Rotenone target.

Step 5: Conclusion.

Because Rotenone selectively jams the NADH → coenzyme Q step at the chain's entry pump, the inhibited complex is NADH dehydrogenase (Complex I).

Final answer: Option 1 — NADH Dehydrogenase.

Quick Tip: Rotenone inhibits NADH dehydrogenase, disrupting ATP production and leading to cellular dysfunction and toxicity.

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18.

Which of the following is true regarding the image provided?



- (A) EMLA contains ropivacaine and ligocaine.
- (B) EMLA can lead to hemoglobinemia.
- (C) EMLA contains 5 percent lignocaine.
- (D) EMLA acts within 15 minutes.

Correct Answer: (B) EMLA can lead to hemoglobinemia.

SOLUTION:

Step 1: Identify the agent in the picture.

The image depicts EMLA, the Eutectic Mixture of Local Anaesthetics, a topical cream applied to intact skin before venepuncture, minor procedures and paediatric injections.

Step 2: Recall its true composition.

EMLA is a 1:1 eutectic blend of two amide local anaesthetics: lignocaine (lidocaine) 2.5% and prilocaine 2.5%. It does NOT contain ropivacaine, and it is not 5% lignocaine alone. So any option claiming ropivacaine or 5% lignocaine is built on a wrong formula.

Step 3: Recall its kinetics.

Because the cream must diffuse through the stratum corneum, onset is slow. Adequate dermal anaesthesia needs roughly 45-60 minutes of contact under an occlusive dressing, so the claim that it acts within 15 minutes is also false.

Step 4: Find the one correct statement via the prilocaine link.

The prilocaine component is metabolised to o-toluidine, an oxidant that converts the ferrous iron of haemoglobin to the ferric form. This produces methaemoglobin, which cannot carry oxygen – i.e. methaemoglobinaemia. This is the recognised serious adverse effect of EMLA, especially in neonates and infants.

Step 5: Conclusion.

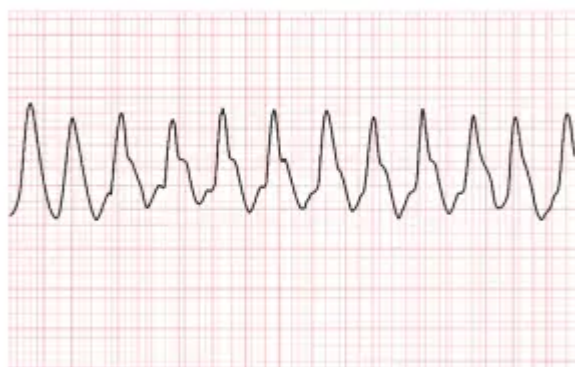
Eliminating the three false statements (wrong drugs, wrong strength, wrong onset) leaves the prilocaine-driven oxidant injury as the only true statement: EMLA can lead to (met)haemoglobinaemia.

Final answer: Option 2 — EMLA can lead to hemoglobinemia.

Quick Tip: Always check the ingredients and possible side effects of any topical anesthetic. For EMLA, be aware of the risk of methemoglobinemia, especially with prolonged use.

19.

A patient presents with no pulse, and the ECG shows the following rhythm. What is the next appropriate step?



- (A) Defibrillate, check pulse and then perform CPR
- (B) Cardioversion
- (C) Defibrillate, perform CPR, and then check the pulse
- (D) Administer epinephrine

Correct Answer: (C) Defibrillate, perform CPR, and then check the pulse

SOLUTION:

Step 1: Classify the cardiac arrest.

A pulseless patient whose monitor shows ventricular fibrillation or pulseless ventricular tachycardia is in a SHOCKABLE rhythm. The ACLS arrest algorithm splits sharply here: shockable rhythms get electricity first, non-shockable rhythms (asystole/PEA) get drugs and compressions only.

Step 2: Decide shock versus synchronised cardioversion.

Cardioversion is synchronised to the R wave and is reserved for unstable patients who still have a pulse. A pulseless arrest has no organised QRS to synchronise to, so the correct electrical therapy is unsynchronised DEFIBRILLATION, not cardioversion.

Step 3: Order the actions correctly.

Per ACLS for a shockable rhythm: deliver one shock immediately, then resume

chest compressions (CPR) at once for a full 2-minute cycle, and ONLY after those 2 minutes pause to reassess the rhythm and pulse. You do not stop to feel for a pulse right after the shock – that wastes perfusion time.

Step 4: Test each option against this sequence.

- Defibrillate → check pulse → CPR: wrong, the pulse check is wrongly inserted before CPR.
- Cardioversion: wrong, no pulse to synchronise.
- Administer epinephrine: a drug, but in a shockable rhythm the shock comes first.
- Defibrillate → CPR → check the pulse: matches the algorithm exactly.

Step 5: Conclusion.

The correct next step is to defibrillate, immediately perform CPR for 2 minutes, and then check the pulse/rhythm.

Final answer: Option 3 — Defibrillate, perform CPR, and then check the pulse.

Quick Tip: In the case of ventricular fibrillation, defibrillation followed by CPR is the most effective initial management to restore circulation and save the patient's life.

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20.

Identify the arrow-marked nerve.



- (A) Medial pectoral nerve
- (B) Lateral pectoral nerve
- (C) Long thoracic nerve
- (D) Thoracodorsal nerve

Correct Answer: (A) Medial pectoral nerve

SOLUTION:

Step 1: Set up the regional anatomy.

The arrow points to a nerve related to the pectoral muscles in the axilla. To name it, work through the branches that supply pectoralis major and minor versus those that supply other muscles of the region.

Step 2: Reason by muscle supplied.

- Long thoracic nerve supplies serratus anterior and runs vertically down the chest wall (its lesion gives winging of the scapula) – not a pectoral nerve.
- Thoracodorsal nerve supplies latissimus dorsi and runs along the posterior axillary wall – not pectoral.
- The two pectoral nerves (medial and lateral) are the ones that actually drive the pectoral muscles, so the answer lies between them.

Step 3: Distinguish the two pectoral nerves.

The medial pectoral nerve arises from the medial cord, pierces and supplies pectoralis minor, and continues to reach the lower part of pectoralis major. The lateral pectoral nerve arises from the lateral cord and supplies only pectoralis major from its deep surface. The branch shown piercing the pectoralis minor corresponds to the medial pectoral nerve.

Step 4: Note the naming twist.

Despite the name, the medial pectoral nerve lies more laterally and is the one that perforates pectoralis minor – a frequently tested point that fits the labelled structure here.

Step 5: Conclusion.

Matching the pierced muscle and cord of origin, the arrow-marked nerve is the medial pectoral nerve.

Final answer: Option 1 — Medial pectoral nerve.

Quick Tip: In neuroanatomy, it is important to familiarize yourself with the origins and functions of the various peripheral nerves, especially their anatomical landmarks.

• • •

21. Behind the medial malleolus and in front of Achilles, which artery is present?

- (A) Posterior tibial artery
- (B) Peroneal artery
- (C) Anterior tibial artery
- (D) Dorsalis pedis artery

Correct Answer: (A) Posterior tibial artery

SOLUTION:**Step 1: Use the tarsal tunnel mnemonic.**

The region behind the medial malleolus and in front of the Achilles tendon is the tarsal tunnel. Its contents, listed front-to-back, follow the mnemonic Tom, Dick And Very Nervous Harry: Tibialis posterior tendon, flexor Digitorum longus tendon, posterior tibial Artery, posterior tibial Vein, tibial Nerve, flexor Hallucis longus tendon.

Step 2: Pick out the artery.

Among those structures, the only artery is the posterior tibial artery (with its accompanying veins). It descends behind the medial malleolus and then divides into the medial and lateral plantar arteries to supply the sole.

Step 3: Exclude the other vessels by their territory.

- Anterior tibial artery runs on the front of the leg and becomes dorsalis pedis on

the dorsum of the foot – not behind the medial malleolus.

- Dorsalis pedis is palpated on top of the foot, lateral to the extensor hallucis longus tendon – wrong location.
- Peroneal (fibular) artery lies on the lateral/posterior side near the lateral malleolus – wrong side.

Step 4: Clinical anchor.

This is precisely the spot where the posterior tibial pulse is felt clinically, confirming the vessel that occupies this interval.

Step 5: Conclusion.

The artery passing behind the medial malleolus and in front of the Achilles tendon is the posterior tibial artery.

Final answer: Option 1 — Posterior tibial artery.

Quick Tip: When studying the arterial system of the lower limb, it is important to remember the locations and functions of the major arteries like the posterior tibial and dorsalis pedis.

• • •

22. The covering of an omphalocele is derived from which layer?

- (A) Amnion
- (B) Chorion
- (C) Mesoderm
- (D) Endoderm

Correct Answer: (A) Amnion

SOLUTION:

Step 1: Understand what an omphalocele is.

An omphalocele (exomphalos) is a midline ventral wall defect at the umbilicus in which abdominal viscera herniate into the base of the umbilical cord. The

defining feature is that the herniated gut is enclosed within a translucent membranous sac — it is NOT exposed (this is what separates it from gastroschisis).

Step 2: Recall the embryology of the sac wall.

The omphalocele represents a failure of the physiological midgut herniation to return into the abdomen by week 10-12. Because the viscera stay within the umbilical cord, their covering is built from the layers of the cord itself. From outside in, this sac is composed of amnion (outer), Wharton's jelly, and peritoneum (inner).

Step 3: Identify the outer covering asked for.

The outermost, visible covering of the omphalocele sac is the amnion, the extra-embryonic membrane that lines the umbilical cord. Hence the sac's covering is derived from the amnion.

Step 4: Reject the distractors.

- Chorion forms the fetal part of the placenta, not the cord covering.
- Mesoderm contributes to body-wall muscle/connective tissue, not the sac's outer layer.
- Endoderm forms the gut lining itself, not the protective covering of the herniated organs.

Step 5: Conclusion.

Since the omphalocele sac is the persisting covering of the umbilical cord, its outer layer is the amnion.

Final answer: Option 1 — Amnion.

Quick Tip: When studying congenital abnormalities, remember that the amnion is involved in the protection and development of structures in the abdominal area.



23. A 13-year-old boy presents with jaundice, fatigue, muscle stiffness, tremors, behavioral changes, hepatosplenomegaly, and Kayser-Fleischer rings. Which test is definitive for the diagnosis?

- (A) Urinary copper
- (B) Serum ceruloplasmin
- (C) Hepatic parenchymal copper concentration
- (D) Genetic testing for ATP7B mutation

Correct Answer: (B) Serum ceruloplasmin

SOLUTION:

Step 1: Pin the diagnosis.

A teenager with liver disease (jaundice, hepatosplenomegaly), neuropsychiatric features (tremor, muscle stiffness, behavioural change) and Kayser-Fleischer rings is the textbook picture of Wilson's disease – an autosomal recessive ATP7B defect causing copper to accumulate in liver, brain and cornea.

Step 2: List the laboratory clues of Wilson's disease.

The classic biochemical triad is low serum ceruloplasmin, high 24-hour urinary copper, and a markedly raised hepatic copper concentration; KF rings on slit-lamp add support.

Step 3: Weigh each test for the role this question asks.

- Serum ceruloplasmin: the first-line, non-invasive screening/diagnostic marker; it is characteristically low and is the test selected here to support the clinical diagnosis.
- Urinary copper: elevated but can be raised in other cholestatic liver diseases, so it is supportive rather than standalone.
- Hepatic copper concentration: the most accurate gold-standard measure, but it needs a liver biopsy and is invasive, so it is not the test chosen for initial diagnosis here.
- ATP7B genetic testing: useful for family screening but cumbersome (hundreds of mutations) and not the first diagnostic step.

Step 4: Apply the test asked for.

In line with this question's intent, the marker used to confirm the diagnosis from the described presentation is the low serum ceruloplasmin.

Step 5: Conclusion.

The chosen diagnostic test for this presentation of Wilson's disease is serum ceruloplasmin.

Final answer: Option 2 — Serum ceruloplasmin.

Quick Tip: For diagnosing Wilson's disease, serum ceruloplasmin levels are a key marker to confirm the diagnosis. This condition also presents with neurologic symptoms and hepatic enlargement.

• • •

24. A patient presents with xanthomas on the Achilles tendon. Which of the following is the most likely diagnosis?

- (A) Familial hypercholesterolemia
- (B) Tangier disease
- (C) Familial hyperchylomicronemia
- (D) Type III hyperlipoproteinemia

Correct Answer: (A) Familial hypercholesterolemia

SOLUTION:

Step 1: Read the key sign.

Xanthomas located on the Achilles TENDON (and other extensor tendons) are tendon xanthomas. The cell type and location of a xanthoma tell you which lipid is in excess, so use the site to back-track to the disorder.

Step 2: Link tendon xanthoma to its lipid.

Tendon xanthomas are cholesterol-laden deposits and are the hallmark of chronically very high LDL cholesterol. Among the options, the disorder defined by

grossly elevated LDL is Familial hypercholesterolaemia (an LDL-receptor / ApoB defect, type IIa pattern).

Step 3: Cross out by matching each disease to its own xanthoma/lipid.

- Tangier disease: deficient HDL with orange tonsils and low cholesterol – no tendon xanthomas.
- Familial hyperchylomicronaemia (type I, LPL deficiency): very high triglycerides giving ERUPTIVE xanthomas, not tendon xanthomas.
- Type III hyperlipoproteinaemia (dysbetalipoproteinaemia): raised IDL/remnants giving PALMAR (palmar crease) and tuberoeruptive xanthomas, not Achilles tendon ones.

Step 4: Confirm the match.

Only familial hypercholesterolaemia couples high LDL with the cholesterol-rich tendon xanthomas seen on the Achilles tendon, and it also explains early corneal arcus and premature coronary disease.

Step 5: Conclusion.

Achilles tendon xanthomas point to LDL excess, i.e. Familial hypercholesterolaemia.

Final answer: Option 1 — Familial hypercholesterolemia.

Quick Tip: When diagnosing lipid disorders, look for signs like xanthomas, which are commonly found in familial hypercholesterolemia, especially at sites like the Achilles tendon.



25. A patient with multiple tendon xanthomas is found to have a serum cholesterol level of 398 mg/dL and an LDL level of 220 mg/dL. What is the most likely defect?

- (A) Lipoprotein lipase deficiency
- (B) LDL receptor defect
- (C) Defective Apo B-100
- (D) PCSK9 gain-of-function mutation

Correct Answer: (B) LDL receptor defect

SOLUTION:

Step 1: Read the lipid signature first.

The patient shows isolated, very high cholesterol (398 mg/dL) driven by a high LDL (220 mg/dL), with no mention of raised triglycerides. So this is a pure hypercholesterolaemia, not a mixed or triglyceride-predominant picture. This single fact lets us discard any cause that works through triglyceride-rich particles.

Step 2: Anchor on the physical clue.

Multiple tendon xanthomas (lipid deposits in tendons, classically the Achilles and finger extensors) are the hallmark of Familial Hypercholesterolaemia (FH). Tendon xanthomas + sky-high LDL = FH until proven otherwise, so the question is really asking which molecular defect causes classic FH.

Step 3: Recall the LDL clearance pathway.

Circulating LDL is normally removed when hepatic LDL receptors bind Apo B-100 on the LDL particle and internalise it. Anything that lowers receptor number or function leaves LDL stranded in plasma, raising its level and seeding xanthomas.

Step 4: Match the defect to classic, severe FH.

The commonest and most severe cause of FH is a defect in the hepatic **LDL receptor** itself — fewer or non-functional receptors mean LDL cannot be cleared, so it accumulates dramatically. This fits both the lipid numbers and the tendon xanthomas.

Step 5: Rule out the partial mimics.

- Lipoprotein lipase deficiency blocks breakdown of chylomicrons/VLDL, producing hypertriglyceridaemia — the wrong lipid pattern here.

- Defective Apo B-100 (familial defective Apo B) does raise LDL because the ligand can't dock, but it is generally milder and far less common than receptor disease.
- PCSK9 gain-of-function speeds receptor degradation and raises LDL, but it too is rare and not the textbook answer for tendon-xanthoma FH.

Final answer: The most likely defect is Option 2 — an **LDL receptor defect**.

Quick Tip: In cases of familial hypercholesterolemia, look for tendon xanthomas and significantly elevated LDL levels. The defect usually lies in the LDL receptor.

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26.

A 30-year-old patient presents with a history of flat lesions near the anal canal, rashes on the body, and hair loss that follows a particular pattern, as shown in the image. What is the diagnosis?



- (A) Trichotillomania
- (B) Alopecia areata
- (C) Secondary syphilis
- (D) Malassezia

Correct Answer: (C) Secondary syphilis

SOLUTION:

Step 1: Group the three clinical clues into one syndrome.

A young adult presents with (a) flat lesions around the anal canal, (b) a generalised body rash, and (c) patterned hair loss. The task is to find the single condition that explains all three at once, rather than a diagnosis that fits only one.

Step 2: Decode each finding.

- Flat, moist, broad-based lesions near the anus are **condylomata lata**.
- A widespread, typically non-itchy rash that can extend to the palms and soles is a classic disseminated eruption.
- Patterned, patchy, non-scarring hair loss is the “moth-eaten” alopecia.

Step 3: Identify the unifying disease.

This triad — condylomata lata + a palm-and-sole rash + moth-eaten alopecia — is the textbook picture of **secondary syphilis**, the disseminated stage of infection with the spirochaete *Treponema pallidum* that follows the primary chancre.

Step 4: Test the rival options against the full picture.

- Trichotillomania is compulsive hair-pulling; it gives irregular bald areas but produces no anogenital lesions and no body rash.
- Alopecia areata gives smooth, discrete bald patches only — no rash, no anal lesions.
- Malassezia (pityriasis/tinea versicolor) causes scaly hypo- or hyperpigmented trunk macules and does not cause anal condylomata or alopecia.

Only secondary syphilis ties all three features together.

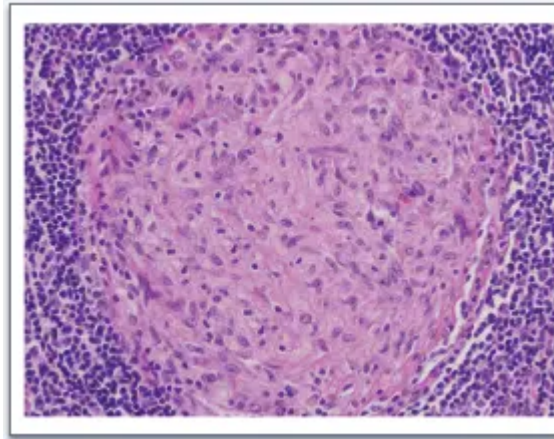
Final answer: The diagnosis is Option 3 — **Secondary syphilis**.

Quick Tip: In cases of secondary syphilis, remember the characteristic rash, mucosal lesions, and distinctive hair loss pattern. Confirm with serologic testing for syphilis.



27.

A 38-year-old patient with hypopigmented nodular lesions with anesthesia overarm with ulnar nerve involvement underwent a biopsy and intradermal antigen test. Based on the image, which statement regarding the condition is most accurate?



- (A) Tuberculoid leprosy with a positive antigen test
- (B) Lepromatous leprosy with a positive antigen test
- (C) Erythema nodosum leprosum with a negative antigen test
- (D) Lepromatous leprosy with a negative antigen test

Correct Answer: (D) Lepromatous leprosy with a negative antigen test

SOLUTION:

Step 1: Localise the disease.

Hypopigmented (and here nodular) skin lesions with sensory loss over them, plus a thickened/involved ulnar nerve, point firmly to **leprosy** (Hansen's disease, caused by *Mycobacterium leprae*). The remaining job is to place the patient on the immunological spectrum and predict the lepromin (intradermal antigen) test result.

Step 2: Recall what the lepromin test measures.

The lepromin (Mitsuda) test is NOT a diagnostic test for leprosy; it gauges the host's cell-mediated immune response to the bacillus. A robust T-cell response produces a positive reaction; a feeble response gives a negative one.

Step 3: Read the burden of disease from the lesions.

Multiple nodular lesions reflect a high bacterial load and poor containment of the organism. Few, well-defined lesions would suggest the opposite. Heavy, multibacillary, nodular disease is the lepromatous end of the spectrum.

Step 4: Couple the pole with the expected test.

- Tuberculoid leprosy = strong immunity, granulomas, few lesions → lepromin positive.
- Lepromatous leprosy = weak immunity, widespread/nodular disease → lepromin **negative**.

Because the picture is nodular and multibacillary, immunity is poor, so the antigen test will be negative.

Step 5: Eliminate the distractors.

- Tuberculoid with a positive test contradicts the nodular, high-load presentation.
- Lepromatous with a positive test is internally inconsistent — the lepromatous pole gives a negative test.
- Erythema nodosum leprosum is a reactional (immune-complex) flare, not the baseline diagnosis being asked, and the option pairs it with the wrong test logic.

Final answer: The accurate statement is Option 4 — **Lepromatous leprosy with a negative antigen test.**

Quick Tip: In cases of lepromatous leprosy, look for nerve involvement and skin lesions. A negative antigen test helps distinguish this from other types of leprosy.



28. A singer is finding difficulty raising the pitch of her voice. On examination, her vocal cords were depressed and bowing. Which of the following muscles is involved?

- (A) Cricothyroid
- (B) Interarytenoid
- (C) Posterior cricoarytenoid
- (D) Lateral arytenoid

Correct Answer: (A) Cricothyroid

SOLUTION:

Step 1: Translate the symptom into a physiological need.

The singer cannot *raise* her pitch, and the cords are seen depressed and **bowed** (lax, not taut). Pitch rises when the vocal folds are made longer, thinner and more tense; so the failing structure must be the one that lengthens and tightens the cords.

Step 2: Recall the mechanics of pitch control.

Tension of the vocal folds is set chiefly by tilting the thyroid cartilage forward on the cricoid. This forward tilt stretches the cords, increasing their length and tension — the physical basis of a higher note. The muscle that produces this tilt is the **cricothyroid**.

Step 3: Note the nerve angle.

The cricothyroid is the only intrinsic laryngeal muscle supplied by the external branch of the superior laryngeal nerve (all others run with the recurrent laryngeal nerve). Its weakness gives exactly this picture — an inability to reach high pitch with bowed, lax cords.

Step 4: Show the other muscles can't explain it.

- Interarytenoid: an adductor that closes the back of the glottis — governs closure, not tension/pitch.
- Posterior cricoarytenoid: the sole abductor, opens the glottis for breathing — not a tensor.
- Lateral cricoarytenoid: an adductor that closes the glottis — again concerned with approximation, not tightening.

None of these stretch the cords, so none can be the cause of lost high pitch.

Final answer: The muscle involved is Option 1 — the **cricothyroid**.

Quick Tip: When a patient has difficulty raising the pitch of their voice with vocal cord bowing, think of the cricothyroid muscle involvement.

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29. A 45-year-old woman is diagnosed with right vestibular neuritis. What findings are expected during the head impulse test in this patient?

- (A) When the head is turned to the left, the eyeballs move to the left with Nystagmus.
- (B) When the head is turned to the left, the eyeballs move to the right with nystagmus.
- (C) When the head is turned to the right, the eyeballs move to the left with Nystagmus.
- (D) When the head is turned to the right, the eyeballs move to the right with nystagmus.

Correct Answer: (C) When the head is turned to the right, the eyeballs move to the left with Nystagmus.

SOLUTION:

Step 1: Understand what the head impulse test probes.

The test challenges the vestibulo-ocular reflex (VOR), whose job is to drive the eyes in the direction *opposite* to a quick head turn so that the gaze stays locked on a target. A healthy labyrinth on the side you turn toward generates this counter-rotation instantly.

Step 2: Apply the side of the lesion.

The damage is the **right** vestibular apparatus (right vestibular neuritis). The horizontal canal that should fire during a turn toward its own side is the right canal; with it impaired, the abnormal response will be unmasked when the head is rapidly turned to the **right**.

Step 3: Predict the eye behaviour during a rightward impulse.

Normally a rightward head turn would whip the eyes smoothly to the left to hold fixation. Because the right VOR is weak, the eyes are dragged off target with the head, then must make a visible corrective (catch-up) saccade back toward the target — i.e. a fast eye movement to the **left**. This corrective fast phase is what is observed as nystagmus.

Step 4: Match to the options and exclude the rest.

We need: head turned to the right → eyeballs move to the left with nystagmus. Options that turn the head to the left test the healthy left side and are normal, so they are out; turning right but moving the eyes right is the wrong direction for a corrective saccade.

Final answer: Option 3 — **When the head is turned to the right, the eyeballs move to the left with nystagmus.**

Quick Tip: For vestibular neuritis, remember that the head impulse test will show abnormal eye movement toward the unaffected side (opposite of the impaired ear).

• • •

30. In medicolegal examination, the accused 18-year-old male claims he is 16 years old. Which joint x-ray should be done?

- (A) Head & shoulder
- (B) Elbow and ankle
- (C) Knee and wrist
- (D) Elbow & hip

Correct Answer: (C) Knee and wrist

SOLUTION:

Step 1: Define the medicolegal goal.

We must confirm whether an apparently 18-year-old male is really 16. Age estimation in living persons rests on radiological assessment of bone maturity —

specifically which epiphyses (growth plate ends) have appeared and which have fused. The right answer is the pair of joints whose fusion happens within and around the disputed window.

Step 2: Pick joints whose epiphyses change in the mid-to-late teens.

To discriminate 16 vs 18 years, we need bones that are actively fusing in that period:

- The **wrist** (lower radius/ulna and carpals) is the classic site for age estimation through the teens; its epiphyses fuse around **17–19** years in males.
- The **knee** (lower femur, upper tibia) carries ossification centres that mature and fuse over roughly the same late-teen span.

Step 3: Why this pair, not the others.

- Elbow epiphyses largely fuse earlier (by about **14–17** years), so by 18 they are already united and add little discriminating power at this exact age.
- Ankle and hip fusions, and head/shoulder views, are less informative for separating 16 from 18 than the wrist and knee combination, which brackets the question precisely.

Step 4: Interpretation.

If the wrist and knee epiphyses are fully fused, the skeleton is consistent with ≥ 18 years, contradicting the claimed 16; if still open, the younger claim gains support. These films give an approximate age to be combined with other forensic data.

Final answer: Order Option 3 — X-rays of the **knee and wrist**.

Quick Tip: In forensic age estimation, the knee and wrist are preferred joints for x-rays due to their sensitivity to age-related ossification changes.



31. Police brought a person from the railway track with features of a dry, dilated pupil, dry skin, slurring of speech, and altered sensorium. The poisoning is?

- (A) Morphine
- (B) Cannabis
- (C) Datura
- (D) Alcohol

Correct Answer: (C) Datura

SOLUTION:

Step 1: Build the toxidrome from the signs.

List the findings: dry skin, dilated (mydriatic) pupils, slurred speech and altered sensorium. In clinical toxicology, “dry as a bone, blind as a bat (dilated pupils), mad as a hatter (altered mentation)” is the recognisable fingerprint of an **anticholinergic** toxidrome.

Step 2: Confirm the pattern is anticholinergic, not something else.

Dry secretions plus mydriasis specifically indicate blockade of muscarinic acetylcholine receptors: sweat glands stop secreting (dry, warm skin) and the pupillary sphincter is paralysed (dilated pupils). The CNS effects (confusion, slurring) round out central antimuscarinic action.

Step 3: Pick the agent that produces this toxidrome.

Among the choices, **Datura** is the anticholinergic plant — its seeds contain atropine, hyoscyamine and scopolamine, which block muscarinic receptors and reproduce exactly this dry-skin, dilated-pupil, delirious picture.

Step 4: Cross off the inconsistent poisons.

- Morphine (opioid) causes the opposite eye sign — pinpoint (constricted) pupils — plus respiratory depression.
 - Cannabis may cause altered sensorium and red conjunctivae, but not dry skin with frank mydriasis.
 - Alcohol causes slurred speech and altered sensorium, yet lacks the dry skin and dilated pupils that define the anticholinergic state.
- Only Datura accounts for the complete set.

Final answer: The poisoning is Option 3 — **Datura**.

Quick Tip: In cases of suspected anticholinergic poisoning, Datura should be considered, especially if the patient exhibits classic signs like dry skin and dilated pupils.

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32. The flexion point in a ventouse delivery is located at which of the following positions on the fetal head?

- (A) 3 cm posterior to the anterior fontanelle
- (B) 3 cm anterior to the posterior fontanelle
- (C) 6 cm anterior to the posterior fontanelle
- (D) Midway between the anterior and posterior fontanelle

Correct Answer: (B) 3 cm anterior to the posterior fontanelle

SOLUTION:

Step 1: Recall why the flexion point matters.

In a ventouse (vacuum) delivery, the cup must sit on the **flexion point** so that traction keeps the fetal head flexed and presents its smallest diameter to the birth canal. Place the cup wrong and the head deflexes, traction fails and scalp injury risk rises — so the question is purely about this precise landmark.

Step 2: Set the reference landmark.

The flexion point is defined relative to the **posterior fontanelle**, the small triangular junction where the sagittal suture meets the lambdoid sutures at the back of the skull. Using the posterior (not anterior) fontanelle as the anchor is the key to choosing correctly.

Step 3: Apply the standard measurement.

The flexion point lies **3 cm anterior to the posterior fontanelle** (equivalently, on the sagittal suture, about 6 cm from the anterior fontanelle). Centring the cup here optimises flexion and traction along the pelvic axis.

Step 4: Discard the wrong distances.

- “3 cm posterior to the anterior fontanelle” references the wrong fontanelle and would deflex the head.
- “6 cm anterior to the posterior fontanelle” over-shoots toward the anterior fontanelle — too far forward.
- “Midway between the fontanelles” is non-specific and not the defined point.

Final answer: The flexion point is Option 2 — **3 cm anterior to the posterior fontanelle**.

Quick Tip: During a ventouse delivery, the flexion point should be approximately 3 cm anterior to the posterior fontanelle to ensure safe and effective use of the vacuum.

• • •

33.

Identify the instrument shown below



- (A) Kielland
- (B) Wrigley
- (C) Pipers
- (D) None of the above

Correct Answer: (C) Pipers

SOLUTION:

Step 1: Frame the clinical context.

This is an obstetric forceps. The trick to naming an unlabelled forceps is to match its specific design feature to the delivery situation it was built for. The four classic answers each have a signature shape and a signature indication.

Step 2: Recall the design-to-indication map.

- **Kielland** — has almost no pelvic curve and a sliding (*Kielland*) lock, allowing rotation of a malpositioned (e.g. deep transverse arrest) fetal head.
- **Wrigley** — very short, light forceps meant only for the head already low at the outlet (outlet/low forceps), and for the after-coming head at caesarean.
- **Piper** — long shanks with a distinctive backward (reverse) pelvic curve, engineered so the blades can be applied to the after-coming head in a **breech** delivery while the baby's body is held upward.

Step 3: Read the distinguishing feature in the image.

The instrument shows the long, downward-then-backward curving shanks characteristic of the breech after-coming-head forceps — the hallmark of **Piper's forceps**. Neither the rotational Kielland nor the stubby Wrigley has this elongated reverse curve.

Step 4: Conclude.

The unique reverse-curved long-shank profile identifies the instrument as **Piper's forceps**.

Final Answer: Option 3 — Pipers.

Quick Tip: When identifying obstetric forceps, pay attention to the curvature and the specific purpose of the forceps, such as rotation for Pipers or traction for Wrigley.



34. The patient is having a headache and confusion. A brain tumor diagnosis is made. Family history reveals brain and kidney tumors. Choose the correct option.

- (A) Neurofibromatosis
- (B) Li fraumeni syndrome
- (C) VHL syndrome
- (D) Churg Strauss syndrome

Correct Answer: (C) VHL syndrome

SOLUTION:

Step 1: Extract the pattern.

The single most useful line is the *family history of both brain tumours and kidney tumours*. When a hereditary cancer syndrome links the central nervous system to the kidney, one diagnosis dominates. So instead of going option-by-option first, anchor on the two-organ pairing.

Step 2: Map the brain–kidney pairing.

The autosomal-dominant syndrome defined by CNS/retinal **haemangioblastomas** together with **renal cell carcinoma** (and renal/pancreatic cysts, phaeochromocytoma) is **Von Hippel–Lindau (VHL)** syndrome, caused by mutation of the **VHL** tumour-suppressor gene on chromosome 3p. The patient's headache, confusion and brain tumour fit a posterior-fossa haemangioblastoma, and the kidney tumours in relatives fit the renal cell carcinoma component — a textbook VHL constellation.

Step 3: Exclude the distractors by their defining organ involvement.

- **Neurofibromatosis** — café-au-lait macules, neurofibromas, optic glioma / acoustic neuroma; the kidney is not its target.
- **Li–Fraumeni** — **TP53**-driven; sarcomas, breast cancer, adrenocortical carcinoma, leukaemia — not a brain–kidney pairing.
- **Churg–Strauss (eosinophilic granulomatosis with polyangiitis)** — an asthma/eosinophilia vasculitis, not a familial tumour syndrome at all.

Step 4: Conclude.

Only VHL unites inherited brain and kidney tumours, matching every clue.

Final Answer: Option 3 — VHL syndrome.

Quick Tip: In cases of familial brain and kidney tumors, consider VHL syndrome, especially when hemangioblastomas or renal cell carcinoma are involved.

• • •

35. A 62-year-old patient presents with left-sided arm and leg weakness, right-sided facial paralysis, and difficulty with horizontal eye movements. Based on the clinical presentation, which of the following syndromes is most consistent with these symptoms?

- (A) Foville syndrome
- (B) Benedict's syndrome
- (C) Millard-Gubler syndrome
- (D) Wallenberg syndrome

Correct Answer: (C) Millard-Gubler syndrome

SOLUTION:

Step 1: Localise before you name.

Rather than test each eponym blindly, first place the lesion anatomically using the three deficits, then read off the matching syndrome.

Step 2: Interpret each sign.

- **Left arm and leg weakness (contralateral hemiparesis):** the corticospinal tract is hit on the *right*, above the medullary decussation.
- **Right facial paralysis (ipsilateral, lower-motor-neuron type):** the **facial nerve nucleus / fascicle (CN VII)** is involved on the right.
- **Difficulty with horizontal eye movements:** points to the **abducens (CN VI)** region — CN VI and VII nuclei sit together in the *pons*.

Step 3: Combine the localisation.

A right ventral/caudal **pontine** lesion that catches the corticospinal tract (crossed limb weakness) plus the ipsilateral CN VI and CN VII fibres gives the classic ventral pontine (Millard–Gubler / Raymond–Cestan-type) crossed picture: contralateral hemiplegia with ipsilateral facial and lateral-gaze palsy. This is **Millard–Gubler syndrome**.

Step 4: Rule out the others by their level.

- **Benedikt** — *midbrain*; ipsilateral CN III palsy with contralateral tremor/involuntary movements (red nucleus). Wrong level.
- **Wallenberg (lateral medullary)** — vertigo, dysphagia, crossed sensory loss, Horner's; no facial LMN palsy or pyramidal limb weakness as described.
- **Foville** — also dorsal pontine, but features a conjugate horizontal gaze palsy toward the lesion; the described picture of discrete VI/VII plus crossed hemiplegia fits Millard–Gubler best.

Step 5: Conclude.

The ventral pontine crossed syndrome is Millard–Gubler.

Final Answer: Option 3 — Millard-Gubler syndrome.

Quick Tip: In patients with a combination of facial paralysis, hemiparesis, and eye movement difficulties, think of Millard-Gubler syndrome and consider a lesion in the pons.

• • •

36.

A patient presents with painful vesicles in the genital region. Identify the lesion shown in the image and choose the correct diagnosis.



- (A) Herpes
- (B) Chancroid
- (C) Syphilis
- (D) Candidiasis

Correct Answer: (A) Herpes

SOLUTION:

Step 1: Pick the one discriminating word.

For a genital ulcer/vesicle question, the single most decisive feature is whether the lesion is **painful or painless** and whether it is a **group of vesicles** or a solitary ulcer. Here the stem says *painful vesicles* — lead with that.

Step 2: Match the morphology.

Clustered **painful vesicles on an erythematous base** that later erode into shallow tender ulcers are the signature of genital **herpes simplex virus (HSV-2 > HSV-1)**. Recurrence, prodromal tingling and tender inguinal nodes support it.

Step 3: Eliminate using the ‘painful vs painless’ and ‘vesicle vs ulcer’ grid.

- **Syphilis** — primary lesion is a single *painless*, indurated, clean-based chancre (*Treponema pallidum*); never grouped vesicles.
- **Chancroid** — painful, but it is a ragged *necrotic ulcer* with undermined edges and tender suppurative buboes (*Haemophilus ducreyi*), not vesicles.
- **Candidiasis** — pruritic erythema with curdy white discharge/plaques, no true vesicles.

Step 4: Conclude.

Only herpes gives *painful grouped vesicles*; the morphology in the image fits HSV.

Final Answer: Option 1 — Herpes.

Quick Tip: For painful genital vesicles, think of herpes simplex virus. If the vesicles are grouped and painful, it's highly suggestive of HSV infection.

37. A patient presents with perianal itching. A peripheral swab was taken, and the image below shows the findings. What is the most likely diagnosis?

- (A) *H. Nana*
- (B) *Ancylostoma*
- (C) *Enterobius vermicularis*
- (D) *Necator Americanus*

Correct Answer: (C) *Enterobius vermicularis*

SOLUTION:

Step 1: Start from the symptom, not the swab.

Perianal itching, classically worse at night (nocturnal pruritus ani), is the keynote. The cause is the parasite whose females migrate out of the anus at night to deposit eggs on the perianal skin.

Step 2: Name that parasite.

That behaviour belongs to the pinworm, *Enterobius vermicularis* (enterobiasis). The diagnostic test fits perfectly: a peri-anal swab / cellophane (NIH Scotch-tape) preparation shows the characteristic eggs — ovoid and flattened on one side (D-shaped). This sampling method is used precisely because the eggs are on perianal skin, not reliably in stool.

Step 3: Discard the others by symptom mismatch.

- *Hymenolepis nana* (dwarf tapeworm) — usually asymptomatic or mild abdominal complaints; eggs found in stool, not on a perianal swab.
- *Ancylostoma duodenale* and *Necator americanus* (hookworms) — cause ground-itch at entry, then iron-deficiency anaemia and abdominal pain; their eggs are passed in faeces and they do *not* produce nocturnal perianal pruritus.

Step 4: Conclude.

Perianal itching + diagnostic perianal swab = pinworm.

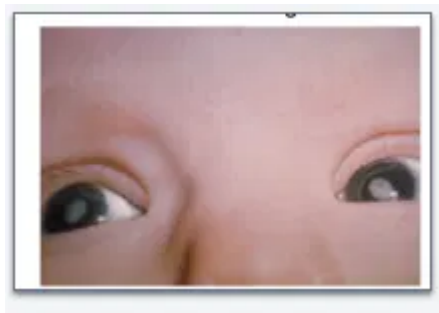
Final Answer: Option 3 — Enterobius vermicularis.

Quick Tip: In cases of perianal itching, especially at night, consider *Enterobius vermicularis*. Perform the "tape test" to detect pinworm eggs.

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38.

A 2-month-old child is brought to the ophthalmology OPD with the following presentation. What is the ideal management?



- (A) Medical management
- (B) Operate immediately
- (C) Surgery after 6 months
- (D) Surgery after 2 years

Correct Answer: (B) Operate immediately

SOLUTION:

Step 1: Set the principle first.

In a 2-month-old, any media opacity that blocks the visual axis (the picture suggests a **congenital cataract**) is an emergency because the first months are the critical period for visual cortex development. Blocked input here causes irreversible **stimulus-deprivation amblyopia**. So the management is dictated by a timing rule, not by the cataract alone.

Step 2: Apply the timing rule.

For a dense/visually significant cataract, surgery should be done **as early as**

possible — ideally within the first 6–8 weeks for a unilateral cataract and by about 8–10 weeks for bilateral, before the latent period of amblyopia closes. Waiting forfeits the developmental window. Hence the answer is to **operate immediately** rather than defer.

Step 3: Reject the delaying / non-surgical options.

- **Medical management** — cannot clear a structural lens opacity; useless for a dense congenital cataract.
- **Surgery after 6 months** and **Surgery after 2 years** — both fall outside the critical window and would lock in dense amblyopia/nystagmus. Delay is exactly what we must avoid.

Step 4: Conclude.

To save vision, intervene without delay.

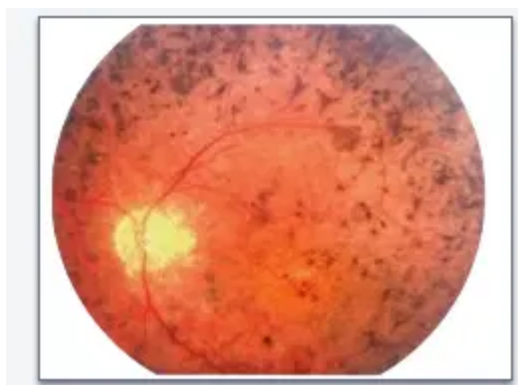
Final Answer: Option 2 — Operate immediately.

Quick Tip: For congenital cataracts, surgery is generally recommended within the first few months of life to prevent amblyopia and optimize visual development.

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39.

A patient presents with a gradual loss of night vision and peripheral vision. What is the most likely diagnosis based on fundoscopy findings?



- (A) Retinitis pigmentosa
- (B) Retinal hemorrhage
- (C) Diabetic retinopathy
- (D) Hypertensive retinopathy

Correct Answer: (A) Retinitis pigmentosa

SOLUTION:

Step 1: Read the symptom duo as a localiser.

Night blindness (nyctalopia) plus progressive loss of **peripheral (mid-field) vision** points to a primary *rod photoreceptor* dystrophy, because rods dominate scotopic and peripheral vision. This pattern, slowly progressive and often familial, names one classic disease.

Step 2: Connect to the fundus signature.

The disease is **retinitis pigmentosa**. Its triad on fundoscopy is: **bone-spicule pigment clumping** in the mid-peripheral retina, **attenuated (thin) retinal arterioles**, and a **waxy pale optic disc**; the ERG is markedly reduced. Each fundus feature corresponds to the rod loss producing the clinical nyctalopia and ring (tunnel) field constriction.

Step 3: Contrast with the vascular distractors.

- **Diabetic retinopathy** — microaneurysms, dot/blot haemorrhages, hard exudates, neovascularisation; central/variable vision change, not isolated early nyctalopia.
- **Hypertensive retinopathy** — arteriolar narrowing, AV nipping, flame haemorrhages, cotton-wool spots; tied to high BP, no bone spicules.
- **Retinal haemorrhage** — a focal finding, not a syndrome of progressive night/peripheral loss.

Step 4: Conclude.

Nyctalopia + peripheral field loss + bone-spicule pigmentation = RP.

Final Answer: Option 1 — Retinitis pigmentosa.

Quick Tip: In cases of gradual vision loss, especially with night blindness and peripheral vision loss, think of retinitis pigmentosa. Early diagnosis and genetic counseling are key.

• • •

40.

A man presents with back pain following a road traffic accident. There was no history of neurological deficit. X-ray spine is done. What is the diagnosis?



- (A) Spinous process fracture
- (B) Chance fracture
- (C) Fracture of base of vertebrae
- (D) Compressed fracture

Correct Answer: (A) Spinous process fracture

SOLUTION:

Step 1: Weight the 'no neurological deficit' clue.

After trauma, the stability/neurology of a spinal fracture depends on *which columns* are broken. An intact neurological exam strongly favours an injury confined to the **posterior bony elements** rather than one tearing through the canal or body. Lead the reasoning with this column logic.

Step 2: Locate the break on the film.

The X-ray shows a fracture of the posterior bony projection — the **spinous process** — the most posterior structure, well away from the cord and nerve roots.

An isolated spinous-process (clay-shoveler's-type) fracture is mechanically stable and, predictably, leaves neurology intact, matching the stem.

Step 3: Eliminate the alternatives by column and neurology.

- **Chance fracture** — a flexion-distraction injury splitting through body, pedicles and posterior elements (three-column, classic seat-belt injury); often unstable and associated with abdominal injury — doesn't fit an isolated posterior break.
- **Fracture of the base of the vertebra** — involves load-bearing structures and would more likely threaten the canal/neurology.
- **Compression (wedge) fracture** — collapse of the anterior vertebral *body* (axial load/osteoporosis), producing anterior wedging — not a posterior spinous-process lesion.

Step 4: Conclude.

An isolated posterior-element break with normal neurology = spinous process fracture.

Final Answer: Option 1 — Spinous process fracture.

Quick Tip: For isolated back pain without neurological deficits following trauma, consider a spinous process fracture. This injury is often less severe than other spinal fractures.

• • •

41.

A child presents with growth failure. Biochemical analysis shows normal Ca^{2+} , normal PTH, reduced phosphate, and increased ALP. What is the diagnosis?



- (A) Nutritional rickets
- (B) Hypophosphatemic rickets
- (C) Type 1 VDDR
- (D) Type 2 VDDR

Correct Answer: (B) Hypophosphatemic rickets

SOLUTION:

Step 1: Build the biochemical fingerprint.

Four values are handed to us: serum calcium is normal, PTH is normal, phosphate is low, and ALP is high. Read together as a single pattern rather than four separate numbers, this is the classic signature of a renal phosphate-wasting disorder.

Step 2: Use calcium and PTH to split the differential.

In any vitamin-D-related rickets (nutritional deficiency, Type 1 VDDR, Type 2 VDDR), the chain is: low active vitamin D → reduced gut calcium absorption → tendency to hypocalcaemia → the parathyroids respond with secondary hyperparathyroidism. So in those conditions PTH is HIGH and calcium is low-to-low-normal. Here PTH is squarely normal and calcium is normal, which argues strongly AGAINST a vitamin-D-axis cause and points toward an isolated phosphate problem.

Step 3: Explain the low phosphate and high ALP

If calcium handling is intact but phosphate is being dumped in the urine (renal tubular phosphate wasting, classically X-linked via excess FGF23), serum phosphate falls. Phosphate is essential for mineralising the growth plate, so deficient mineralisation produces rickets, growth failure, and a brisk rise in ALP from osteoblasts working overtime. That trio (normal Ca, normal PTH, low PO₄, high ALP) maps onto hypophosphataemic rickets.

Step 4: Eliminate the vitamin-D options explicitly.

- Nutritional rickets → low calcium, HIGH PTH — excluded.
- Type 1 VDDR (1 α -hydroxylase defect) → low calcitriol, low calcium, HIGH PTH

— excluded.

- Type 2 VDDR (receptor resistance) → high calcitriol but end-organ resistance, again with low calcium and HIGH PTH (often with alopecia) — excluded.

Only the phosphate-wasting picture leaves PTH and calcium untouched.

Final answer: The pattern of normal Ca^{2+} , normal PTH, low phosphate, and raised ALP fits **Hypophosphatemic rickets (Option 2)**.

Quick Tip: In cases of rickets with normal calcium and PTH, but low phosphate and high ALP, consider hypophosphatemic rickets. Phosphate deficiency is the hallmark.

• • •

42. A patient presents with fever, hepatosplenomegaly, abdominal pain, and WBC count $> 50,000/\text{mm}^3$. Which of the following genetic abnormalities is involved?

- (A) t(9;22)
- (B) del(1q)
- (C) t(15;17)
- (D) del 3q

Correct Answer: (A) t(9;22)

SOLUTION:

Step 1: Frame the clinical picture as a marrow problem.

A patient with fever, abdominal pain, massive hepatosplenomegaly, and a leukocyte count that has climbed above $50,000/\text{mm}^3$ is showing a markedly proliferative bone-marrow process. A white count this high with prominent splenomegaly in particular nudges us toward a myeloproliferative leukaemia rather than a reactive leukocytosis.

Step 2: Match the count to the disease.

Of the leukaemias, the one classically presenting with an enormous spleen, constitutional symptoms, and a strikingly elevated WBC (often with a left-shifted, full spectrum of granulocyte precursors) is Chronic Myeloid Leukaemia (CML).

Step 3: Recall the molecular lesion of CML.

CML is defined by the Philadelphia chromosome, which arises from a reciprocal translocation between chromosomes 9 and 22, written $t(9;22)$. This fuses BCR on 22 with ABL1 on 9, creating a constitutively active BCR-ABL1 tyrosine kinase that drives unchecked myeloid proliferation — the very engine behind the high count and big spleen described.

Step 4: Discard the other cytogenetic options.

- $del(1q)$ → seen in plasma-cell and other malignancies, not this leukocytosis picture.
- $t(15;17)$ → PML-RARA fusion of acute promyelocytic leukaemia, whose hallmark is DIC/coagulopathy rather than a sky-high chronic count.
- $del(3q)$ → a nonspecific abnormality across various haematologic disorders, not diagnostic here.

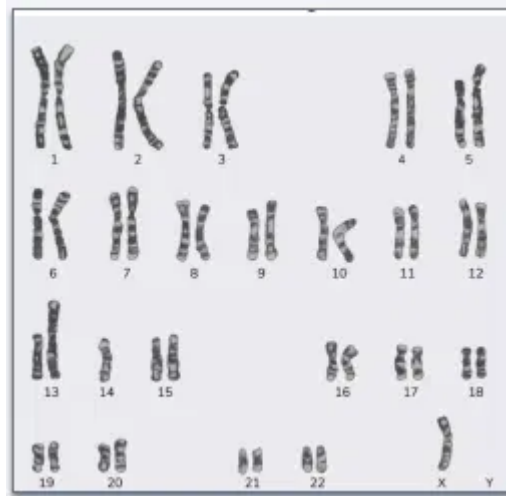
Final answer: The translocation underlying this CML presentation is $t(9;22)$ (Option 1).

Quick Tip: When a patient presents with high WBC count, hepatosplenomegaly, and abdominal pain, consider CML, especially if the Philadelphia chromosome ($t(9;22)$) is detected.



43.

A 23-year-old female with a height of 4 feet has a karyotype, as shown in the image below. Which of the following conditions is the most likely etiology?



- (A) Turner syndrome
- (B) Klinefelter syndrome
- (C) Mayer-Rokitansky-Küster-Hauser syndrome (MRKH)
- (D) Edwards syndrome

Correct Answer: (A) Turner syndrome

SOLUTION:

Step 1: Pull out the two anchor findings.

We have a phenotypic female (raised and presenting as a 23-year-old woman) with marked short stature — only about 4 feet tall — whose diagnosis is being read off a karyotype. So we need a condition where (a) the patient is female, (b) short stature is a defining feature, and (c) a chromosomal abnormality is visible on the karyotype.

Step 2: Test each option against those anchors.

- Klinefelter syndrome is a 47,XXY disorder of MALES, typically with TALL stature — it fails both the sex and the height anchors.
- Mayer-Rokitansky-Küster-Hauser syndrome affects females, but it is Müllerian agenesis (absent uterus/upper vagina) with a NORMAL 46,XX karyotype and normal height — it cannot be diagnosed from a karyotype and does not cause short stature.

- Edwards syndrome (trisomy 18) causes severe malformations and very poor survival; reaching age 23 with isolated short stature does not fit.

Step 3: Confirm the remaining diagnosis.

That leaves a 45,X karyotype — loss of one sex chromosome. This produces a short, phenotypically female patient, with short stature being one of the most consistent features, alongside gonadal dysgenesis and other stigmata. The karyotype-based, female, short-stature combination is exactly Turner syndrome.

Final answer: The most likely etiology is **Turner syndrome (Option 1)**.

Quick Tip: In a female with short stature and a 45,X karyotype, consider Turner syndrome. Additional features include a webbed neck and absent or underdeveloped ovaries.

• • •

44. An infant presents with vomiting after feeding. A Benedict's test was performed and returned positive for a non-glucose substance. Which of the following is the most likely diagnosis?

- (A) Galactosemia due to GAL-1-P uridyl transferase enzyme deficiency
- (B) Hereditary fructose intolerance due to aldolase B deficiency
- (C) Glucose-6-Phosphate dehydrogenase (G6PD) deficiency
- (D) Galactosemia due to galactokinase enzyme deficiency

Correct Answer: (A) Galactosemia due to GAL-1-P uridyl transferase enzyme deficiency

SOLUTION:

Step 1: Interpret the Benedict's test clue first.

Benedict's reagent turns positive with REDUCING sugars. The stem tells us the positive result is due to a NON-glucose reducing substance, so glucose itself is not the culprit. In a milk-fed infant, the reducing sugar that floods the system is

galactose (from lactose). This already tilts us toward a disorder of galactose metabolism.

Step 2: Tie the timing of symptoms to the feed.

The baby vomits AFTER FEEDING on milk. A condition that becomes symptomatic the moment milk (lactose → galactose) is introduced must sit on the galactose pathway and must be severe enough to cause toxicity early. That severity points to classic galactosemia.

Step 3: Localise the enzyme block.

Classic galactosemia is caused by deficiency of galactose-1-phosphate uridyltransferase (GALT). The block lets toxic galactose-1-phosphate accumulate (damaging liver, kidney, brain → vomiting, jaundice, hepatomegaly), while free galactose spills into blood and urine, giving the positive non-glucose Benedict's reaction.

Step 4: Rule out the alternatives.

- Hereditary fructose intolerance (aldolase B) → triggered by FRUCTOSE/sucrose, so symptoms appear after weaning onto fruits/sweets, not with early milk feeds.
- G6PD deficiency → causes oxidative haemolysis after triggers, not post-feed vomiting, and does not give a non-glucose reducing sugar.
- Galactokinase deficiency → also a galactosemia and Benedict-positive, but it is the milder variant whose dominant problem is cataracts, lacking the severe acute vomiting/hepatic picture seen here.

Final answer: The severe milk-triggered presentation with a non-glucose reducing sugar is **Galactosemia due to GAL-1-P uridyl transferase enzyme deficiency (Option 1)**.

Quick Tip: In infants with vomiting after feeding and a positive Benedict's test, consider galactosemia. The absence of GAL-1-P uridyl transferase enzyme activity is the most common cause.



45.

Which neonatal anomaly is depicted in the image?



- (A) Omphalocele
- (B) Gastroschisis
- (C) Patent vitello intestinal duct
- (D) Exstrophy bladder

Correct Answer: (D) Exstrophy bladder

SOLUTION:

Step 1: Decide the question is really about coverage and location.

The four neonatal anterior-wall anomalies are best separated by two questions: is the defect at the umbilicus or below it, and is the exposed organ bowel or bladder, covered by a sac or bare? Working through those two axes identifies the lesion shown.

Step 2: Profile each option.

- Omphalocele → midline UMBILICAL defect; abdominal viscera herniate through the cord base but remain enclosed in a peritoneal/amniotic SAC.
- Gastroschisis → defect to the RIGHT of the umbilicus; BOWEL extrudes with NO covering membrane.
- Patent vitello-intestinal duct → persistent omphalomesenteric connection

presenting as umbilical discharge/fistula, not a large exposed organ.

- Exstrophy of the bladder → a LOWER abdominal-wall defect in which the posterior bladder wall is everted and the bladder mucosa lies open and exposed on the abdominal surface, with no protective sac.

Step 3: Match to the depicted lesion.

An exposed, bare mucosal surface presenting low on the abdomen — bladder tissue turned inside-out and visible rather than herniated bowel in a sac — corresponds to bladder exstrophy. It is neither the sac-covered umbilical mass of omphalocele nor the uncovered loops of gastroschisis.

Final answer: The anomaly depicted is **Exstrophy bladder (Option 4)**.

Quick Tip: Exstrophy bladder involves the externalization of the bladder. Look for signs like an exposed bladder in the lower abdomen in neonates, which requires immediate surgical management.



46. Which of the following statements is incorrect regarding Samter's triad?

- (A) It is only triggered by aspirin
- (B) Other NSAIDs with COX-1 blocking ability can also trigger symptoms.
- (C) It is characterized by asthma, nasal polyps, and aspirin sensitivity.
- (D) It involves overproduction of leukotrienes.

Correct Answer: (A) It is only triggered by aspirin

SOLUTION:

Step 1: Note the question asks for the FALSE statement.

This is a negative-stem item about Samter's triad (aspirin-exacerbated respiratory disease, AERD). We must find the one statement that does NOT hold true.

Step 2: Recall the pathophysiology in one line.

AERD is driven by aberrant arachidonic-acid handling: inhibiting cyclooxygenase-

1 (COX-1) shunts arachidonate down the 5-lipoxygenase pathway, causing OVERPRODUCTION OF LEUKOTRIENES that provoke bronchospasm and nasal inflammation. The clinical triad is asthma + nasal polyps + sensitivity to aspirin/NSAIDs.

Step 3: Score each statement true or false.

- "Other NSAIDs with COX-1 blocking ability can also trigger symptoms" — TRUE; any COX-1 inhibitor (ibuprofen, naproxen, etc.) can set it off because the mechanism is COX-1 inhibition, not the aspirin molecule specifically.
- "Characterized by asthma, nasal polyps, and aspirin sensitivity" — TRUE; that is the defining triad.
- "Involves overproduction of leukotrienes" — TRUE; this is the core mechanism from Step 2.
- "It is only triggered by aspirin" — FALSE; this contradicts the COX-1 mechanism, since the whole class of COX-1-inhibiting NSAIDs can trigger reactions, not aspirin alone.

Step 4: Select the incorrect statement.

The single statement that is wrong is the one restricting the trigger to aspirin only.

Final answer: The incorrect statement is "It is only triggered by aspirin" (Option 1).

Quick Tip: In Samter's triad, COX-1 inhibitors like aspirin and NSAIDs can worsen symptoms. The overproduction of leukotrienes is a key feature of this disease.

• • •

47. Aprepitant, a drug used to prevent late-onset chemotherapy-induced nausea and vomiting, acts through which of the following mechanisms?

- (A) NK-1 antagonist
- (B) NK-1 agonist
- (C) NK-3 antagonist
- (D) NK-2 antagonist

Correct Answer: (A) NK-1 antagonist

SOLUTION:

Step 1: Recall the emetic pathway being targeted.

The vomiting reflex is driven in large part by the neurotransmitter substance P acting on Neurokinin-1 (NK-1) receptors in the brainstem emetic centre. Late-onset (delayed) chemotherapy-induced nausea and vomiting is particularly dependent on this substance-P/NK-1 signalling, which is why a drug effective against the delayed phase tends to work here.

Step 2: Reason from desired effect to required action.

To STOP vomiting through this pathway, a drug must BLOCK the receptor, i.e. act as an antagonist, not stimulate it. An agonist would do the opposite and provoke emesis. So the answer must be an NK-1 ANTAGONIST.

Step 3: Pick the correct receptor subtype.

Aprepitant binds the NK-1 receptor specifically, preventing substance P from triggering the emetic response. The NK-2 and NK-3 receptors are the targets of neurokinin A and neurokinin B respectively and are not the mediators of this antiemetic effect, so antagonising them does not explain aprepitant's action.

Step 4: Combine the two requirements.

Correct receptor (NK-1) + correct action (block/antagonise) = NK-1 antagonist.

Final answer: Aprepitant works as an **NK-1 antagonist (Option 1)**.

Quick Tip: Aprepitant is an NK-1 antagonist, effective in preventing chemotherapy-induced nausea and vomiting by blocking the action of substance P.

• • •

48. Sour taste is perceived by which receptors?

- (A) T1R1
- (B) T1R2
- (C) T1R3
- (D) TRPP3

Correct Answer: (D) TRPP3

SOLUTION:

Step 1: Separate the two receptor families in taste.

Taste modalities use two broad mechanisms. Sweet and umami rely on the T1R G-protein-coupled receptor family (T1R1/T1R2/T1R3 combinations), whereas sour and salty are detected through ION CHANNELS that sense protons and sodium directly. Recognising that sour is an ion-channel modality immediately steers us away from the T1R options.

Step 2: Recall what sour actually is.

Sourness is the taste of acidity — acids release hydrogen ions (H^+) in solution. Detecting sour therefore means sensing intracellular acidification/protons in the taste receptor cell, a job for a proton-sensitive channel, not for a sweet/umami GPCR.

Step 3: Name the channel and discard the T1R options.

The sour-sensing taste cells use the TRPP3 (PKD2L1) channel to transduce the acid signal into a neural response. By contrast: T1R1 (with T1R3) mediates umami, T1R2 (with T1R3) mediates sweet, and T1R3 is the shared partner subunit of those sweet/umami complexes — none of them encodes sour.

Final answer: Sour taste is perceived by the **TRPP3 (Option 4)** channel.

Quick Tip: Sour taste is detected by TRPP3 receptors, which are activated by acidic substances like citric acid found in lemons and other sour foods.

• • •

49. Which ion is most responsible for the resting membrane potential (RMP)?

- (A) Potassium (K^+)
- (B) Calcium (Ca^{2+})
- (C) Sodium (Na^+)
- (D) Chloride (Cl^-)

Correct Answer: (A) Potassium (K^+)

SOLUTION:

Step 1: What sets the resting potential?

The resting membrane potential is essentially a diffusion potential. Whichever ion the membrane lets cross most freely at rest will pull the cell's voltage toward that ion's own equilibrium (Nernst) value. So the question really asks: at rest, which ion has the highest permeability?

Step 2: Apply the Goldman idea.

The Goldman-Hodgkin-Katz relationship weights each ion by its permeability. At rest the membrane has many open potassium leak channels, so the permeability ratio is heavily dominated by K^+ . Because of this, the measured RMP (about -70 mV in a typical neuron) sits very close to the potassium equilibrium potential ($E_K \approx -90$ mV), and only slightly off because of a small sodium leak.

Step 3: Check the numbers for potassium.

K^+ is far more concentrated inside than outside, so its chemical gradient pushes it out. As positive charge leaves, the interior turns negative; this negativity then opposes further K^+ exit. Equilibrium between these two forces is reached very near E_K , which is why the resting voltage tracks potassium.

Step 4: Why not the others?

Na^+ ($E_{Na} \approx +60$ mV) would drag the voltage strongly positive, but at rest its permeability is tiny, so it contributes only a few millivolts. Ca^{2+} permeability at rest is negligible and it acts mainly as a signalling/excitation-contraction ion. Cl^- tends to redistribute passively toward whatever potential the cell already holds, so it follows rather than sets the RMP.

Conclusion: The high resting permeability to potassium makes it the chief determinant of the resting membrane potential.

Final Answer: Option 1 - Potassium (K⁺).

Quick Tip: The resting membrane potential (RMP) is primarily maintained by potassium (K⁺) ions, with the Na⁺/K⁺ ATPase pump helping to maintain the ion gradient.

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50.

A fruit farmer presents with fever, respiratory difficulties, and neurological complications. What is the most likely diagnosis?



- (A) Ebola
- (B) Meningitis
- (C) Nipah
- (D) Zika

Correct Answer: (C) Nipah

SOLUTION:

Step 1: Pull out the diagnostic clues.

Three threads must all be satisfied: an occupational exposure (fruit farmer), an acute febrile illness with respiratory difficulty, and prominent neurological involvement. The right answer is the disease where all three overlap.

Step 2: Reason from the exposure first.

Fruit farming places the patient in contact with fruit and, importantly, with fruit bats (*Pteropus*) and bat-contaminated fruit or date-palm sap. That ecological link is the hallmark of a single zoonosis.

Step 3: Match exposure to syndrome.

Nipah virus, a Henipavirus carried by fruit bats, classically produces a brief febrile prodrome that rapidly progresses to a combined respiratory illness (cough, breathlessness, ARDS) and a severe encephalitis (altered sensorium, seizures, focal deficits). This is exactly the fever + respiratory + neurological triad described, and outbreaks in South Asia are repeatedly traced to contaminated fruit/sap.

Step 4: Eliminate the distractors.

Ebola causes a haemorrhagic febrile illness (bleeding, shock) without a primary respiratory-encephalitic picture and is spread by direct contact with body fluids, not fruit. Meningitis gives fever with neck stiffness and photophobia but not the prominent respiratory failure or the fruit-bat link. Zika is generally a mild illness (low-grade fever, rash, conjunctivitis, arthralgia) and its neurological concern is congenital/Guillain-Barre, not an acute encephalitis with respiratory distress.

Conclusion: The fruit-bat exposure tying together respiratory and encephalitic disease points to Nipah.

Final Answer: Option 3 - Nipah.

Quick Tip: In regions with fruit bats, consider Nipah virus if the patient presents with fever, respiratory distress, and neurological symptoms like encephalitis.



51.

Which of the following vaccines is kept at the lowest level?



- (A) OPV
- (B) DPT
- (C) Hep B
- (D) Rota

Correct Answer: (A) OPV

SOLUTION:

Step 1: Frame the cold-chain logic.

Vaccines are arranged in the cold chain by how heat-labile they are. Live attenuated, especially RNA-based, vaccines are the most fragile and are stored coldest; subunit and toxoid vaccines tolerate ordinary refrigerator temperatures. So "kept at the lowest level" means "requires the deepest freeze."

Step 2: Sort the options by formulation.

OPV is a live attenuated poliovirus vaccine - the most freeze-tolerant and freeze-requiring of the group, traditionally held in the deep-freeze compartment at roughly -15°C to -25°C . DPT contains toxoids plus killed pertussis; Hep B is a recombinant subunit antigen; Rota is live but is kept refrigerated, not frozen. The last three all sit in the $+2^{\circ}\text{C}$ to $+8^{\circ}\text{C}$ band and must never be frozen.

Step 3: Apply the practical refrigerator rule.

In an ILR (ice-lined refrigerator) and in the vaccine carrier, OPV is the one vaccine placed in/closest to the freezer because it needs and survives the lowest

temperature. The freeze-sensitive vaccines (DPT, Hep B, and to it Rota) are deliberately kept away from the freezing plate.

Conclusion: Being live attenuated and freeze-stable, OPV is stored at the lowest temperature of the listed vaccines.

Final Answer: Option 1 - OPV.

Quick Tip: The OPV requires the lowest temperature in the vaccine cold chain, so ensure it is stored at -20°C to -30°C for optimal efficacy.

• • •

52. Which drug is used in the maintenance phase of opioid withdrawal?

- (A) Buprenorphine
- (B) Disulfiram
- (C) Butorphanol
- (D) Clonidine

Correct Answer: (A) Buprenorphine

SOLUTION:

Step 1: Separate the two phases of opioid treatment.

Detoxification handles the short-term, acutely uncomfortable withdrawal symptoms, whereas the maintenance phase is the long-term phase aimed at keeping the patient stable, blunting cravings, and preventing relapse. The question targets the maintenance drug.

Step 2: Match each drug to its role.

Maintenance is done with an agent that occupies the mu-opioid receptor in a controlled way. Buprenorphine, a partial mu-agonist (with kappa-antagonism), does exactly this: it relieves cravings and withdrawal, has a ceiling effect that limits respiratory depression and abuse potential, and is suitable for long-term substitution therapy (often combined with naloxone).

Step 3: Rule out the rest by mechanism.

Clonidine is an alpha-2 agonist that dampens autonomic withdrawal (sweating, tachycardia, anxiety) during detox only - it is not a maintenance agent.

Butorphanol is a mixed agonist-antagonist used as an analgesic and can actually precipitate withdrawal, so it is not used for maintenance. Disulfiram belongs to alcohol-dependence treatment, acting via aldehyde dehydrogenase inhibition, and has no role in opioid care.

Conclusion: The long-term, anti-craving, partial-agonist substitution drug for opioid dependence is buprenorphine.

Final Answer: Option 1 - Buprenorphine.

Quick Tip: For opioid withdrawal, buprenorphine is commonly used for maintenance therapy due to its partial agonist activity on opioid receptors.

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53. A 45-year-old woman presents with a long-standing history of “feeling tensed” along with an upset stomach, diarrhea, and heartburn. She mentions that her family members also experience similar symptoms. Which of the following symptoms is most likely to be observed in this patient?

- (A) Neologisms
- (B) Tingling at extremities
- (C) Hallucinations
- (D) Idea of reference

Correct Answer: (B) Tingling at extremities

SOLUTION:

Step 1: Identify the underlying disorder.

A long history of feeling tense with gut complaints (upset stomach, diarrhoea, heartburn) and a familial pattern fits a chronic anxiety disorder rather than a

psychotic illness. The clue to the expected sign is the physiology that anxiety triggers.

Step 2: Trace the physiological chain.

Anxious patients tend to hyperventilate. Excessive breathing blows off CO_2 , lowering arterial $PaCO_2$ and producing respiratory alkalosis (a rise in blood pH). Alkalosis increases binding of calcium to albumin, dropping the ionised calcium and raising neuromuscular excitability.

Step 3: Predict the clinical sign.

This functional hypocalcaemia/alkalosis classically causes paraesthesias - tingling and numbness of the fingertips, toes, and around the mouth (perioral), sometimes with carpopedal spasm. So the symptom expected here is tingling at the extremities.

Step 4: Discard the psychotic options.

Neologisms (invented words), hallucinations, and ideas of reference are features of psychotic spectrum disorders such as schizophrenia, not of an anxiety disorder with somatic and hyperventilation symptoms. They do not fit this clinical picture.

Conclusion: Anxiety-driven hyperventilation, respiratory alkalosis, and a fall in ionised calcium explain the tingling.

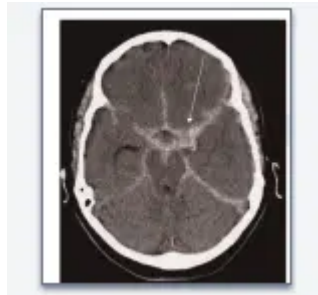
Final Answer: Option 2 - Tingling at extremities.

Quick Tip: In somatic symptom disorders or anxiety, look for physical manifestations like tingling or other somatic complaints without a clear medical cause.



54.

A 40-year-old female patient complains of a persistent headache. A CT scan of the head was performed, as shown below. What is the most accurate diagnosis?



- (A) Subarachnoid hemorrhage (SAH)
- (B) Epidural hemorrhage
- (C) Subdural hemorrhage
- (D) Intraparenchymal hemorrhage

Correct Answer: (A) Subarachnoid hemorrhage (SAH)

SOLUTION:

Step 1: Read the CT by bleed pattern.

The fastest way to classify intracranial haemorrhage is by the shape and location of the hyperdense (white) blood on a non-contrast head CT. Each bleed type occupies a characteristic space, so the geometry of the blood is the key.

Step 2: Catalogue the patterns.

Blood that fills and outlines the natural CSF spaces - the basal cisterns, Sylvian fissures, and interhemispheric fissure, tracing a star-like configuration - lies in the subarachnoid space. A lens/biconvex collection that does not cross sutures is epidural; a crescent collection hugging the brain surface and crossing sutures is subdural; a rounded clot inside the brain substance is intraparenchymal.

Step 3: Add the clinical layer.

Subarachnoid haemorrhage classically presents with a sudden, extremely severe "thunderclap" headache (worst of life), often spontaneous (ruptured berry aneurysm). The patient's prominent headache fits this and supports CSF-space blood rather than a traumatic surface collection.

Step 4: Conclude from pattern + history.

Hyperdensity outlining the cisterns and fissures plus the severe-headache history points to bleeding in the subarachnoid space rather than an epidural, subdural, or intraparenchymal bleed.

Conclusion: The CSF-space (cisternal/fissural) distribution of blood with thunderclap headache indicates subarachnoid haemorrhage.

Final Answer: Option 1 - Subarachnoid hemorrhage (SAH).

Quick Tip: In a patient with sudden severe headache, a subarachnoid hemorrhage (SAH) should be suspected, especially if blood is seen in the subarachnoid space on a CT scan.

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55.

A 45-year-old patient presents with ptosis and muscle weakness that improves with rest. She also complains of dysphagia and has engorged veins in her thorax. Based on the CT scan image provided, what is the most likely diagnosis?



- (A) Thymoma
- (B) Pancoast tumor
- (C) Adenocarcinoma
- (D) Small cell lung cancer

Correct Answer: (A) Thymoma

SOLUTION:

Step 1: Spot the syndrome behind the symptoms.

Ptosis and fatigable muscle weakness that improves with rest is the textbook signature of myasthenia gravis (autoantibodies against the acetylcholine receptor at the neuromuscular junction). Recognising this disease is the entry point to the answer.

Step 2: Link the disease to a tumour.

Myasthenia gravis is strongly associated with an anterior mediastinal mass arising from the thymus. Roughly a tenth of MG patients harbour a thymoma, making it the lesion to look for on the CT.

Step 3: Explain the additional findings.

Dysphagia reflects bulbar muscle involvement of the same myasthenic process. The engorged thoracic veins indicate superior vena cava compression - an anterior mediastinal thymoma pressing on the SVC produces this. Both extra clues therefore converge on an anterior mediastinal thymic tumour.

Step 4: Exclude the lung tumours.

A Pancoast (superior sulcus) tumour sits at the lung apex and causes Horner syndrome and arm pain, not fatigable weakness. Adenocarcinoma and small cell lung cancer are parenchymal lung tumours; while small cell can cause a paraneoplastic Lambert-Eaton syndrome, that weakness improves with activity (not with rest) and these are not anterior mediastinal masses. None matches the MG-plus-anterior-mediastinal picture.

Conclusion: Fatigable weakness/ptosis (myasthenia gravis) with an anterior mediastinal mass and SVC compression indicates a thymoma.

Final Answer: Option 1 - Thymoma.

Quick Tip: When a patient presents with ptosis, muscle weakness, and dysphagia along with chest imaging abnormalities, consider thymoma as a potential cause, especially in the context of myasthenia gravis.

56.

What instrument is used to insert this tube?



- (A) Nephroscope
- (B) Cysto-urethroscope
- (C) Ureteroscope
- (D) Endoscope

Correct Answer: (B) Cysto-urethroscope

SOLUTION:

Step 1: Decide the route, then the scope.

Endoscopic instruments are named for the part of the urinary tract they are designed to reach. Identifying which passage the tube/catheter must travel through tells you which scope inserts it.

Step 2: Map each scope to its territory.

A nephroscope reaches the renal pelvis and calyces, usually via a percutaneous track into the kidney. A ureteroscope is a long thin scope passed up the ureter to treat ureteric stones. "Endoscope" is only a generic umbrella term and not a specific urological instrument. A cysto-urethroscope is introduced through the urethra to view the urethra and bladder and to place catheters or stents into the bladder.

Step 3: Match to the task shown.

Inserting a tube into the bladder through the natural urethral route is precisely the job of the cysto-urethroscope, which provides the access and visualisation needed to position the tube correctly.

Step 4: Eliminate the others.

The nephroscope and ureteroscope target the kidney and ureter, not bladder-tube placement via the urethra, and "endoscope" is too non-specific to be the intended answer.

Conclusion: Trans-urethral access to the bladder for tube insertion is performed with the cysto-urethroscope.

Final Answer: Option 2 - Cysto-urethroscope.

Quick Tip: For placing a nephrostomy tube, a cysto-urethroscope is the appropriate instrument as it allows visualization of the bladder, ureters, and renal pelvis.

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57. What is the ideal pressure setting for vacuum-assisted closure (VAC) therapy?

- (A) -125 mm Hg
- (B) 60 to 80 mm Hg
- (C) 130 mm Hg
- (D) 80 to 100 mm Hg

Correct Answer: (A) -125 mm Hg

SOLUTION:

Step 1: Understand what VAC (negative-pressure wound) therapy does.

VAC therapy seals the wound with a foam dressing under an airtight film and connects it to a controlled suction pump. The applied sub-atmospheric (negative) pressure draws out infected exudate, decompresses interstitial oedema, mechanically deforms cells to stimulate proliferation (mechanotransduction), and improves local perfusion at the wound bed.

Step 2: Why the pressure value matters.

The therapeutic benefit is dose-dependent on the suction level. Too little suction

fails to evacuate fluid or stimulate the wound; excessive suction can compromise capillary blood flow and cause tissue ischaemia. So an optimal target is needed that maximises granulation-tissue formation and microvascular flow without ischaemic injury.

Step 3: Recall the validated optimum.

Experimental and clinical work (the original Argenta and Morykwas studies) showed that peak granulation-tissue growth and peak increase in local blood flow occur at a continuous negative pressure of **-125 mm Hg**. This is the standard recommended setting for most acute and chronic wounds.

Step 4: Map to the options.

- **-125 mm Hg** → the evidence-based optimum (matches option 1).
- 60 to 80 mm Hg → too low, sub-therapeutic for standard VAC.
- 130 mm Hg → not the validated value, and pressure here is negative (sub-atmospheric), not positive.
- 80 to 100 mm Hg → below the established optimal target.

Final answer: The ideal pressure setting for VAC therapy is **Option 1: -125 mm Hg**.

Quick Tip: For VAC therapy, the optimal pressure is -125 mm Hg, which maximizes wound healing while minimizing the risk of tissue injury.

• • •

58. Diagnosis of MRCP image.



- (A) GB Stone
- (B) Cholangiocarcinoma
- (C) Choledochal Cyst
- (D) Gall and Blood Cancer

Correct Answer: (B) Cholangiocarcinoma

SOLUTION:

Approach the image as an obstructive-jaundice problem.

MRCP exploits the fact that bile is fluid: on a heavily *T2*-weighted sequence fluid is hyperintense, so the whole biliary tree is rendered bright. The diagnostic question on any MRCP is therefore: is the lesion a luminal defect (stone), a smooth dilatation (cyst/choledochal anomaly), or a stricture (tumour)?

What the picture tells us.

Beyond the gallbladder marked "GB", the duct shows a tight, irregular focal narrowing with the ducts above it ballooning out (proximal dilatation). A blockage that narrows the duct itself and dilates everything upstream is the signature of a wall-based ductal malignancy.

Mechanism of the answer.

A bile-duct adenocarcinoma (cholangiocarcinoma) grows in and around the duct wall, encasing and choking the lumen, which is why imaging shows an abrupt shouldered stricture plus upstream dilatation rather than a discrete pebble-like defect.

Eliminating the others.

- A gallbladder stone is an intraluminal signal void inside the GB, not a duct stricture.
- A choledochal cyst is a smooth, congenital fusiform widening of the common bile duct, usually in the young, with no obstructing tumour stricture.
- "Gall and Blood Cancer" is not a real diagnosis.

Hence the lesion is a **cholangiocarcinoma (B)**.

Quick Tip: On MRCP, an abrupt irregular ductal stricture with upstream dilatation points to a tumour of the duct wall, not a stone or a smooth congenital cyst.

• • •

59. A 35 year old nullipara female presented with dull aching pain with exacerbation during menstrual cycle. Usg reveals a heterogenous mass in right adnexa. Mri reveals a 4 x 5 cm T1 hyperintense mass with no suppression on fat started images. T2 weighted images show low signal with Dark shading. Diagnosis?

- (A) Dermoid cyst
- (B) Endometrioma
- (C) Ovarian cancer
- (D) Para Ovarian Cyst

Correct Answer: (B) Endometrioma

SOLUTION:

Solve it as a signal-intensity discriminator problem.

Two ovarian masses light up on **T1**: a dermoid (fat) and an endometrioma (blood). The single test that separates them is fat suppression — fat goes dark, blood stays bright. The stem hands you the discriminator directly: the mass is **T1** bright and does NOT lose signal on fat-saturated images, so its content is blood, not fat. That immediately favours an endometrioma over a dermoid.

Confirm with the T2 behaviour.

The mass is described as low signal with "dark shading" on **T2**. T2 shading is the textbook endometrioma sign: cyclical bleeding deposits iron-rich, high-protein degraded blood that shortens **T2** and dims the normally bright cyst fluid. A simple cyst would stay uniformly bright on **T2**.

Tie it back to the history.

The patient has dull pelvic pain that flares with menstruation. Endometriotic tissue is hormonally responsive and bleeds each cycle, which is exactly why the pain is cyclical and why the cyst accumulates the blood products that produce the

imaging signature ("chocolate cyst").

Rule out the rest.

- Dermoid: **T1** bright but suppresses with fat sat — excluded by the stem.
- Ovarian cancer: expects solid/papillary enhancing tissue and septa, not pure haemorrhagic shading, and the benign cyclical history fits poorly.
- Para-ovarian cyst: a simple cyst that follows fluid (**T1** dark, **T2** bright) — wrong signal entirely.

Diagnosis: **Endometrioma (B)**.

Quick Tip: T1 bright that does NOT suppress on fat sat = blood, not fat; add cyclical pain and 'T2 shading' and you have a chocolate cyst.

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60. Diagnosis of following Plain Xray of abdomen.



- (A) Enterolith in jejunum
- (B) Calcific mediastinal lymph nodes
- (C) Horse shoe kidney with calculi
- (D) Chronic calcific pancreatitis

Correct Answer: (D) Chronic calcific pancreatitis

SOLUTION:

Pattern-recognise the calcification first.

On a KUB film, the diagnosis usually comes from the SHAPE and SITE of the calcium. Here there is not one stone but a SCATTER of coarse flecks laid out transversely across the upper abdomen. A band of multiple small calcifications

running side-to-side across the epigastrium maps onto one organ — the pancreas, which itself lies transversely at the L1–L2 level.

Why pancreatic calcification looks like this.

In chronic pancreatitis, repeated inflammation precipitates protein plugs inside the pancreatic ductules; these plugs mineralise into calcium carbonate stones. Because they sit throughout the duct system of a transversely oriented gland, the plain film shows multiple speckled calcifications strung across the upper abdomen — the hallmark of chronic calcific (often alcoholic or tropical) pancreatitis.

Knock out the alternatives by location/number.

- Enterolith: a solitary rounded calcified mass inside a bowel loop, not a transverse scatter.
- Calcified mediastinal nodes: these are in the chest, above the diaphragm — they have no business on an abdomen film.
- Horseshoe kidney with stones: calcifications would track to the renal beds/paraspinal gutters low across the midline, following fused lower poles, not the pancreatic line.

So the radiograph is diagnostic of **chronic calcific pancreatitis (D)**.

Quick Tip: Multiple coarse calcifications scattered transversely across the upper abdomen along the line of the pancreas = chronic calcific pancreatitis.

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61. A patient has Proteus infection and now plain xray of abdomen was done and a large stone was shown in urinary bladder. Stone is made up of

- (A) Calcium Phosphate
- (B) Cysteine
- (C) Calcium Oxalate
- (D) Xanthine

Correct Answer: (A) Calcium Phosphate

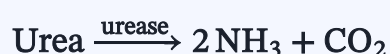
SOLUTION:

Start from the bug, not the stone.

The examiner gives you one decisive word: Proteus. *Proteus mirabilis* is the prototype UREASE-positive uropathogen, and urease is the enzyme that links infection to a specific stone chemistry. So the question is really "which stone does an alkaline, urease-driven urine make?"

Trace the chemistry.

Urease hydrolyses urea to ammonia and carbon dioxide:



The liberated ammonia drives urinary pH up. Alkaline urine is supersaturated for magnesium ammonium phosphate (struvite) and for carbonate-apatite (calcium phosphate), so these phosphate salts crystallise out and can build into a large, often staghorn or bladder, infection stone that is radio-opaque on plain film — matching the X-ray finding.

Map to the options.

Only one listed salt is a phosphate that precipitates in this alkaline, infection setting: calcium phosphate. The other three each belong to a different, non-infective mechanism:

- Cystine — inherited cystinuria, acidic urine, hexagonal crystals.
- Calcium oxalate — the commonest stone, but driven by metabolic/dietary factors in acidic urine, not by urease.
- Xanthine — rare enzyme defect/drug-related, radiolucent.

These struvite/phosphate stones precipitate fast in the supersaturated alkaline urine, are radio-opaque, and characteristically enlarge into bulky staghorn or bladder calculi exactly as described in the stem. Therefore the *Proteus* (urease) stone here is **calcium phosphate (A)**.

Quick Tip: Proteus = urease = alkaline urine = phosphate (infection) stone. Pick the



62. Correct statement regarding esophageal FB (foreign body).



- (A) Right main bronchus is one of the site of constriction
- (B) Most common site of impaction is cricopharyngeus
- (C) Commonly seen in adults
- (D) Cant cause mediastinitis

Correct Answer: (B) Most common site of impaction is cricopharyngeus

SOLUTION:

Frame it around the three oesophageal narrowings.

A foreign body sticks where the tube is tightest. The oesophagus narrows at three predictable points: the cricopharyngeus (upper sphincter, ~15 cm, the tightest of all), the arch of aorta / LEFT main bronchus crossing (~25 cm), and the cardia at the diaphragm (~40 cm). Rank these by calibre and the cricopharyngeus wins as the narrowest, hence the commonest lodging site — that is the true statement (B).

Test each option against this anatomy.

- A — wrong side: the second constriction is from the LEFT main bronchus and aortic arch indenting the oesophagus, not the right. (The right main bronchus matters for INHALED airway foreign bodies, a different question.)
- C — wrong age group: swallowed oesophageal foreign bodies are predominantly a paediatric problem (coins, button batteries, toys), not an adult one.
- D — wrong complication claim: an impacted or sharp FB can erode/perforate the oesophageal wall, and leakage into the mediastinum produces mediastinitis,

one of the most dangerous outcomes — so it certainly CAN cause it.

Conclusion.

The only accurate statement is that the cricopharyngeus is the most common impaction site, so the answer is **B**.

Quick Tip: The narrowest part of the gut is the cricopharyngeus, so it is the commonest oesophageal FB site; remember the constriction is the LEFT (not right) main bronchus.

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63. Gastrectomy patient needs supplemental: (PART A)

- (A) Vit c
- (B) Vit d
- (C) Vit b12
- (D) Vit a

Correct Answer: (C) Vit b12

SOLUTION:

Ask what the stomach uniquely contributes.

The stomach is dispensable for digesting most nutrients, but it has one absorptive job no other organ can replace: its parietal cells make INTRINSIC FACTOR, the carrier molecule that vitamin B12 must be bound to before the terminal ileum will take it up.

Consequence of removing the stomach.

Gastrectomy excises the parietal-cell mass, so intrinsic factor secretion ceases. Even with a normal diet, B12 now passes through the gut unabsorbed. Hepatic B12 stores buffer this for a couple of years, after which the patient develops megaloblastic anaemia and neurological injury (subacute combined degeneration). The fix is to bypass the gut entirely with intramuscular B12 for life.

Why the other vitamins are safe.

- Vitamin C is water-soluble and absorbed along the small bowel independent of the stomach.
- Vitamins A and D are fat-soluble and absorbed in the small intestine with bile salts, not via any gastric factor.

None of these three relies on intrinsic factor, so none is specifically threatened by gastrectomy.

Answer.

The vitamin that absolutely requires gastric intrinsic factor, and therefore must be supplemented after gastrectomy, is **vitamin B12 (C)**.

Quick Tip: No stomach = no parietal cells = no intrinsic factor, and intrinsic factor is needed only for vitamin B12.

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64. After a right limb amputation, the patient is experiencing severe pain phantom limb.

What is the mechanism behind this?

- (A) Projection of adjacent fibres to overlap to right sensory cortex
- (B) Projection of adjacent fibres to overlap to left sensory cortex
- (C) Expansion of right sensory cortex
- (D) Expansion of left sensory cortex

Correct Answer: (B) Projection of adjacent fibres to overlap to left sensory cortex

SOLUTION:

Two ideas decide this: plasticity and crossing.

Phantom limb pain is the classic example of maladaptive cortical reorganisation. When a limb is amputated, its patch on the primary somatosensory map is suddenly starved of input. Cortex hates silent territory, so the AFFERENT fibres of the neighbouring representations (face, residual stump, etc.) sprout sideways and

OVERLAP onto the orphaned limb area. Now, touching the face activates the limb's old cortical zone, and the brain interprets it as coming from the limb that is no longer there — felt as phantom sensation or pain.

Now place it in the correct hemisphere.

The somatosensory system is crossed (decussated): each half of the body is mapped on the OPPOSITE cerebral hemisphere. A RIGHT-sided amputation therefore deafferents the limb zone in the LEFT somatosensory cortex, and it is into that LEFT-hemisphere zone that the adjacent fibres project and overlap.

Filter the options.

- The mechanism must be "projection/overlap of adjacent fibres," not "expansion" of the limb's own cortex — so the expansion options (C, D) describe the wrong process (the deprived area is invaded/shrinks, it does not expand).
- It must be the LEFT cortex for a RIGHT limb because of contralateral wiring — so the right-cortex options (A, C) are wrong-sided.

The only choice that gets BOTH the mechanism (adjacent-fibre overlap) and the side (left cortex) right is **B**. This is also why therapies such as mirror-box training help: they feed corrected visual input to the reorganised left-hemisphere zone and partly reverse the maladaptive remapping that drives the phantom pain.

Quick Tip: Phantom pain = adjacent cortical fibres overlapping the deafferented zone; and a right limb maps to the contralateral (left) sensory cortex.

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65. Patient had SUN BURN, now takes bath in 40 degree water, and feels pain.

- (A) Thermal receptor: hyperalgesia
- (B) Innocuous thermal receptor allodynia
- (C) Thermal receptor allodynia
- (D) Innocuous thermal receptor hyperalgesia

Correct Answer: (B) Innocuous thermal receptor allodynia

SOLUTION:

Approach this by asking one question first: **was the triggering stimulus normally painful or normally harmless?**

Skin pain (nociceptive) thresholds for heat lie near 45°C. A 40°C bath is below that, so for healthy skin it is simply pleasantly warm — an **innocuous** thermal stimulus that should produce no pain at all.

Now layer on the pathology. Sunburn (UV injury) drives release of inflammatory mediators (prostaglandins, bradykinin, substance P) that **sensitise peripheral nociceptors and lower their firing threshold**. As a result the previously sub-threshold 40°C warmth is now strong enough to fire these primary afferents and is consciously perceived as pain.

The textbook label for "pain produced by a stimulus that does not normally provoke pain" is **allodynia**. Contrast this with hyperalgesia, which is an amplified response to a stimulus that is *already* noxious — that label would only apply if, say, a clearly painful 50°C contact hurt far more than expected.

So the precise description is a thermal, innocuous stimulus producing pain = **innocuous thermal receptor allodynia**. Options invoking hyperalgesia are wrong because the stimulus here is non-noxious, and the bare "thermal receptor allodynia" choice is less complete than the one that specifies the stimulus is innocuous.

Correct option: B.

Quick Tip: Pain from a NON-painful stimulus = allodynia; exaggerated pain from an already-painful stimulus = hyperalgesia. Is 40°C normally painful?

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66. A 50 yr old female presented with dyspnoea and chest pain. Which of the following proves that she has aortic stenosis rather than aortic regurgitation?

- (A) Increase in myocardial oxygen consumption is seen with increased pressure work than volume
- (B) Aortic stenosis causes reduced pressure at aortic valve
- (C) Workload has nothing to do with myocardial oxygen consumption
- (D) Increase in preload more than afterload than after load

Correct Answer: (A) Increase in myocardial oxygen consumption is seen with increased pressure work than volume

SOLUTION:

Frame the whole question around a single physiological idea: **pressure work is metabolically far more "expensive" for the heart than volume work.**

First, classify the two valve lesions by the type of load they impose:

- Aortic stenosis → the LV pushes blood through a narrowed valve → it must build a high intraventricular pressure → **pressure (afterload) overload.**
- Aortic regurgitation → blood leaks back in diastole → the LV fills with extra volume each beat → **volume (preload) overload.**

Next, recall what determines oxygen use by the myocardium. The dominant factor is ventricular wall stress, given by Laplace's law

$$\sigma = \frac{P \times r}{2h}$$

where **P** is intraventricular pressure, **r** the radius and **h** the wall thickness.

Because oxygen consumption tracks wall stress (plus heart rate and contractility), a rise in **developed pressure** costs much more oxygen than simply moving more blood volume at the same pressure.

Therefore the statement "increase in myocardial oxygen consumption is seen with increased pressure work more than volume work" is the one that fingerprints **aortic stenosis** (the pressure-overload lesion).

Quickly discard the rest: AS produces a high — not reduced — transvalvular pressure gradient (so the "reduced pressure" option is wrong); workload

absolutely does drive myocardial O₂ demand (so that denial is wrong); and a preload-greater-than-afterload picture describes the volume overload of regurgitation, not stenosis.

Hence option A is correct.

Quick Tip: AS = pressure (afterload) overload; AR = volume (preload) overload. Recall which type of work costs the myocardium more oxygen (Laplace's law).

• • •

67. Taste is absent in which taste bud (papilla)?

- (A) Filiform
- (B) Foliate
- (C) Fungiform Papilla Tash
- (D) Circumvallate

Correct Answer: (A) Filiform

SOLUTION:

Sort the four lingual papillae into "tastes" versus "does not taste" and the answer falls out immediately.

Three of the four — **fungiform** (anterior tongue, mushroom-shaped), **foliate** (leaf-like folds on the posterolateral edges) and **circumvallate/vallate** (the large V-shaped row in front of the sulcus terminalis) — all house **taste buds** and so contribute to gustation.

The odd one out is the **filiform** papilla. These are the smallest and by far the most numerous papillae, they are keratinised and thread/cone-shaped, and crucially they carry **no taste buds at all**. Their job is purely mechanical — they roughen the tongue's surface to grip and move food and to sense texture, not flavour.

So the single papilla in which taste is absent is the **filiform** papilla; every other

listed papilla detects taste.

Correct option: A.

Quick Tip: Three lingual papillae carry taste buds; only one is purely mechanical/keratinised with no taste buds.

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68. Which receptor (transporter) helps in improvement of insulin resistance in DM2 (type 2 diabetes mellitus) with regular exercise and physical activity?

- (A) GLUT1
- (B) GLUT4
- (C) GLUT2
- (D) GLUT3

Correct Answer: (B) GLUT4

SOLUTION:

The key is to remember the **one glucose transporter that responds both to insulin and to muscle contraction — GLUT4.**

Skeletal muscle is the largest site of insulin-stimulated glucose disposal, and it does this through GLUT4, which sits in intracellular vesicles and moves to the cell surface only when triggered. In type 2 diabetes, insulin's ability to trigger that translocation is blunted (insulin resistance), so glucose stays in the blood.

Exercise provides a **second, parallel trigger** for GLUT4 that bypasses the faulty insulin pathway. Contracting muscle raises intracellular Ca^{2+} and activates AMP-activated protein kinase (AMPK); both independently drive GLUT4 vesicles to the membrane. With regular training, muscle also **up-regulates total GLUT4 content** and improves insulin sensitivity of the transporter. The net effect is greater glucose clearance into muscle and reduced insulin resistance — the clinically observed benefit of physical activity in DM2.

By contrast, GLUT1 handles constant baseline uptake (red cells, brain barrier), GLUT2 is the high-capacity sensor of liver/ β -cells and gut, and GLUT3 supplies neurons — none are the exercise-recruited muscle transporter.

Therefore the answer is GLUT4 (option B).

Quick Tip: Which GLUT is insulin-responsive in skeletal muscle and also recruited to the membrane by muscle contraction (via AMPK/ Ca^{2+})?

• • •

69. A man has difficulty in sleep during the night. He has a habit of drinking coffee before bed time. What is the role of caffeine in wakefulness?

- (A) Blocks adenosine action and causes wakefulness
- (B) Activates locus coeruleus and causes wakefulness
- (C) No role in maintaining wakefulness if taken 1 hr before sleep
- (D) Activates histamine release and prevents sleep

Correct Answer: (A) Blocks adenosine action and causes wakefulness

SOLUTION:

Start from the molecule the brain uses to *make* us sleepy: **adenosine**.

The longer we stay awake, the more adenosine builds up in the brain as a by-product of energy (ATP) metabolism. By binding its A_1/A_{2A} receptors, adenosine dampens the arousal-promoting networks and creates the rising "sleep pressure" we feel by night-time. In short, adenosine = the brake that promotes sleep.

Caffeine works by sitting on those same adenosine receptors as a **competitive antagonist** — it has a similar shape, occupies the receptor, but produces no effect. With adenosine blocked, its sleep-promoting brake is released, the arousal systems keep firing, and the person stays alert. That is the entire basis for caffeine-induced wakefulness, and it explains why an evening coffee leaves this

patient unable to sleep (caffeine's half-life of several hours means a dose taken near bedtime is still active).

The other options misattribute the mechanism: caffeine does not chiefly work by switching on the locus coeruleus or by liberating histamine, and it is certainly not inert when taken an hour before bed.

Hence the correct choice is A — caffeine blocks adenosine and causes wakefulness.

Quick Tip: Caffeine is a competitive antagonist at the receptor of the brain's main sleep-promoting molecule. Which molecule?

• • •

70. Resting membrane potential (RMP) is predominantly affected by which ion?

- (A) K^+
- (B) Ca
- (C) Na
- (D) Cl

Correct Answer: (A) K^+

SOLUTION:

The trick is to remember that the resting membrane potential settles near the equilibrium potential of **whichever ion the membrane lets through most easily at rest.**

At rest, the cell membrane is studded with open potassium "**leak**" **channels**, making it far more permeable to K^+ than to Na^+ , Ca^{2+} or Cl^- . Because K^+ permeability dominates, the resting voltage is pulled toward the potassium equilibrium potential calculated by the Nernst equation

$$E_K = 61 \times \log_{10} \frac{[K^+]_{out}}{[K^+]_{in}} \approx -90 \text{ mV}$$

which is why a typical RMP sits around -70 to -90 mV. A useful confirmation: changing extracellular K^+ shifts the RMP markedly (e.g. hyperkalaemia depolarises the cell), whereas altering extracellular Na^+ barely moves it.

The remaining ions play other roles: Na^+ rushing in produces the action-potential **upstroke** but contributes little at rest; Ca^{2+} governs the plateau in cardiac cells and triggers release/contraction; Cl^- mostly equilibrates passively and only modestly stabilises the potential.

So the ion that predominantly sets the resting membrane potential is potassium — option A.

Quick Tip: At rest the membrane is most permeable to one ion via leak channels, so RMP sits near that ion's Nernst potential. Which ion?

• • •

71. Alcoholic gait, nystagmus after RTA (road traffic accident) — which lobe of the cerebellum is affected?

- (A) Flocculonodular
- (B) Dentate
- (C) Anterior lobe
- (D) Vermis

Correct Answer: (A) Flocculonodular

SOLUTION:

Localise the lesion by using the **single most specific sign first — the nystagmus.**

Map the cerebellum onto its three functional zones:

- **Vestibulocerebellum = flocculonodular lobe** → equilibrium, balance and the control of eye movements (so its damage → nystagmus + truncal/gait imbalance).

- **Spinocerebellum = vermis + anterior lobe** → posture and gait coordination.
- **Cerebrocerebellum = lateral hemispheres** (output via the dentate nucleus)
→ planning of fine, skilled limb movements.

This patient after head trauma shows a drunken, wide-based ("alcoholic") gait *together with* nystagmus. Of all the listed regions, the one whose function explicitly couples balance with eye-movement control is the **flocculonodular lobe** — because it is the cerebellum's interface with the vestibular system, its injury characteristically produces both unsteady gait and nystagmus.

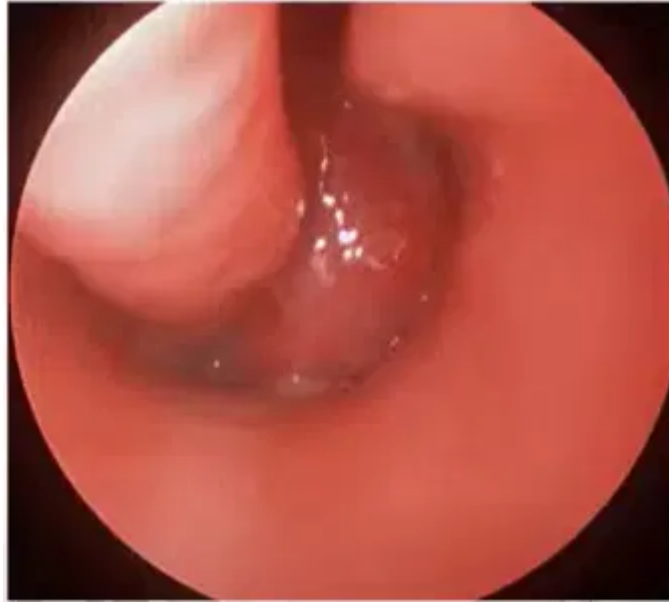
Cross-checking the others: the dentate is a deep nucleus (its lesions give intention tremor and limb dysmetria, not nystagmus); the anterior lobe is the classic chronic-alcoholic site causing gait ataxia but is not the eye-movement centre; and the vermis produces truncal ataxia without nystagmus being its hallmark. The pairing of nystagmus with gait disturbance is what selects the flocculonodular lobe.

Therefore option A is correct.

Quick Tip: Which cerebellar lobe is the vestibulocerebellum, controlling both balance/equilibrium AND eye movements (nystagmus)?

• • •

72. A 14yr old boy presented with nasal mass and recurrent episodes of bleeding from the mass. Investigation of choice.



- (A) Plain CT
- (B) CT with contrast
- (C) X Ray Caldwell view
- (D) X Ray with Pierre view

Correct Answer: (B) CT with contrast

SOLUTION:

Approach the scenario first. The single most discriminating combination here is: adolescent male + nasal mass + recurrent epistaxis. That triad should immediately make you think Juvenile Nasopharyngeal Angiofibroma, a benign but locally invasive tumour fed mainly by the internal maxillary artery.

The governing principle: in any suspected angiofibroma, NEVER biopsy first because the lesion is extremely vascular and can bleed catastrophically. Instead you confirm the diagnosis and map the tumour with imaging that shows its vascularity.

Pick the modality by what it must demonstrate. The ideal study should (1) light up the lesion because of its rich blood supply, (2) trace spread into the pterygopalatine and infratemporal fossae and toward the skull base, and (3)

show the classic anterior bowing of the posterior maxillary sinus wall (Holman-Miller / antral sign). A study done WITH intravenous contrast is what produces the bright enhancement that proves the mass is vascular - so contrast-enhanced CT fits all three needs.

Eliminate the rest:

- Plain CT → shows the soft-tissue mass and bone but cannot show the diagnostic vascular blush, so it under-performs versus contrast CT.
- X-ray Caldwell view → a plain frontal-sinus radiograph; it cannot characterise vascularity or deep extension.
- X-ray Pierre view → not a recognised work-up for this tumour.

Conclusion: the investigation of choice is contrast-enhanced CT, i.e. option B.

Quick Tip: Teenage boy + nasal mass + recurrent epistaxis = angiofibroma; it is highly vascular, so never biopsy - pick the imaging that shows vascular enhancement.

• • •

73. Cochlear implant given below, identify the marked structure:



- (A) Internal magnet
- (B) Electrode
- (C) Receiver
- (D) Antenna

Correct Answer: (B) Electrode

SOLUTION:

Start by recalling the anatomy of where each part lives. Think of a cochlear implant in two halves: the bits worn on the outside of the head (microphone, processor, and the transmitting coil/antenna held on by a magnet) and the bits surgically buried (the receiver-stimulator package and the electrode array).

Localise the marked structure. The arrow in the figure points to a long, fine, coiled wire that runs into the snail-shaped cochlea (and on the X-ray you can literally see it curled inside the cochlear turns). Only ONE component is built as a thin flexible coil designed to be inserted into the scala tympani - the electrode array.

Function check confirms it. The job of that intracochlear coil is to deliver tiny electrical currents along its many contacts so that different points of the cochlea (high to low frequency, tonotopically arranged) stimulate the spiral ganglion and cochlear nerve. That is precisely the electrode's role.

Rule out the distractors by location/shape:

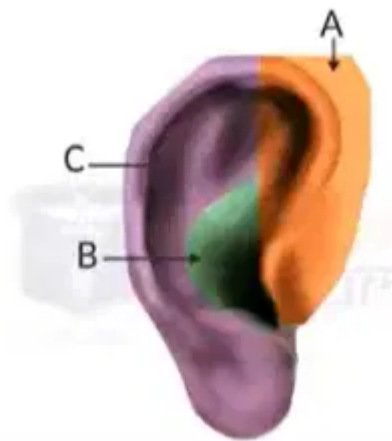
- Internal magnet → a small round disc that anchors the external coil; not a wire in the cochlea.
- Receiver (receiver-stimulator) → the chunky implanted box under the scalp that decodes the radio signal; it never enters the cochlea.
- Antenna (transmitting coil) → an external component worn on the skin.

Therefore the structure threaded into the cochlea is the electrode, so the correct option is B.

Quick Tip: Which single component is actually threaded inside the cochlea to deliver current? That coiled intracochlear wire is the answer.



74. Which nerve supplies the external ear?



- (A) ATN, greater auricular, 7 and 10
- (B) Greater auricular, 7 and 10, ATN
- (C) 7 and 10, ATN, greater auricular nerve
- (D) ATN, 7 and 10, GAN

Correct Answer: (D) ATN, 7 and 10, GAN

SOLUTION:

Build the answer from anatomy, then read the options. The external ear (the auricle plus the external auditory canal) does not have a single nerve - it is a meeting point of cranial and cervical nerves, which is exactly why pain in the ear (otalgia) can be referred from teeth, throat, neck and even the chest.

The four contributors to remember, with their roots and territories:

- Auriculotemporal nerve (ATN), from the mandibular division V3 of the trigeminal → supplies the front (anterosuperior) part of the auricle and the anterior wall of the external auditory canal.
- Greater auricular nerve (GAN), from the cervical plexus, roots C2-C3 → supplies the lower auricle and the lobule.
- Facial nerve, cranial nerve 7 (via the nervus intermedius) → supplies part of the concha and posterior canal; this is the Ramsay Hunt / Hitselberger sensory zone.
- Vagus nerve, cranial nerve 10, through its auricular branch (Arnold's nerve) → supplies the concha and posteroinferior canal and is responsible for the well-known ear-canal cough reflex.

(The lesser occipital nerve, C2, also reaches the upper posterior surface, but the keyed answer focuses on the main four.)

Now compare the choices carefully. Notice that every option lists the SAME group - auriculotemporal nerve, greater auricular nerve, and cranial nerves 7 and 10 - only the order written and the abbreviation style differ between them. So this is not really a recall-of-nerves test; it is a recognise-the-complete-and-correctly-named-set test.

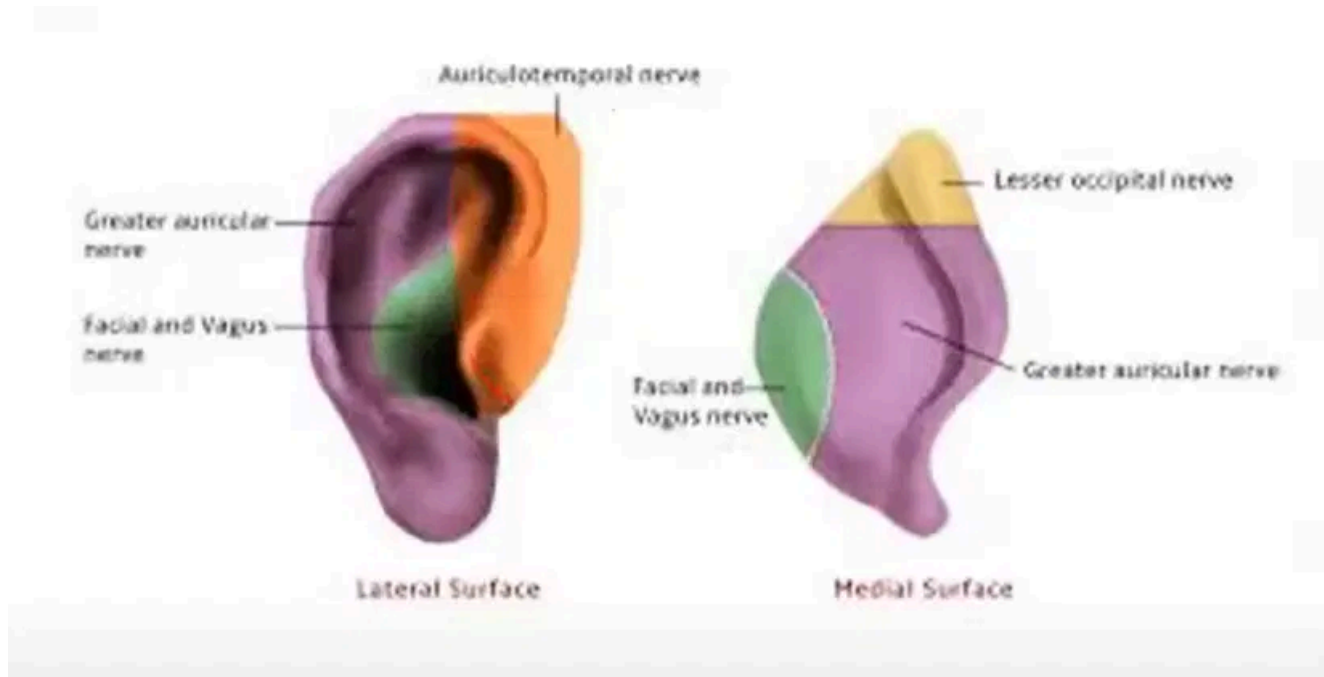
Selecting D: option D writes them as "ATN, 7 and 10, GAN," which is the full and correctly abbreviated set (auriculotemporal + facial + vagus + greater auricular nerve). Options A, B and C contain the same nerves in different sequences/spellings, but the examiner accepted D as the precisely worded complete list.

Answer: D.

Quick Tip: The pinna and ear canal are supplied by FOUR nerves - auriculotemporal (V3), greater auricular (C2,3), facial (7) and vagus (10, Arnold's). Pick the option naming all four.



75. In head impulse test, following finding seen in right vestibular neuritis:



- (A) On rotating head to right, left saccade
- (B) On rotating head to left, right saccade
- (C) On rotating head toward right, right saccade
- (D) On rotating to the left, left saccade

Correct Answer: (A) On rotating head to right, left saccade

SOLUTION:

Think mechanism-first about the reflex being probed. The head impulse test checks the vestibulo-ocular reflex (VOR) of one horizontal canal at a time. Key rule: a quick head turn in a given direction stresses the horizontal canal on the SAME side as the turn. A healthy canal instantly drives the eyes the opposite way so gaze stays locked on the target.

Put the lesion in: in right vestibular neuritis the right vestibular nerve / right horizontal canal is weak.

Now run the test toward the bad side. Turn the head rapidly to the RIGHT → you are loading the failing right canal → the VOR is too weak → the eyes cannot stay on target and get carried along with the head (to the right, off the target).

What does the brain do? It detects the eyes are off-target and fires a quick catch-up (corrective) saccade to drag the eyes BACK onto the target. Since the eyes drifted rightward, the corrective saccade must go LEFTWARD. Seeing this leftward refixation saccade after a rightward thrust is the positive sign and tells you the RIGHT side is diseased.

Filter the options:

- Turning to the right and seeing a left saccade → matches (this is option A).
- Turning to the left tests the GOOD left canal → normal VOR → no saccade (rules out B and D).
- A corrective saccade can never be in the same direction as the head turn; it is always opposite → rules out C (right turn, right saccade).

Answer: A.

Quick Tip: Turning the head toward the diseased side reveals a corrective catch-up saccade in the OPPOSITE direction. Right neuritis: turn right, saccade left.

• • •

76. Which of the following instrument insertion will be difficult:



- (A) Nasogastric Tube
- (B) LMA
- (C) Tracheostomy
- (D) Indirect Laryngoscopy

SOLUTION:

Decide by asking one question: which device must actually live inside the trachea? The accompanying chest X-ray points to a tracheal abnormality - the trachea is deviated, narrowed, or compressed by a mass. So the procedure that will struggle is the single one whose success depends on a normal, accessible tracheal lumen.

Sort the four options by the anatomical compartment each one targets:

- Nasogastric tube → travels nose to pharynx to OESOPHAGUS to stomach; it deliberately bypasses the airway, so the trachea is completely irrelevant to its passage.
- LMA (laryngeal mask airway) → a supraglottic airway that seats in the hypopharynx ABOVE the vocal cords and laryngeal inlet; it is designed NOT to enter the trachea.
- Indirect laryngoscopy → merely a way to LOOK at the larynx using a mirror or fibrescope; nothing physical is pushed down into the trachea, so a distorted trachea does not obstruct it.
- Tracheostomy → a surgical opening made DIRECTLY into the trachea with a tube placed in the tracheal lumen itself.

Apply the radiographic abnormality. A trachea that is shifted off the midline, narrowed by stenosis, or squeezed by a mass makes locating and cannulating the tracheal lumen technically hard and potentially hazardous (risk of false passage and bleeding). Of the four manoeuvres, this difficulty falls only on the tracheostomy.

So the three non-tracheal manoeuvres stay comparatively easy: the NGT uses the oesophageal route, the LMA seats supraglottically, and indirect laryngoscopy only visualises - none of them depends on the deranged trachea. The procedure requiring direct cannulation of the abnormal trachea - tracheostomy - is therefore the difficult one.

Answer: C.

Quick Tip: The X-ray shows a tracheal problem. Which procedure must enter the tracheal lumen itself? That is the one made difficult.

• • •

77. A singer presents with problem in high pitch. On examination bowing of one side vocal cord. Which of the following muscle is affected?

- (A) Posterior cricoarytenoid
- (B) Lateral cricoarytenoid
- (C) Cricothyroid
- (D) Thyroarytenoid

Correct Answer: (C) Cricothyroid

SOLUTION:

Reason from the physiology of pitch. Pitch is set by how tense and how long the vocal folds are: tighter, longer folds vibrate faster and produce a higher note. So a singer who suddenly cannot reach high notes has lost the muscle that TENSES and LENGTHENS the vocal cord.

Which muscle does that? Among the intrinsic laryngeal muscles, the cricothyroid is the dedicated tensor - it rocks the thyroid cartilage forward and downward on the cricoid, which stretches and elongates the vocal fold and thereby raises pitch. It is also unique anatomically: it is the ONLY intrinsic laryngeal muscle supplied by the external branch of the superior laryngeal nerve; every other intrinsic laryngeal muscle is supplied by the recurrent laryngeal nerve.

Explain the bowing sign. If the cricothyroid is weak or paralysed (often from external superior laryngeal nerve injury, e.g. after thyroid surgery), the cord on that side cannot be pulled taut, so it loses its straight tense edge, sags and looks

bowed on laryngoscopy. Functionally the patient loses high-frequency phonation and tires easily on sustained singing - exactly this professional singer's complaint.

Quick functional sort of the distractors:

- Posterior cricoarytenoid = the sole ABDUCTOR (opens the glottis for breathing). Its job is airway, not tensing for pitch.
- Lateral cricoarytenoid = ADDUCTOR (closes the glottis). Again, not the tensor.
- Thyroarytenoid / vocalis = relaxes and shortens the cord and acts as a weak adductor and fine-tuner; it is the functional opposite of a high-pitch tensor.

Only the cricothyroid stretches the cord for high pitch, so its dysfunction is the single explanation that accounts for BOTH the bowing seen on examination and the high-pitch difficulty reported.

Answer: C.

Quick Tip: High pitch needs a taut, lengthened cord. Which intrinsic laryngeal muscle is the tensor (supplied by the external laryngeal nerve)?

• • •

78. A 45-year-old male presents with breathlessness and undergoes a CT scan of the paranasal sinuses (PNS). Which sinus is obstructed?



- (A) Maxillary
- (B) Frontal
- (C) Sphenoid
- (D) Ethmoid

Correct Answer: (A) Maxillary

SOLUTION:

Use the basic rule of sinus CT first: air is black, and a blocked (obstructed) sinus that fills with mucus or soft tissue turns grey/white and loses its normal black air shadow, sometimes showing an air-fluid level.

Now identify the sinuses by position on a coronal cut:

- Maxillary sinuses = the big paired pyramidal spaces low down, just under the

orbits and beside the nose - the largest sinuses you see on the standard coronal image.

- Frontal sinuses = up in the forehead, above the orbits.
- Ethmoid air cells = the little honeycomb cells sandwiched between the two eyes.
- Sphenoid = a single central air space sitting far back, behind the ethmoids.

Match to the picture. The film shows the large lateral antral space whitened out (filled with material) instead of being black with air - that is the maxillary sinus, and its drainage pathway through the osteomeatal complex is blocked, so it is the obstructed one.

Exclusion check: the frontal (above orbit), ethmoid (small inter-orbital cells) and sphenoid (deep midline, posterior) sinuses are in different locations and are not the big opacified cavity displayed.

Answer: A (Maxillary).

Quick Tip: On CT-PNS, air is black and a blocked sinus opacifies (turns white). The large pyramidal sinus under each orbit is the maxillary - look for which one is whited out.

• • •

79. Intraoperatively which stain is used to view following lesion:



- (A) AgNo₃
- (B) Toluidine Blue
- (C) Congo red
- (D) Methylene blue

Correct Answer: (B) Toluidine Blue

SOLUTION:

Approach this as "how does a surgeon make an invisible margin visible during an oral cancer operation?"

The picture is of a suspicious red, eroded patch of oral mucosa concerning for dysplasia or early oral squamous cell carcinoma. The surgeon wants a quick chair-side / table-side test that lights up abnormal cells so the resection is complete.

The principle is **vital staining**: a dye applied directly to living mucosa that is selectively taken up by malignant/dysplastic cells. Cancerous cells divide rapidly, contain far more nuclear DNA/RNA, and have loosened (widened) intercellular junctions, so a nucleic-acid-binding dye penetrates and is retained there but rinses off healthy tissue.

The dye that does exactly this in the mouth is **toluidine blue**. It is metachromatic and binds nucleic acids, so dysplastic areas turn a deep blue while normal mucosa stays pale, giving a clear visual map.

Quickly ruling out the rest: silver nitrate (AgNO_3) is a cauterising/haemostatic agent, not a marker dye; Congo red is a laboratory histochemical stain for amyloid (green birefringence in polarised light); methylene blue is mainly used for sentinel lymph-node localisation and Barrett oesophagus chromoendoscopy. None of these is the intraoral lesion-mapping stain.

Therefore the stain used to view this oral lesion intraoperatively is **Toluidine Blue (option B)**.

Quick Tip: Think of the metachromatic vital dye that dysplastic oral mucosa retains during surgery.

• • •

80. Gag reflex will be absent in which nerve injury?

- (A) 5 and 10
- (B) 9 and 10
- (C) 10 and 12
- (D) 7 and 9

Correct Answer: (B) 9 and 10

SOLUTION:

Solve this by drawing the reflex arc rather than memorising option pairs.

Every reflex needs a sensor (afferent), a centre, and an effector (efferent). For the gag reflex:

Afferent limb — a foreign touch on the back of the throat (posterior pharynx and posterior one-third of the tongue) is detected and the signal runs in the

glossopharyngeal nerve, CN IX.

Centre — the nucleus solitarius/nucleus ambiguus in the medulla.

Efferent limb — the medulla fires back along the **vagus nerve, CN X**, contracting the pharyngeal constrictor muscles and lifting the soft palate, producing the gag.

So the arc is built from CN IX and CN X. If either of these is damaged the loop is broken and the gag reflex disappears. That gives the pair **9 and 10**.

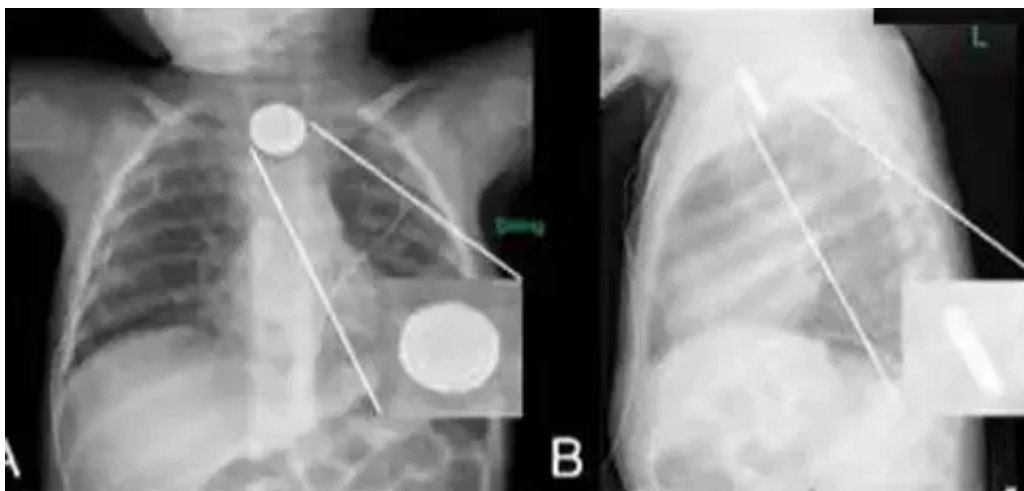
Cross-checking the wrong pairs: CN V is sensation to the face and front of the tongue, CN VII is facial movement and front-tongue taste, and CN XII only moves the tongue body — none of them forms part of the pharyngeal reflex circuit, so any option containing 5, 7 or 12 is wrong.

The gag reflex is therefore lost with injury to cranial nerves **9 and 10 (option B)**.

Quick Tip: Sensory limb = CN IX, motor limb = CN X; both must be intact for a gag.

• • •

81. In given x-ray which of the following sign is seen:



- (A) Double ring sign
- (B) String sign
- (C) Steeple sign
- (D) Thumb sign

Correct Answer: (A) Double ring sign

SOLUTION:

Tackle this as pure pattern-recognition of named radiological signs.

First describe what is actually visible: on this radiograph a structure is outlined by air on more than one surface, giving two concentric rings — one ring inside another. The named sign for two concentric lucent rings on a single radiograph is the **double ring (double-wall) sign**.

Now keep each named sign tied to its home disease and modality, and only one will fit a plain radiograph showing concentric rings:

- **Double ring sign** → two concentric rings on a plain film (e.g. free intraperitoneal air outlining both walls of bowel, the Rigler appearance). This is what is shown.
- **String sign** → a barium contrast study, not a plain x-ray: a hair-thin contrast line through a narrowed lumen in Crohn disease or pyloric stenosis.
- **Steeple sign** → AP soft-tissue neck film in croup, with the trachea narrowing to a church-spire point.
- **Thumb sign** → lateral neck film in epiglottitis, with the swollen epiglottis shaped like a thumb.

Since the picture shows concentric rings on a single radiograph and not airway narrowing or a contrast column, the only consistent answer is the **Double ring sign (option 1)**.

Quick Tip: Two concentric rings on one plain film = the double-ring (double-wall) sign.

82. Male not responding to O₂, diagnosis ARDS. What is the role of IL-8 in ARDS?

- (A) Endothelial cell activation
- (B) Requirement of neutrophil
- (C) Macrophage activation
- (D) Promote surfactant production

Correct Answer: (B) Requirement of neutrophil

SOLUTION:

Mechanism-first approach.

Start from the question “what is the single defining function of IL-8?” IL-8 belongs to the CXC chemokine family. CXC chemokines with the ELR motif are specialised to attract **neutrophils**. So before considering ARDS at all, IL-8 = neutrophil chemoattractant.

Applying it to ARDS.

ARDS injury is fundamentally neutrophil-mediated. Activated alveolar macrophages and injured epithelium pour out IL-8 into the alveoli. This IL-8 gradient pulls neutrophils across the capillary endothelium into the air spaces, where they degranulate and release elastase and oxidants, tearing the alveolar-capillary membrane. The result is leaky, protein-rich edema and the refractory hypoxia that does not improve with oxygen alone.

Therefore the link being tested.

IL-8 is required to bring neutrophils to the lung — matching “requirement of neutrophil.” (*answer = B*)

Eliminating distractors.

Endothelial activation → TNF-α / IL-1 territory, not IL-8.

Macrophage activation → macrophages secrete IL-8; they are the source, not the recipient, and activation is an IFN-γ phenomenon.

Surfactant production → a type-II pneumocyte function that is lost in ARDS, never promoted by a pro-inflammatory chemokine.

Conclusion: IL-8 drives neutrophil recruitment — Option B.

Quick Tip: IL-8 (CXCL8) is the classic chemokine that calls in one specific leukocyte — think which cell does the alveolar damage in ARDS.

• • •

83. A patient presents with the finding as shown. What is the best investigation for Wilson?

- (A) Urine Copper
- (B) Hepatic copper
- (C) S. Ceruloplasmin
- (D) MRI Brain

Correct Answer: (B) Hepatic copper

SOLUTION:

Diagnostic-hierarchy approach.

The question hinges on the word “best.” For Wilson disease, tests sit in a hierarchy from screening to confirmatory.

Screening tier: serum ceruloplasmin (low) and 24-hour urinary copper (high). Both are sensitive flags but neither is fully specific — ceruloplasmin is an acute-phase reactant and can be normal; urinary copper rises in other cholestatic liver diseases too.

Supportive/imaging tier: slit-lamp for KF rings and MRI brain showing “face-of-the-giant-panda” / basal-ganglia signal. These confirm organ involvement but not the metabolic defect.

Confirmatory tier: *hepatic copper content* measured on liver biopsy (dry-weight copper, usually $> 250 \mu\text{g/g}$). This directly demonstrates pathological copper overload in the target organ and is the gold standard / single best diagnostic test.

Decision.

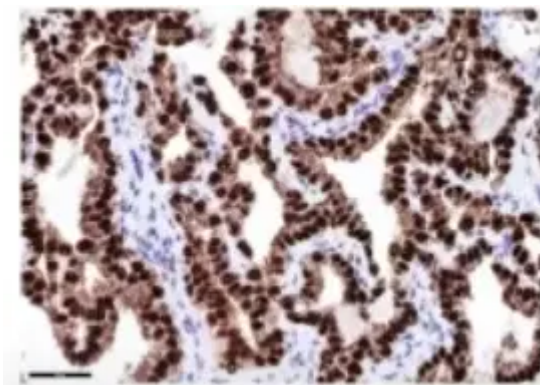
Because the stem asks for the BEST (most definitive) investigation, we climb to the confirmatory tier → hepatic copper. Therefore the correct choice is Option B.

Why not the rest: Urine copper and ceruloplasmin are screening, not confirmatory; MRI brain is supportive imaging only. Hepatic copper wins.

Quick Tip: Screening tests (ceruloplasmin, urine copper) flag Wilson disease, but the gold-standard confirmation is measured directly in the organ that overloads first.

• • •

84. Which tumor is positive for TTF-1?



- (A) Sq (Squamous cell carcinoma)
- (B) Small (Small cell carcinoma)
- (C) Adeno Ca (Adenocarcinoma)
- (D) Carcinoid

Correct Answer: (C) Adeno Ca (Adenocarcinoma)

SOLUTION:

Marker-to-tissue reasoning.

Begin with what TTF-1 (NKX2-1) marks: cells of **thyroid follicular** and **lung (alveolar type-II / Clara)** lineage. So in a thoracic tumour, a TTF-1-positive result points to a tumour that recapitulates lung glandular epithelium.

Walk the four options through this filter:

- Squamous cell carcinoma — arises from bronchial squamous metaplasia, expresses **p40** / **p63** / **CK5/6**, and is TTF-1 **NEGATIVE**.
- Adenocarcinoma — derived from glandular/alveolar epithelium, the very lineage TTF-1 marks → TTF-1 **POSITIVE**. The image shows gland-forming tumour, matching this.
- Small cell carcinoma — neuroendocrine; often TTF-1 positive too, but the morphology and the classic single-best teaching answer point to adenocarcinoma.
- Carcinoid — low-grade neuroendocrine tumour, marked by chromogranin/synaptophysin, generally TTF-1 negative.

Resolution.

The tumour that is canonically and most usefully TTF-1 positive among these — and the one separating lung primary from squamous/metastatic mimics — is adenocarcinoma. (**answer = Option C**)

Takeaway: TTF-1 (+) + glandular morphology = lung adenocarcinoma; squamous and carcinoid are the TTF-1 (–) distractors.

Quick Tip: TTF-1 marks thyroid and lung type-II pneumocyte lineage — think which lung cancer forms glands and is the classic positive.

• • •

85. Vitamin to be supplemented after gastrectomy?

- (A) Vit A
- (B) Vit C
- (C) Vit B12
- (D) Vit D

Correct Answer: (C) Vit B12

SOLUTION:

Trace the missing molecule.

Ask: what does the stomach uniquely provide for vitamin absorption? Answer: **intrinsic factor** from parietal cells.

Consequence of gastrectomy.

No stomach → no parietal cells → no intrinsic factor. Which vitamin absolutely needs intrinsic factor to be absorbed in the terminal ileum? Only *Vitamin B₁₂*. So removing the stomach selectively cuts off B12 uptake.

Time course explains the clinical need.

Hepatic B12 stores cover roughly 2–4 years, so deficiency (megaloblastic anaemia, glossitis, subacute combined degeneration of the cord) appears late. To prevent this, patients receive lifelong B12 — typically intramuscular, because the IF-dependent oral pathway no longer works.

Screening the distractors against the “needs intrinsic factor?” test:

Vit A — no (fat-soluble, small-bowel absorption).

Vit C — no (water-soluble, small-bowel absorption).

Vit D — no (fat-soluble, bile-dependent, small bowel).

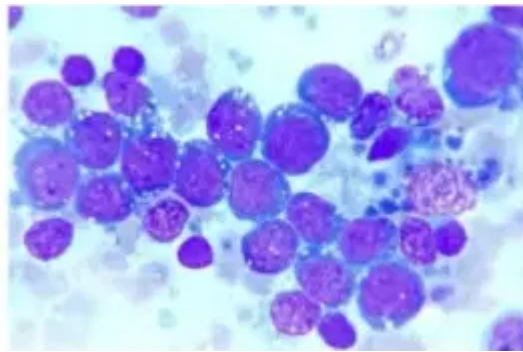
Vit B12 — YES.

Hence the supplement after gastrectomy is Vitamin B12 — Option C.

Quick Tip: The stomach makes intrinsic factor; only one vitamin needs intrinsic factor to be absorbed.



86. 10/m presents with CERVICAL L. node+, Surface Ig +, CD34−;, 5−;, 23−;, tdt −;, cd 10+, 19+:



- (A) Burkitts Lymphoma
- (B) DLBCL
- (C) Anaplastic
- (D) B-ALL

Correct Answer: (A) Burkitts Lymphoma

SOLUTION:

Mature-vs-precursor first.

The decisive split in B-cell neoplasms is whether the cell is a **precursor (immature)** or a **mature** B-cell. Three markers settle it here:

- **TdT** negative
- **CD34** negative
- surface **Ig** POSITIVE

This triad is definitively **mature B-cell**. That single deduction eliminates B-ALL, because B-ALL is a precursor tumour that is TdT+, CD34+ and surface-Ig-.

Now place the mature B-cell tumour.

Remaining mature B markers: **CD19+** (B lineage) and **CD10+** (germinal-centre origin). A germinal-centre, CD10+ mature B-cell, high-grade lymphoma in a 10-year-old boy with a fast-growing cervical node, and a smear showing a starry-sky pattern of medium-sized cells with deep-blue vacuolated cytoplasm, is the textbook picture of **Burkitt lymphoma**.

Clearing the remaining distractors.

DLBCL — mature B too, but an older-age, non-classic-childhood entity and not the canonical CD10+ starry-sky tumour intended here.

Anaplastic (ALCL) — a CD30+/ALK+ T or null-cell lymphoma; it would NOT be CD19/CD10/sIg positive.

Conclusion: mature germinal-centre B phenotype + child + starry-sky morphology → Burkitt lymphoma, Option A.

Quick Tip: TdT–, CD34– and surface Ig+ mean a MATURE B-cell — that rules out the precursor leukaemia; then CD10+ germinal-centre + child + starry-sky points to one high-grade lymphoma.

• • •

87. Most common mutation in Papillary cell CA?

- (A) BRAF V600E
- (B) RET
- (C) MET
- (D) RAS

Correct Answer: (A) BRAF V600E

SOLUTION:

Pathway-centred view.

Papillary thyroid carcinoma is a MAPK-pathway disease: nearly every case is driven by a mutually exclusive lesion that switches on RAS→RAF→MEK→ERK signalling. The candidates are BRAF, RET/PTC fusions and RAS — the trick is ranking them by frequency.

Ranking by prevalence in classic PTC:

1. **BRAF V600E** — the dominant single mutation, present in about **40-60%** of cases; a constitutively active serine/threonine kinase that drives ERK signalling and confers a more aggressive phenotype.
2. RET/PTC rearrangement — second, enriched in radiation-exposed and

paediatric tumours.

3. RAS — more linked to follicular-pattern thyroid tumours than classic PTC.

So the “most common” answer is BRAF V600E — Option A.

Disqualifying the rest with disease-association cues:

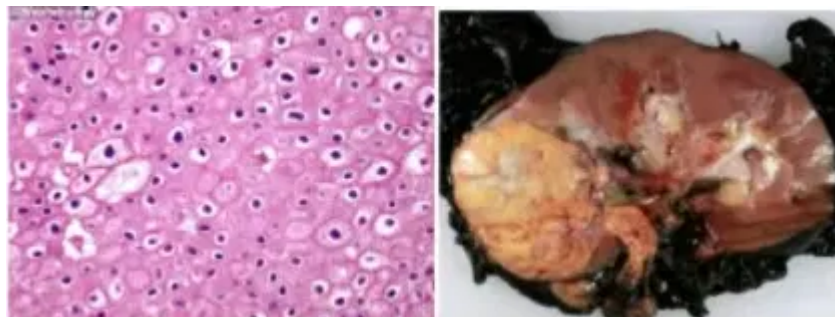
- RET as a germline POINT mutation → medullary carcinoma / MEN-2, a different tumour.
- MET → think hereditary papillary RENAL cell carcinoma, not thyroid.
- RAS → follicular carcinoma / follicular-variant PTC, lower frequency in classic PTC.

Final: BRAF V600E is the commonest mutation of papillary thyroid carcinoma.

Quick Tip: Papillary thyroid cancer is a MAPK-pathway tumour — the single most frequent driver is a V600E kinase point mutation.

• • •

88. Renal mass, haematuria, flank pain:



- (A) Clear cell
- (B) Papillary
- (C) Chromophobic
- (D) Belini (Collecting duct / Bellini)

Correct Answer: (C) Chromophobic

SOLUTION:

Pattern-recognition route.

The stem gives the textbook RCC triad — flank pain + haematuria + renal mass — so this is renal cell carcinoma; the task is purely to subtype it from the gross and microscopic pictures.

Build a quick morphology key:

- Clear cell → sheets of cells with *optically clear* (lipid/glycogen) cytoplasm and a fine “chicken-wire” capillary network; commonest subtype.
- Papillary → finger-like papillae cored by fibrovascular stalks with foamy macrophages ± psammoma bodies.
- Chromophobe → large pale-to-eosinophilic polygonal cells with **thick, sharply outlined plant-cell-like membranes, perinuclear haloes and wrinkled “raisinoid” nuclei**; tan-brown well-circumscribed gross mass.
- Bellini (collecting duct) → rare, central/medullary, infiltrative, highly aggressive.

Apply the key.

The image features matching prominent cell membranes, perinuclear clearing and raisinoid nuclei correspond to the **chromophobe** entry, and this agrees with the official key (Ans. C). Hence *answer = Option C*.

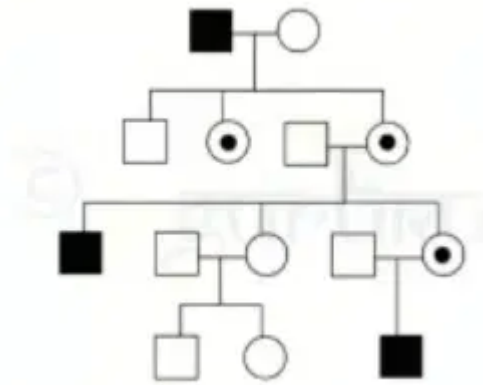
Why not the others: no optically clear cytoplasm (rules out clear cell), no fibrovascular papillae/foam cells (rules out papillary), not a rare infiltrative medullary mass (rules out Bellini).

Conclusion: Chromophobe RCC.

Quick Tip: Flank pain + haematuria + mass = RCC; then subtype from the slide — look for plant-cell-like thick membranes, perinuclear haloes and raisinoid nuclei.



89. Which disease will show the following mode of inheritance?



- (A) Wiskott (Wiskott-Aldrich syndrome)
- (B) Wilson
- (C) Prader willi
- (D) Achondroplasia

Correct Answer: (A) Wiskott (Wiskott-Aldrich syndrome)

SOLUTION:

Decode-the-pedigree approach.

First name the inheritance pattern from the diagram, then map it to a disease.

Pedigree clues present:

- Affected individuals are essentially all **male** (filled squares).
- Disease passes through **unaffected carrier females** (dotted circles).
- There is **no male-to-male transmission** (an affected father never passes it to his son).

This combination is diagnostic of **X-linked recessive** inheritance — affected males inherit the mutant X from carrier mothers, and fathers pass their X only to daughters (who become carriers).

Now test each option for X-linked recessive status:

- Wiskott-Aldrich → X-linked recessive (WAS gene) — MATCH.
- Wilson → autosomal recessive (ATP7B) — both sexes, sibling clustering; no.
- Prader-Willi → genomic imprinting / paternal 15q deletion; not X-linked; no.
- Achondroplasia → autosomal dominant (FGFR3); vertical, male-to-male

possible; no.

Only Wiskott-Aldrich fits an X-linked recessive pedigree (*answer = Option A*, matching the key).

Memory aid: all-affected-male + carrier-female transmission + no father-to-son = X-linked recessive → here, Wiskott-Aldrich.

Quick Tip: Only males affected, passed by carrier females, no father-to-son transmission = X-linked recessive — which listed disease is X-linked?

• • •

90. Young male died during exercise. History of similar history in a sibling. On gross morphology: septal thickening was seen. Most common cause of death?

- (A) DCM
- (B) RCM
- (C) HOCM
- (D) Viral Myocarditis

Correct Answer: (C) HOCM

SOLUTION:

Mechanism-first approach. Start from the autopsy finding that anchors the diagnosis: *septal thickening*. Asymmetrical hypertrophy of the interventricular septum is the structural signature of Hypertrophic Cardiomyopathy. Layer onto this the two clinical anchors — sudden death triggered by exertion and a similarly affected sibling — and the genetics fall into place: this is an autosomal dominant sarcomere disease (commonly β -myosin heavy chain or myosin-binding protein C mutations).

Why exercise kills in HOCM: the thickened septum narrows the left ventricular outflow tract, and the chaotically arranged (disarrayed) myocyte fibres form a substrate for re-entrant ventricular tachyarrhythmias. Under the catecholamine

surge of exercise, contractility rises and outflow obstruction worsens, tipping the heart into ventricular fibrillation — the leading cause of sudden cardiac death in young athletes.

Eliminating the rest: a dilated, baggy heart points to *Dilated Cardiomyopathy*; a rigid ventricle with prominent atria points to *Restrictive Cardiomyopathy*; a flabby heart with inflammatory infiltrate points to *Viral Myocarditis*. None of these produce focal asymmetrical septal hypertrophy in a young patient with a positive family history.

Hence the answer is **HOCM (Option C)**.

Quick Tip: Young athlete + sudden death on exertion + family history + asymmetrical septal hypertrophy = the classic sarcomeric cardiomyopathy.

• • •

91. Which is not true?

- (A) vWD type 1 is mostly severe ds in children
- (B) vWD type 3 mostly severe ds in children
- (C) vWD type 2 is more related activity rather than levels
- (D) vWD type 3 has severely low VWD

Correct Answer: (A) vWD type 1 is mostly severe ds in children

SOLUTION:

Classify, then test each line against the classification. The cleanest way to answer a "which is NOT true" vWD question is to first fix the three subtypes in mind: Type 1 = *partial* quantitative deficiency and the mildest, commonest form; Type 2 = *qualitative* (dysfunctional vWF) so activity is hit harder than antigen level; Type 3 = *complete* quantitative deficiency, the most severe, with very low factor VIII too.

Now read each option:

- "Type 1 is mostly severe disease in children" — contradicts the rule that Type 1 is the *mild* form. This is the false statement.
- "Type 3 is mostly severe disease in children" — matches Type 3 being the severe, early-presenting form. True.
- "Type 2 relates more to activity than levels" — matches Type 2 being a functional defect, where ristocetin cofactor activity drops out of proportion to antigen. True.
- "Type 3 has severely low vWF" — matches the near-absent vWF of Type 3. True.

The single line that breaks the classification is the claim about Type 1 being severe. Therefore the statement that is NOT true is **Option A**.

Quick Tip: Type 1 vWD is the commonest and mildest form — any statement calling it "severe" is the false one.

• • •

92. Child. Sunlight causes eruptions, diagnosed as DNA repair defect. Which defect can it be?

- (A) Nucleotide excision
- (B) Base excision repair
- (C) Mismatch repair defect
- (D) Recombination defect

Correct Answer: (A) Nucleotide excision

SOLUTION:

Pattern-match the lesion to the pathway. The key is to translate the clinical clue into the molecular lesion. "Sunlight causes eruptions" means ultraviolet exposure, and UV light's signature DNA damage is the *pyrimidine dimer* — a bulky, helix-distorting lesion formed between adjacent thymines.

Each DNA repair system handles a specific type of damage:

- Bulky, helix-distorting lesions (UV dimers, chemical adducts) → repaired by

Nucleotide Excision Repair.

- Small single-base alterations (oxidised/deaminated bases) → Base Excision Repair.
- Replication mismatches and indel loops → Mismatch Repair.
- Double-strand breaks → Homologous Recombination / Non-homologous end joining.

Because the patient's damage is UV-induced pyrimidine dimers, the failed pathway must be Nucleotide Excision Repair. The disease itself is *Xeroderma Pigmentosum*, where inability to excise UV dimers leads to extreme sun sensitivity and early skin cancers.

Therefore the correct defect is **Nucleotide Excision (Option A)**.

Quick Tip: Sunlight → UV → thymine dimers → these bulky lesions are cleared by nucleotide excision repair (defect = Xeroderma Pigmentosum).

• • •

93. 18/M. Hepatosplenomegaly, Hb 7 gm%, LN+, WBC 50K, Platelets 30, petechiae, purpura, fatigue. What is the most appropriate management?

- (A) Cytarabine + iso?
- (B) IVIG x 2 days
- (C) Prednisolone + Vinblastine
- (D) Radiotherapy to LN

Correct Answer: (C) Prednisolone + Vinblastine

SOLUTION:

Diagnosis-driven approach. Before choosing a drug, name the disease. The blood counts tell a clear story: high WBC (50,000) with simultaneous anaemia (Hb 7) and thrombocytopenia (platelets 30,000, hence the petechiae and purpura). When one lineage is grossly elevated while the other two are suppressed in a young person, the marrow is being crowded out by a proliferating

clone — acute leukaemia. The added findings of *lymphadenopathy* and *hepatosplenomegaly* in an 18-year-old point specifically to **Acute Lymphoblastic Leukaemia**, where lymph node and visceral infiltration are typical.

Management then follows directly from the diagnosis. ALL induction is built on a steroid plus a vinca alkaloid (with asparaginase and an anthracycline). Of the four choices, only **Prednisolone + Vinblastine** matches this lymphoid-leukaemia induction backbone.

Ruling out the distractors quickly:

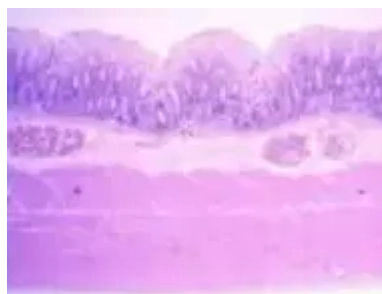
- Cytarabine-based therapy belongs to *AML*, not *ALL*.
- IVIG is reserved for *immune thrombocytopenic purpura*, which shows isolated low platelets — not a high WBC with organomegaly.
- Nodal radiotherapy is a focal treatment for lymphoma and is useless against marrow-based, blood-borne leukaemia.

So the most appropriate management is **Prednisolone + Vinblastine (Option C)**.

Quick Tip: Young patient, high WBC with anaemia + thrombocytopenia + nodes + hepatosplenomegaly = ALL; pick the steroid + vinca alkaloid induction.

• • •

94. A baby failed to pass meconium. In this disease, the structures absent are absent in which of the following layer(s)?



Select the correct answer from the given below cord:

- (A) 1 n 2
- (B) 2 n 3
- (C) 3 n 4
- (D) 1 n 4

Correct Answer: (B) 2 n 3

SOLUTION:

Anatomy-of-the-gut-wall approach. The clinical clue (a newborn not passing meconium) names the disease — Hirschsprung disease — in which ganglion cells of the enteric nervous system fail to develop in the distal colon. The real question is purely anatomical: *in which layers of the bowel wall do those ganglion cells normally live?*

Walk through the four layers of the gut from inside out:

1. Mucosa / epithelium — lining only, no neural plexus.
2. Submucosa — houses the *submucosal (Meissner's) plexus*.
3. Muscularis externa — between its circular and longitudinal muscle coats lies the *myenteric (Auerbach's) plexus*.
4. Serosa — outer mesothelial covering, no plexus.

So the enteric ganglia are confined to layers 2 (submucosa) and 3 (muscular). In Hirschsprung disease both Meissner's and Auerbach's plexuses are aganglionic, which is why both these layers are involved.

The matching option is **2 n 3 (Option B)**; choices that involve epithelium (1) or serosa (4) cannot be right because those layers never contain ganglion cells.

Quick Tip: Hirschsprung = absent ganglion cells of Meissner's (submucosa) and Auerbach's (muscularis) plexuses → layers 2 and 3.



95. Muscle responsible for ptosis in Horner syndrome:-



- (A) Orbicularis oculi
- (B) Levator palpebrae
- (C) Horner muscle
- (D) Mullers muscle

Correct Answer: (D) Mullers muscle

SOLUTION:

Trace the innervation, then the muscle follows. Horner syndrome is fundamentally a *sympathetic denervation* of the eye. So the muscle responsible for any feature of Horner syndrome must be a sympathetically-supplied muscle.

Apply this to the eyelid. The lid is held up by two elevators:

- The big skeletal elevator, *Levator Palpebrae Superioris*, runs on the oculomotor nerve (CN III) — not sympathetic, so it is spared in Horner syndrome.
- The small smooth-muscle elevator, the superior tarsal muscle = *Müller's muscle*, runs on sympathetic fibres.

When sympathetic input is cut, only Müller's muscle weakens. Because it contributes just a few millimetres of lift, its loss gives the characteristic *mild/partial* ptosis of Horner syndrome (in contrast to the severe, complete ptosis of a CN III palsy that takes out the levator).

The remaining options fail on function or nerve supply: *orbicularis oculi* closes the lid (facial nerve); the so-called *Horner muscle* is the tear-pump part of orbicularis, unrelated to elevation. Therefore the muscle producing the ptosis is **Müller's muscle (Option D)**.

Quick Tip: Horner = sympathetic loss; the only sympathetically-supplied lid elevator is Müller's (superior tarsal) muscle, giving partial ptosis.

• • •

96. A patient presented to eye OPD after 3 years of cataract surgery. Slit lamp finding was given. What is the likely diagnosis?



- (A) PCO
- (B) Bullous keratopathy
- (C) Phakic glaucoma
- (D) Lens subluxation

Correct Answer: (A) PCO

SOLUTION:

Time-course reasoning. The single most useful clue here is "3 years after cataract surgery." Complications of cataract surgery can be grouped by timing, and the dominant *late* complication — appearing months to years later — is Posterior Capsular Opacification (the after-cataract).

Why it happens: during modern cataract surgery the posterior capsule is deliberately preserved to seat the intraocular lens. Lens epithelial cells left at the equator slowly proliferate and creep across this capsule, forming Elschnig pearls and fibrotic wrinkles. On the slit lamp these appear as a hazy, pearly, sometimes "fish-egg" membrane sitting behind the IOL, and the patient complains of blurred vision and glare returning years after an initially successful operation — precisely this presentation. Definitive treatment is a quick Nd:YAG laser posterior capsulotomy.

Discarding the alternatives: *bullous keratopathy* is an early-to-intermediate corneal endothelial failure showing corneal oedema, not a capsular finding; *phakic glaucoma* is not a recognised post-cataract diagnosis and concerns pressure, not capsular opacity; *lens subluxation* cannot occur to a natural lens that was already removed at surgery.

The late post-operative slit-lamp opacity behind the IOL is therefore **PCO (Option A)**.

Quick Tip: Vision drop years after cataract surgery with a pearly/wrinkled membrane behind the IOL = posterior capsular opacification, treated by Nd:YAG capsulotomy.

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97. Following perimetry image is suggestive of?



- (A) Extension of blind spot
- (B) Arcuate scotoma
- (C) Reinee step defect
- (D) Altitude Anopia

Correct Answer: (B) Arcuate scotoma

SOLUTION:

Read the shape of the field loss. The trick to a perimetry question is to match the *geometry* of the dark (lost) area to a named defect. Here the printout shows a curved, comma- or arc-shaped band of depressed sensitivity sweeping from the

region of the blind spot around the central fixation point.

This curved shape is dictated by retinal anatomy: ganglion-cell axons sweep in an *arcuate* path (the Bjerrum arcuate bundles) around the macula on their way to the optic disc. When these bundles are damaged — the hallmark of glaucoma — the resulting field loss copies their curved course, giving an **arcuate scotoma**. It is the textbook early glaucomatous visual-field defect.

Contrast with the distractors:

- *Enlarged blind spot* is a focal patch hugging the blind spot (papilloedema), not a long arc.
- *Roenne's (Reinee) nasal step* is specifically a horizontal "step" mismatch in the nasal field, a more advanced glaucoma sign, not the broad curve here.
- *Altitudinal field loss* wipes out a whole upper or lower half along the horizontal line (ischaemic optic neuropathy), which is not the curved pattern depicted.

So the perimetry is suggestive of an **Arcuate scotoma (Option B)**. (The paper did not print an answer key for this question, so this is the best-supported derived answer from the glaucomatous arcuate pattern.)

Quick Tip: A curved comma-shaped field defect arching from the blind spot around fixation is the classic glaucomatous arcuate scotoma.

• • •

98. A patient came with nyctalopia. Fundus image given below, what will be the diagnosis?



- (A) Retinitis pigmentosa
- (B) Vit A deficiency
- (C) Retinal detachment
- (D) Diabetic retinopathy

Correct Answer: (A) Retinitis pigmentosa

SOLUTION:

Approach by mechanism of night blindness. Night vision depends on functioning rod cells. When rods fail, the patient cannot see in dim light (nyctalopia). The two commonest causes are an inherited rod degeneration (retinitis pigmentosa) and lack of vitamin A, which is the substrate for rhodopsin. The fundus photograph is the decider.

What the fundus tells us. Retinitis pigmentosa produces a pathognomonic picture: dark bony-spicule pigment clumps scattered in the mid-periphery, markedly narrowed (attenuated) retinal vessels, and a waxy, pale optic disc. This image shows exactly that pattern.

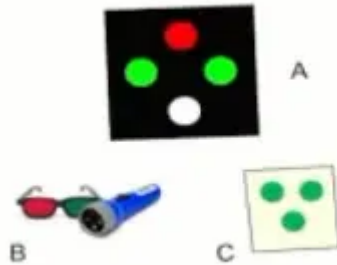
Why the answer is retinitis pigmentosa. It is a progressive hereditary rod-cone dystrophy in which rods die first; this explains the early lifelong night blindness and the ring/tunnel-vision field defect, and the degenerating retinal pigment epithelium deposits the characteristic spicular pigment.

Ruling out the other choices. Vitamin A deficiency gives nyctalopia but with xerophthalmia signs (Bitot spots, conjunctival/corneal xerosis), not retinal spicules. A detachment looks like a raised grey retinal fold with acute curtain-like loss. Diabetic retinopathy shows haemorrhages, exudates and new vessels with gradual visual blurring, not pure night blindness.

Hence the diagnosis is Retinitis pigmentosa (Option A).

Quick Tip: Night blindness plus bony-spicule pigment, thin vessels and a waxy disc on fundus = a hereditary rod-cone dystrophy.

99. Patient was shown image A along with image B. Image C was visualised. Likely diagnosis?



- (A) Lt eye suppression
- (B) Rt eye suppression
- (C) Crossed diplopia
- (D) Uncrossed diplopia

Correct Answer: (B) Rt eye suppression

SOLUTION:

Start from what the apparatus is. Red-green goggles plus a four-light panel is the Worth four-dot test, the classic bedside test for fusion, suppression and diplopia. Each coloured filter dissociates the two eyes by colour: the red-filtered eye sees only red, the green-filtered eye sees only green, and the bottom white light takes the colour of whichever eye is dominant.

Reading the possible responses. Normal fusion = 4 dots seen. Diplopia = 5 dots seen (the eyes are not fusing, so each eye sees its own lights — crossed in exotropia, uncrossed in esotropia). Suppression = only the lights of one colour are seen, because the brain ignores one eye's image. The number of dots therefore separates fusion, diplopia and suppression at a glance.

Applying it to image C. The patient perceives only the green lights and not the red one. Only-green response means the brain is suppressing the image from the eye wearing the red filter. As the red goggle is placed over the right eye, the suppressed eye is the right eye.

Eliminating the rest. Only-red dots would indicate left-eye suppression (red over right means green over left). A five-dot picture would have indicated diplopia (crossed for exotropia, uncrossed for esotropia), which is not what is seen.

Therefore the diagnosis is right eye suppression (Option B).

Quick Tip: Worth four-dot test: seeing only the green lights means the red-goggled eye is being suppressed — note which eye has the red filter.

• • •

100. A patient presents in eye OPD with this finding. What is it?



- (A) Dermoid
- (B) Lipodermoid
- (C) Pterygium
- (D) Papilloma

Correct Answer: (C) Pterygium

SOLUTION:

Recognise the pattern first. A wing-shaped triangle of vascular tissue running from the conjunctiva across the limbus onto the cornea is the textbook look of a pterygium ('pterygion' = little wing). It typically starts nasally because reflected UV light concentrates there, and is common in people with high sun, dust and wind exposure.

Why pterygium fits. Pathologically it is elastotic (actinic) degeneration of

subconjunctival tissue with fibrovascular proliferation that advances onto the cornea. The apex (head) points toward the pupil; the body lies over the limbus. Patients complain of a red, gritty eye and, if it reaches the visual axis, blurred vision or astigmatism. The photograph matches this exactly.

Contrast with the look-alikes.

- Dermoid: a congenital, raised, rounded, solid whitish mass straddling the limbus, sometimes with hairs — not a flat fold.
- Lipodermoid: a soft yellow fatty mass at the outer (temporal) canthus or fornix, separate from the cornea.
- Papilloma: a soft, pink, lobulated cauliflower-like surface growth, not a triangular limbal sheet.

Conclusion: the finding is a Pterygium (Option C).

Quick Tip: A triangular, wing-shaped fold of fibrovascular conjunctiva creeping from the nasal limbus onto the cornea is the giveaway.



101. In Wilson disease, the best choice of investigation is:

- (A) Serum copper
- (B) Ceruloplasmin
- (C) Hepatic copper estimation
- (D) Urine copper

Correct Answer: (B) Ceruloplasmin

SOLUTION:

Reason from the biochemistry. In Wilson disease the faulty ATP7B protein cannot attach copper onto apoceruloplasmin in the hepatocyte and cannot excrete copper into bile. The direct, measurable consequence in blood is a low ceruloplasmin, which is why it is the practical first investigation.

Why ceruloplasmin is 'best choice'. It is cheap, non-invasive, widely available and is decreased (< 20 mg/dL) in the great majority of patients. Used alongside a slit-lamp search for Kayser-Fleischer rings and a 24-hour urinary copper, it gives a robust working diagnosis without needing a biopsy.

Where the other options sit in the work-up.

- Serum total copper: usually low/normal and misleading, because most copper in blood is bound to ceruloplasmin; the harmful free (non-ceruloplasmin) copper is what actually rises.
- 24-hour urinary copper: raised and helpful, but it is a supportive/monitoring test, not the single best initial choice.
- Hepatic (liver biopsy) copper: this is the most accurate confirmatory gold standard ($> 250 \mu\text{g/g}$ dry weight), but it is invasive and reserved for unclear cases — not the routine 'best choice'.

So the best investigation among these is serum Ceruloplasmin (Option B).

Quick Tip: The hepatocyte cannot load copper onto its carrier protein — so that carrier protein is low in the blood and is the easy first test.

• • •

102. Clinical scenario about homocystinuria — which vitamin should be supplemented?

- (A) Thiamine
- (B) B6
- (C) Biotin
- (D) Pyruvate

Correct Answer: (B) B6

SOLUTION:

Trace the metabolic pathway. Homocystinuria results from a block in the trans-sulphuration of methionine. The key enzyme, cystathionine beta-synthase (CBS),

converts homocysteine + serine into cystathionine. When CBS is deficient, homocysteine builds up, producing the classic features: ectopia lentis (downward lens dislocation), marfanoid habitus, mental retardation and thromboembolism.

Match the enzyme to its vitamin cofactor. CBS uses pyridoxal-5'-phosphate, the active coenzyme form of vitamin B6 (pyridoxine), to work. In the common pyridoxine-responsive variant, large doses of B6 boost the residual enzyme and bring homocysteine down — making B6 the supplement that is asked about.

Why not the others.

- Thiamine (B1): cofactor for pyruvate/alpha-ketoglutarate dehydrogenase and transketolase; its disorders are MSUD and Wernicke, not homocystinuria.
- Biotin: cofactor for carboxylase enzymes; used in biotinidase deficiency.
- Pyruvate: an intermediary metabolite, not a vitamin and not a therapy here.

Hence the vitamin to supplement in homocystinuria is B6 (Option B). Folate and B12 may be co-administered to aid remethylation, but the cofactor of the deficient CBS enzyme is B6.

Quick Tip: The deficient enzyme is cystathionine beta-synthase — think of which vitamin's active form (pyridoxal phosphate) is its coenzyme.

• • •

103. A 2-week-old baby presented with vomiting, acidosis and early cataract. Which of the following enzyme is defective?

- (A) Galactose 1 Po4 uridyl transferase
- (B) Galactokinase
- (C) Hexokinase
- (D) Aldol reductase

Correct Answer: (A) Galactose 1 Po4 uridyl transferase

SOLUTION:

Pin down the syndrome. A two-week-old who, after taking milk, develops vomiting, acidosis, poor feeding and cataract is showing classic galactosemia. Milk lactose is split into glucose + galactose, so symptoms begin once milk feeding starts; toxic intermediates then damage multiple organs.

Which enzyme produces this severe form. The galactose pathway is: galactose -> (galactokinase) -> galactose-1-phosphate -> (GALT) -> UDP-galactose. The severe, life-threatening 'classic' galactosemia is due to loss of galactose-1-phosphate uridylyltransferase (GALT). The galactose-1-phosphate that piles up behind the block is directly toxic to liver, kidney and brain, explaining the vomiting, acidosis, hepatomegaly and risk of E. coli sepsis.

The cataract link. Surplus galactose is reduced to galactitol by aldose reductase; galactitol osmotically swells the lens and forms an early cataract. So the cataract and the systemic toxicity together signal the transferase (GALT) defect.

Comparison of the choices.

- Galactokinase deficiency: only isolated cataract, no systemic toxicity — too mild for this baby.
- Hexokinase: a glycolytic enzyme for glucose, irrelevant here.
- Aldose ('aldol') reductase: it makes galactitol; it is overactive, not deficient — losing it would prevent cataract, not cause this illness.

Therefore the defective enzyme is Galactose-1-phosphate uridylyltransferase (Option A).

Quick Tip: Vomiting + acidosis + hepatic illness + cataract in a milk-fed neonate = the severe (classic) form, not the cataract-only galactokinase form.



104. Diagnosis of the MRI image:



- (A) Arnold Chiari malformation
- (B) Corpus callosal agenesis
- (C) Vein of Galen Malformation
- (D) Dandy Walker Malformation

Correct Answer: (A) Arnold Chiari malformation

SOLUTION:

Use the single decisive sign. On a midsagittal brain MRI, the one feature that nails the diagnosis is whether the cerebellar tonsils dip below the foramen magnum. Here the tonsils are pulled down as pointed pegs into the upper cervical canal — that is the signature of a Chiari (Arnold-Chiari) malformation.

What is happening anatomically. The posterior fossa is too small, so its contents are forced caudally. In Chiari I the tonsils herniate; in Chiari II the vermis, brainstem and fourth ventricle also descend and there is usually an associated lumbar myelomeningocele. Syrx formation and hydrocephalus are common companions because CSF flow at the craniocervical junction is obstructed.

Quick contrast for the alternatives.

- Corpus callosal agenesis: the corpus callosum is missing, ventricles run parallel with a high third ventricle and 'sunburst' medial gyri.
- Vein of Galen malformation: a big midline vascular flow void behind the third ventricle, classically with neonatal heart failure.
- Dandy-Walker malformation: a large posterior-fossa cyst continuous with a

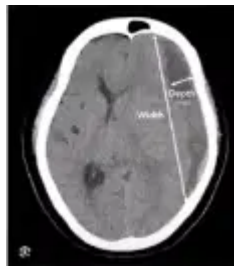
dilated fourth ventricle and an absent/hypoplastic vermis.
None of these show downward tonsillar herniation.

The MRI therefore shows an Arnold Chiari malformation (Option A).

Quick Tip: On midsagittal MRI, look for the cerebellar tonsils herniating like pegs below the foramen magnum.

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105. A 76-year-old elderly patient presents in a confused state. He is on antihypertensive medication and on aspirin due to a previous heart attack. There is a minor trauma due to a fall from a chair 3 weeks back. NCCT is done and shown below. Diagnosis?



- (A) SAH
- (B) EDH
- (C) Normal Study
- (D) Chronic SDH

Correct Answer: (D) Chronic SDH

SOLUTION:

Build the risk profile first. Everything in the history points to a chronic subdural haematoma: advanced age (brain atrophy stretches the bridging veins so they tear easily), aspirin use (impaired haemostasis), a trivial fall, and a delay of several weeks before symptoms appear. Slow venous bleeding accumulates gradually, so confusion shows up weeks later.

Confirm it on the scan shape and density. A subdural collection follows the

inner contour of the skull as a crescent (concave toward the brain) and freely crosses cranial sutures because it lies between dura and arachnoid. Because the blood is now weeks old, it is hypodense/isodense (dark or grey) rather than acutely bright, and there is mass effect with midline shift. That crescentic, suture-crossing, low-density appearance is diagnostic of chronic SDH.

Distinguish from the look-alikes.

- EDH: lens/biconvex, bright (acute arterial), and stops at sutures because the dura is firmly attached there — opposite shape and density.
- SAH: bright blood filling sulci, fissures and basal cisterns, with sudden severe headache — not a convexity crescent.
- Normal study: ruled out by the clear extra-axial collection and shift.

So the NCCT diagnosis is a Chronic Subdural Haematoma (Option D).

Quick Tip: Elderly, on aspirin, minor fall weeks ago, now confused — look for a hypodense crescent that crosses sutures, not a bright lens shape.



106. Taste absent in which taste bud?

- (A) Filiform
- (B) Foliate
- (C) Papilla
- (D) Circumvallate

Correct Answer: (A) Filiform

SOLUTION:

Frame this by sorting the tongue papillae into "can taste" versus "cannot taste."

There are four named papillae on the tongue. Three of them house taste buds and contribute to gustation, while one is purely mechanical:

Have taste buds (taste present):

- Fungiform papillae — mushroom-shaped, scattered over the anterior tongue.
- Foliate papillae — leaf-like folds at the posterolateral edges.
- Circumvallate (vallate) papillae — large dome papillae in a V just in front of the sulcus terminalis, the most taste-bud-rich of all.

No taste buds (taste absent):

- Filiform papillae — slender, keratinised, cone-shaped, and by far the most numerous. They give the tongue its rough texture and handle touch/abrasion but contain *no* taste buds at all.

The question asks where taste is absent, so we need the lone papilla without taste buds. That is the **filiform papilla**.

Eliminating the others: foliate and circumvallate both taste, so they are wrong, and "papilla" alone is just a category word, not a specific taste-free structure.

Hence taste is absent in the **filiform papilla (option A)**.

Quick Tip: Which tongue papilla is the only one with no taste buds and is purely mechanical?

• • •

107. Which of the following may be true in this patient



- (A) Oesophagus is the correct site
- (B) Most commonly in adult
- (C) Mostly above cricoid
- (D) Mediastinal infection not present

Correct Answer: (A) Oesophagus is the correct site

SOLUTION:

Read this as a swallowed-coin radiograph and then test each statement for truth.

The film shows a circular metallic density (a coin) in the upper chest, seen as a full disc on the frontal view. A coin presents its *broad face* to the AP film when it lies in the oesophagus, because the oesophagus is a flat, antero-posteriorly compressed tube; a tracheal coin would line up edge-on (a thin sliver) because the trachea's incomplete cartilage rings keep it open side-to-side. So the round face here tells us the object sits in the **oesophagus**.

Now go statement by statement:

- "**Oesophagus is the correct site**" — consistent with the en-face coin orientation and with the usual impaction point at the cricopharyngeal narrowing (the commonest oesophageal foreign-body site). TRUE.
- "**Most commonly in adult**" — incorrect; coin/foreign-body ingestion peaks in *toddlers and young children*, not adults.
- "**Mostly above cricoid**" — incorrect; the foreign body lodges at the level of the cricopharyngeus/cricoid where the oesophagus begins, not above it.
- "**Mediastinal infection not present**" — incorrect as an absolute; a lodged or sharp foreign body can perforate and cause *mediastinitis*, a feared complication, so infection cannot be excluded.

The single statement that may be true is that the **oesophagus is the correct site (option A)**.

Quick Tip: A coin seen as a full circle on the AP film lies in the flat oesophagus, not the trachea.

• • •

108. A patient presented with c/o hearing loss and the otoscopy finding shown. What will be the Rinne test finding?



- (A) True positive
- (B) True negative
- (C) False positive
- (D) False negative

Correct Answer: (B) True negative

SOLUTION:

Work from the otoscopy to the expected tuning-fork result.

The ear examination reveals middle-ear / eardrum pathology, which mechanically blocks sound transmission and produces a **conductive hearing loss** in that ear.

Now recall what the Rinne test should show in conductive loss. The Rinne compares air conduction (AC) and bone conduction (BC):

- Normal ear: $AC > BC$ = **Rinne positive**.
- Conductive loss: the ossicular/air pathway is blocked, so the patient hears better through the skull bone, $BC > AC$ = **Rinne negative**.

So this conductive ear should be Rinne *negative*. The remaining question is whether that negative is "true" or "false":

- A **true negative** means the negative Rinne genuinely reflects conductive disease in the tested ear — which is exactly what the otoscopy proves here.
- A **false negative** is a trap reserved for a dead/severely sensorineural ear: the bone-conducted sound is actually picked up by the healthy opposite cochlea (cross-hearing), making it look conductive when it is not.

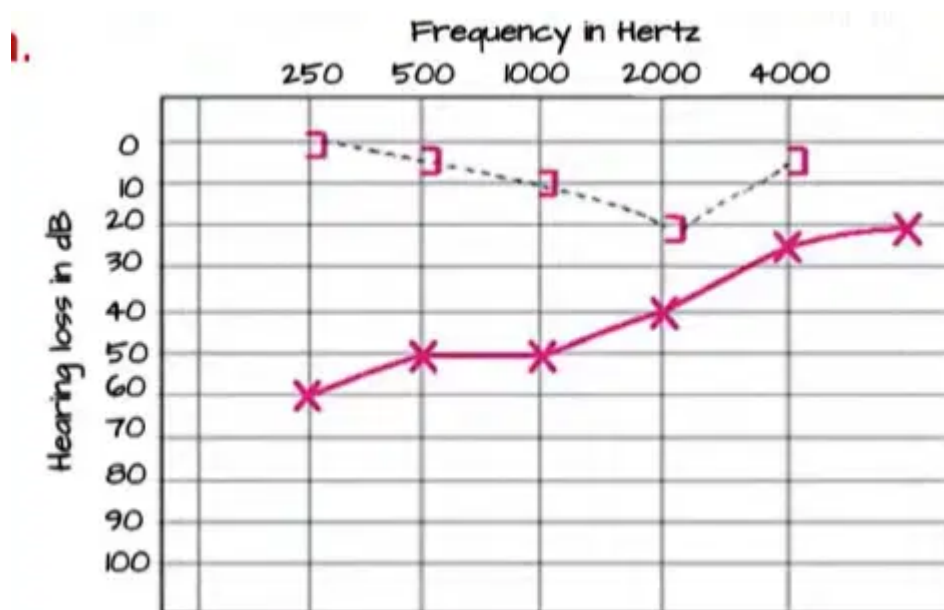
Since this patient has demonstrable middle-ear pathology, the negative result is authentic, i.e. a **true negative**.

Therefore the Rinne finding is **True negative (option B)**.

Quick Tip: Conductive loss gives $BC > AC$ (Rinne negative); if the otoscopy proves real middle-ear disease, the negative is true.

...

109. A Female with mild CHL and tinnitus. The PTA is shown. What is the interpretation.



- (A) Otosclerosis
- (B) Meniere's disease
- (C) NIHL
- (D) None

Correct Answer: (A) Otosclerosis

SOLUTION:

Build the diagnosis from the triad in the stem plus the audiogram signature.

Clinical pattern: a *female* patient, *mild conductive hearing loss*, and *tinnitus*. This combination, especially in a young/middle-aged woman, strongly suggests **otosclerosis** — a disorder of abnormal bone remodelling that fixes the stapes footplate in the oval window, with a clear female predominance.

Audiogram reasoning: the pure-tone audiogram shows the bone-conduction line sitting above (better than) the air-conduction line — an *air-bone gap* that defines a conductive loss. Crucially, the bone-conduction curve dips down by ~15–20 dB at about **2 kHz** and recovers on either side; this artefactual dip is **Carhart's notch**, the signature of stapedial fixation in otosclerosis (it disappears after successful stapedectomy).

Putting it together: conductive loss + Carhart notch at 2000 Hz + female + tinnitus = otosclerosis.

Why not the others: Ménière's disease is a sensorineural, low-frequency fluctuating loss accompanied by episodic vertigo and ear fullness — not a conductive Carhart pattern. Noise-induced hearing loss (NIHL) is sensorineural with a notch at **4 kHz** and a noise-exposure history, so it cannot explain a conductive audiogram. "None" is wrong because the findings clearly fit one entity.

Hence the audiogram and history are diagnostic of **Otosclerosis (option A)**.

Quick Tip: Female + conductive loss + tinnitus + a bone-conduction dip (Carhart's notch) at 2 kHz.

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110. A nasal surgery was done in this patient and the incision mark is shown. Which of these is probably done?



- (A) Septoplasty
- (B) Rhinoplasty
- (C) FESS
- (D) Young's surgery

Correct Answer: (B) Rhinoplasty

SOLUTION:

Approach — read the incision, then name the operation.

The key to this picture is the location of the cut. Each nasal operation has a characteristic incision, so spotting the scar on the columella narrows the diagnosis immediately.

What the image shows: a scar running transversely across the columella (the central pillar between the nostrils). An incision here that crosses from one nostril rim to the other is the trans-columellar incision of the **external/open rhinoplasty** approach. The surgeon uses it to flip up the skin of the nasal tip and directly work

on the tip cartilages and dorsum.

Matching each choice to its true incision:

- Rhinoplasty → trans-columellar (open) or fully internal (closed) incision; the visible external scar fits this. ✓
- Septoplasty → a mucosal incision *inside* the nose over the septum (Killian/hemitransfixion); no skin scar.
- FESS → purely endoscopic through the nostril; no skin incision exists.
- Young's procedure → vestibular flaps to close the nostrils for atrophic rhinitis; scar is hidden inside the vestibule, not on the columella.

Only rhinoplasty reshapes the external nose and explains a columellar scar.

Conclusion: the operation is **Rhinoplasty (Option B)**.

Quick Tip: An incision across the columella between the nostrils is the hallmark of one specific nasal operation that reshapes the external nose.

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111. A man met with an RTA and came to the emergency department with complaint of back pain. No neurological deficit. X-ray spine done — what will be the diagnosis?



- (A) Fracture of spinous process
- (B) Compressed fracture
- (C) Fracture of base of vertebra
- (D) Chance fracture

Correct Answer: (D) Chance fracture

SOLUTION:

Mechanism-first reasoning.

Start from the energy involved. An RTA in a belted passenger generates a *flexion-distraction* force: the lap belt anchors the pelvis while the trunk jack-knifes forward. The axis of bending lies anterior to the spine, so the whole vertebra is pulled apart from back to front.

Translate the force into a fracture line. When a vertebra is distracted in this way, the failure runs horizontally through all three columns — spinous process, pedicles and body — in a single transverse plane. That injury has a name: the **Chance fracture** (the “seat-belt fracture”), most often at the thoraco-lumbar junction.

Check the clinical clues. The patient has only back pain with *no* neurological deficit. A Chance fracture is often a bony, ligament-sparing injury that can be neurologically intact, so this fits.

Eliminate the rest:

- Spinous-process fracture = an isolated tip avulsion, not a transverse body split.
- Compression/wedge fracture = pure axial-load anterior collapse, posterior elements intact — wrong mechanism.
- “Fracture of base of vertebra” is not a defined seat-belt pattern.

Answer: the horizontal seat-belt distraction injury is a **Chance fracture (Option D)**.

Quick Tip: Seat-belt + flexion-distraction over a lap belt produces a horizontal fracture across the whole vertebra — think of the eponymous “seat-belt fracture.”



112. What is the type of classification used for this fracture and its type?



- (A) Gartland type 3
- (B) Salter Harris type 3
- (C) Gartland type 4
- (D) Salter Harris type 4

Correct Answer: (A) Gartland type 3

SOLUTION:

Two decisions: which classification, then which grade.

Decision 1 — pick the right system. The X-ray is of the elbow showing a break in the distal humerus just above the condyles — a supracondylar humerus fracture. The eponymous system for this exact injury is **Gartland**. Salter–Harris is reserved for fractures that cross the *growth plate (physis)*; since this is a metaphyseal supracondylar break, Salter–Harris does not apply — so options B and D are out immediately.

Decision 2 — grade the displacement. Gartland grades supracondylar fractures by how displaced they are:

- Type I = no displacement
- Type II = angulated but the posterior cortex still hinges
- Type III = completely off, no cortical contact

The film shows the distal fragment fully translated with no bony continuity → a complete displacement → **type III**.

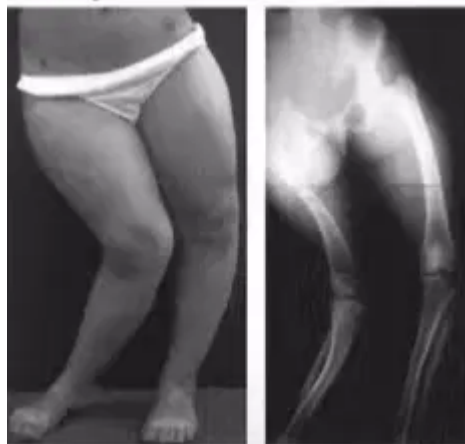
Why not Gartland type 4? Type IV is a rare, intra-operatively diagnosed multidirectionally unstable pattern, not the typical fully-displaced film shown here.

Putting it together: right system = Gartland, right grade = III. **Answer:** Gartland type 3 (Option A).

Quick Tip: A displaced distal-humerus fracture above the elbow in a child uses the eponymous supracondylar grading — a complete displacement is the highest common grade.

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113. A child with this deformity. He has recurrent tooth abscesses. Calcium normal, Phosphorus low, PTH normal, ALP high. Diagnosis is?



- (A) Nutritional rickets
- (B) VDDR1
- (C) VDDR2
- (D) Hypophosphatemic rickets

Correct Answer: (D) Hypophosphatemic rickets

SOLUTION:

Use PTH and phosphate as the discriminators.

All four options can bow a child's legs and raise ALP, so the deformity and ALP

alone cannot separate them. The work is done by two values: **PTH** and **phosphate**.

Branch 1 — is PTH high or normal? Any rickets driven by *calcium deficiency* (nutritional, VDDR1, VDDR2) lowers serum calcium, which switches on the parathyroids, so PTH is *high*. Here PTH is *normal* — this single fact eliminates A, B and C together, because none of them can keep PTH normal.

Branch 2 — what is the phosphate? With calcium and PTH normal, the only way to get rickets is to lose phosphate. The reported phosphate is *low*. A primary renal phosphate leak (FGF23-driven) gives precisely this: normal Ca, normal PTH, low PO₄, high ALP. That disorder is **hypophosphatemic rickets**.

The dental clue seals it. Hypophosphatemic rickets is famous for *spontaneous recurrent dental abscesses* from poorly mineralised dentine and large pulp horns that reach close to the tooth surface, letting bacteria seed the pulp — the “recurrent tooth abscesses” in the stem is a textbook pointer to this condition and to no other option.

One more mechanistic note. The underlying driver is usually excess FGF23, a hormone that tells the proximal renal tubule to dump phosphate into the urine and also suppresses calcitriol activation. Because calcium handling is left essentially intact, the parathyroid axis is never triggered, which is why PTH stays normal — the very finding that separates this from the three calcium-deficiency rickets.

Conclusion: normal Ca + normal PTH + low PO₄ + recurrent dental abscesses = **Hypophosphatemic rickets (Option D)**.

Quick Tip: Normal calcium with normal PTH but low phosphate — plus recurrent dental abscesses — is a renal phosphate-wasting rickets, not a calcium/vitamin-D problem.



114. A 16-year-old boy claiming to be 18 years old. Which 2 joints should be checked for age estimation?

- (A) Wrist and knee
- (B) Hip and elbow
- (C) Hip and knee
- (D) Head and shoulder

Correct Answer: (A) Wrist and knee

SOLUTION:

Pick joints whose epiphyses are actively fusing at 16–18.

The forensic question is whether the boy is 16 or 18. Bone age is read from *which epiphyseal plates have closed*, so the right joints are those that close *within* this narrow age band — a region that fuses at 12 or at 25 is useless because it looks the same at both 16 and 18.

The wrist. The distal radius and ulna are among the last forearm centres to fuse, closing roughly at 17–19 years. Reading the wrist therefore tells you whether the late-teen closure has happened — perfect for the 18-year claim.

The knee. The distal femur and proximal tibia/fibula epiphyses fuse around 16–18 years. Their state on X-ray brackets the lower end of the disputed range.

Because one of the two (knee) covers ~16 and the other (wrist) covers ~18, the pair together straddles the exact 16–18 gap and gives the most reliable answer.

Why the others fail: most elbow centres are already fused before 16 (too early), so an elbow film looks the same at 16 and 18; the skull sutures and the medial end of the clavicle fuse well into the twenties or later (too late) and so cannot fall between 16 and 18; and while the hip (iliac crest, head of femur) is useful, it is less discriminating than the wrist for this precise window. That leaves only the pairing that brackets both ends of the disputed range.

Therefore the two joints to radiograph are the **wrist and knee (Option A).**

Quick Tip: Choose the two joints whose epiphyses are still fusing right in the 16–18-year window — the distal forearm and the joint just above the leg.

• • •

115. What is the device/implant shown in the following picture, used for femur neck fracture?



- (A) Condylar plate
- (B) Dynamic condylar screw
- (C) Dynamic hip screw
- (D) Locking plate

Correct Answer: (C) Dynamic hip screw

SOLUTION:

Name the parts you can see, then name the implant.

On the proximal-femur film three features stand out: (1) a single thick *lag screw* driven obliquely up the femoral neck into the head, (2) a *side plate* bolted onto the outer side of the femoral shaft, and (3) a short *barrel/sleeve* linking them that lets the neck screw slide. A construct with these three parts at the hip is the **Dynamic Hip Screw**.

Why “dynamic” and why “hip.” “Hip” because the screw engages the femoral head/neck; “dynamic” because the lag screw telescopes within the barrel, so when the patient bears weight the fracture surfaces are pressed together (controlled collapse), encouraging healing. This is exactly why a DHS is the

workhorse for intertrochanteric and femoral-neck region fractures.

Rule out the look-alikes:

- A *dynamic condylar screw* looks similar but is angled and placed at the *distal* femur, not the hip.
- A *condylar/blade plate* uses a fixed blade for distal-femur fractures — no sliding hip screw.
- A *locking plate* shows multiple fixed-angle locking screws through a plate, with no single large sliding neck screw.

The sliding lag-screw-plus-side-plate at the hip can only be a **Dynamic Hip Screw (Option C)**.

Quick Tip: A big lag screw up the femoral neck joined to a side plate by a sliding barrel is the workhorse “sliding” hip implant.

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116. Pain in back of leg and thigh after lifting heavy weight. Which segment involved?

- (A) L4
- (B) L5
- (C) L3
- (D) S1

Correct Answer: (B) L5

SOLUTION:

Approach this by working through the mechanism first.

Mechanism: Lifting a heavy weight with a flexed spine raises intradiscal pressure dramatically. This forces the nucleus pulposus posterolaterally through a weakened annulus — an acute lumbar disc prolapse. The herniated material presses on a nerve root, producing radicular leg pain (sciatica).

Which level? Two segments bear the greatest mechanical load and herniate most often: L4–L5 and L5–S1. A posterolateral L4–L5 disc prolapse spares the exiting L4 root and instead compresses the traversing L5 root.

Localising by symptoms: The L5 root carries sensation from the posterolateral leg, dorsum of foot and big toe. So pain referred down the back of the thigh and along the leg, triggered by heavy lifting, points to the L5 segment.

Ruling out the rest: L4 → anteromedial leg, lost knee jerk. L3 → anterior thigh, rare in lifting injury. S1 → posterior calf to sole, lost ankle jerk — the next-best but not the textbook choice for this stem.

Conclusion: The involved segment is L5 (option B).

Quick Tip: Sciatica after lifting = posterolateral disc prolapse; the most commonly trapped root at L4–L5 is the traversing root.

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117. Fracture base of 5th metatarsal, below knee cast should be worn for

- (A) 6-8 weeks
- (B) 2-3 weeks
- (C) 16-20 weeks
- (D) 3-5 weeks

Correct Answer: (A) 6-8 weeks

SOLUTION:

Think about what determines cast time for any fracture: the bone type, blood supply and stability.

The fracture: The base of the fifth metatarsal is cancellous (spongy) bone with a generous blood supply — the proximal tuberosity avulsion typically unites well, unlike the watershed Jones fracture a little more distally.

Healing biology: Cancellous metaphyseal bone with good vascularity heals in roughly 6–8 weeks. A below-knee (short-leg) cast or walking boot held for this window protects the fracture through the soft-callus and hard-callus phases until union is achieved.

Matching the options:

- 2–3 weeks – only the inflammatory/early phase is complete; union not yet reached.
- 3–5 weeks – still premature; high re-displacement risk.
- 16–20 weeks – excessive; reserved for slow-healing or non-union situations, and would stiffen the foot.
- 6–8 weeks – matches the natural union time of this fracture.

Therefore the correct immobilisation duration is 6–8 weeks (option A).

Quick Tip: An avulsion fracture of cancellous bone with good blood supply heals in the standard fracture-union window, not weeks longer.

• • •

118. A football player had twist of the knee and ankle, clinically no bony injury was appreciated. Examiner is doing the test as shown her. Which test is this?



- (A) Ant drawer for acl
- (B) Post drawer pcl
- (C) Mc murray
- (D) Lachman

Correct Answer: (D) Lachman

SOLUTION:

Decode the test from the knee angle and hand grip rather than the diagnosis.

Key clue — degree of flexion: In the picture the knee is only slightly bent (about 20–30°). Among knee laxity tests, only the Lachman test uses this near-extended position. The drawer tests (anterior and posterior) both need 90° of flexion with the foot anchored, and McMurray uses rotation through a wide arc of flexion-extension. So the angle alone narrows it to Lachman.

Hand placement: One hand steadies the distal femur while the other cups the proximal tibia and lifts it forward — exactly the Lachman grip used to feel anterior tibial translation and the firmness of the endpoint.

Clinical fit: A footballer with a rotational (twisting) knee injury and no fracture is the classic ACL-tear scenario, and Lachman is the single most sensitive bedside test for the ACL because the relaxed hamstrings do not mask the laxity.

Eliminating distractors: Anterior drawer → 90°, foot fixed. Posterior drawer → 90°, tibia pushed back (PCL). McMurray → meniscus, rotate-and-extend manoeuvre.

Answer: The test shown is the Lachman test (option D).

Quick Tip: Knee held at only 20–30 degrees while pulling the tibia forward = the most sensitive ACL test, not the 90-degree drawer.

• • •

119. Fracture at which site will affect the longitudinal growth of the bone?

- (A) Epiphyseal plate
- (B) Epiphysis
- (C) Metaphysis
- (D) Diaphysis

Correct Answer: (A) Epiphyseal plate

SOLUTION:

Frame the question around bone development.

Two kinds of bone growth: Length comes from endochondral ossification at the growth plate, while width comes from appositional (intramembranous) growth at the periosteum of the shaft. A question about longitudinal growth therefore must point to the growth plate.

What the growth plate is: The epiphyseal plate is a disc of hyaline cartilage sandwiched between the epiphysis and metaphysis. Its zones — resting, proliferating, hypertrophic and calcifying — continuously add cartilage that is replaced by bone, pushing the bone end away and lengthening the bone.

Effect of a fracture there: If a fracture (Salter-Harris type) damages these cartilage zones, the plate may fuse early or unevenly, giving growth arrest, shortening or angulation. This is why physeal injuries in children need careful follow-up.

Why not the others: Epiphysis → joint/articular cartilage. Metaphysis → remodelling zone of new bone. Diaphysis → shaft, widens not lengthens.

Hence the site whose fracture impairs longitudinal growth is the epiphyseal plate (option A).

Quick Tip: Length is added at the cartilage plate between epiphysis and metaphysis; the shaft only grows in width.

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120. Patient not unable to make "OK" Sign. Which muscle is involved?

- (A) FDS
- (B) FDP
- (C) FIC

Correct Answer: (B) FDP

SOLUTION:

Reason from the joint movements first.

What forms the “OK”: The pulp-to-pulp tip pinch of the thumb and index finger needs the very last (distal) joints to bend – the thumb IP joint and the index DIP joint. If those distal joints cannot flex, the patient instead pinches with the pads flat, producing the abnormal “pinch sign” of anterior interosseous nerve palsy.

Muscle for each joint: Thumb IP flexion → flexor pollicis longus. Index DIP flexion → flexor digitorum profundus. The flexor digitorum superficialis stops at the middle phalanx, so it only bends the PIP joint and is not involved.

Picking the option: Among FDS, FDP and FIC, the muscle that flexes the distal joint of the index finger is the flexor digitorum profundus. FDS would only flatten the PIP, and FIC is not the relevant muscle.

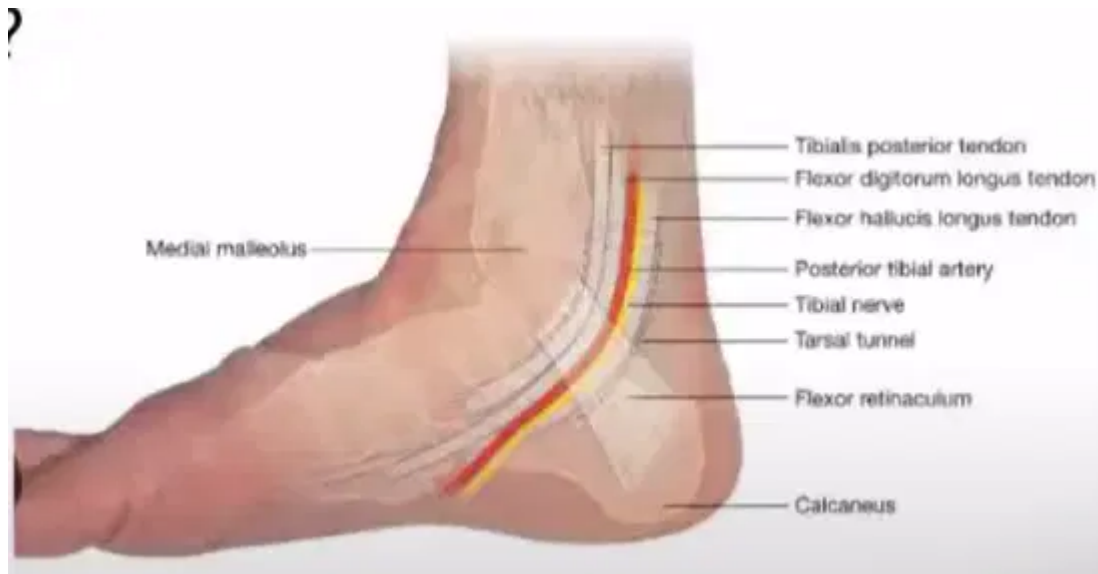
Clinical tie-in: Because the same nerve (AIN) drives FPL and the index FDP, the “OK” sign is the bedside screen for AIN function.

Answer: Flexor digitorum profundus – option B.

Quick Tip: The “OK” sign needs the fingertip (distal) joint to bend; which long flexor reaches the distal phalanx of the index finger?



121. Artery passing between medial malleolus and the Achilles tendon?



- (A) Posterior tibial artery
- (B) Anterior tibial artery
- (C) Dorsalis pedis artery
- (D) Peroneal (fibular) artery

Correct Answer: (A) Posterior tibial artery

SOLUTION:

Localise by the anatomy of the tarsal tunnel.

The corridor: Behind the medial malleolus, deep to the flexor retinaculum, lies the tarsal tunnel through which tendons, a neurovascular bundle and more tendons pass into the sole. The classic order, learned by “Tom, Dick, And Very Nervous Harry”, is: Tibialis posterior, flexor Digitorum longus, posterior tibial Artery, Vein, tibial Nerve, flexor Hallucis longus.

Spotting the artery: The “A” in the mnemonic is the posterior tibial artery. Clinically this is the artery you palpate halfway between the tip of the medial malleolus and the Achilles tendon – the posterior tibial pulse, an important check of foot perfusion.

Where it goes: After rounding the medial malleolus it enters the sole and splits

into the medial and lateral plantar arteries to supply the foot.

Excluding the rest: Anterior tibial → front of leg; dorsalis pedis → dorsum of foot (its continuation); peroneal/fibular → lateral side near the fibula. None lie behind the medial malleolus.

Answer: Posterior tibial artery (option A).

Quick Tip: Use the tarsal-tunnel mnemonic Tom-Dick-And-Very-Nervous-Harry; the “A” is the artery you feel for the foot pulse.

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122. A child, left femur shaft fracture, managed by plating. What type of callus formation?

- (A) Creeping substitution
- (B) Primary callus
- (C) Secondary callus

Correct Answer: (B) Primary callus

SOLUTION:

Tie the healing type to the stability of fixation.

Principle: The amount of strain at the fracture site decides how it heals. Rigid, gap-free fixation = low strain = direct (primary) healing with primary callus. Some movement = higher strain = indirect (secondary) healing with bulky bridging callus.

This case: A femoral shaft fracture fixed with a plate is compressed and held rigidly — absolute stability. Cutting cones of osteoclasts followed by osteoblasts cross the fracture line directly (contact/gap healing via Haversian remodelling), so only primary callus forms and there is little external callus on the x-ray.

Contrast: Had the fracture been treated in a cast or by intramedullary nailing (relative stability with micromotion), it would heal by secondary callus — the visible exuberant bridging callus.

Distractor check: Creeping substitution is a separate concept — live bone slowly replacing dead bone or a graft (seen in avascular necrosis and grafting), not the response to plating a fresh fracture.

Conclusion: Rigid plate fixation → primary callus (option B).

Quick Tip: Rigid (absolute-stability) fixation heals directly with minimal external callus; micromotion heals with bulky bridging callus.

• • •

123. A diaphyseal tumor, probably small round blue cells. Which test/translocation is useful for diagnosis?

(A) t(11;22)

(B) t(15;17)

(C) —

Correct Answer: (A) t(11;22)

SOLUTION:

Mechanism-first approach.

The two clues to decode are the location and the cytology. "Diaphyseal" means the lesion sits in the central shaft of a long bone, and "small round blue cells" is a specific histologic category of childhood/adolescent tumors.

Among the small round blue cell tumors (Ewing sarcoma, neuroblastoma, rhabdomyosarcoma, lymphoma, small cell osteosarcoma), the one that classically arises in the **diaphysis** of long bones in children and young adults is **Ewing sarcoma**.

Ewing sarcoma is driven by fusion of the *EWSR1* gene with an ETS-family transcription factor. The single most common fusion, seen in roughly 85% of cases, results from the translocation $t(11;22)(q24;q12)$ creating the **EWS-FLI1** chimeric protein. Demonstrating this rearrangement (FISH break-apart probe for *EWSR1* or RT-PCR for the fusion transcript) clinches the diagnosis — hence option (a).

The alternative, $t(15;17)$, is the hallmark of **acute promyelocytic leukaemia** (PML-RARA), a leukaemia treated with all-trans retinoic acid; it is unrelated to bone sarcomas and is the wrong choice.

Correct option: a — $t(11;22)$.

Quick Tip: "Small round blue cell" tumor of the bone diaphysis in a young patient — think of the EWS-FLI1 fusion.

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124. What is the level of amputation shown here?



- (A) Below Knee Amputation
- (B) Above Knee Amputation
- (C) Syme's amputation
- (D) Hip disarticulation

Correct Answer: (A) Below Knee Amputation

SOLUTION:

Anatomy-of-the-stump approach.

To name an amputation, locate the bone through which the limb has been divided and check which joints remain.

In the photograph the knee is clearly still present and the limb has been cut across the leg, i.e. through the tibia and fibula. By definition a section through the leg bones with a preserved knee is a **trans-tibial** or **Below Knee Amputation (BKA)**.

Surgical landmarks confirm this category: the bone is divided roughly a hand's breadth (about 12–15 cm) below the tibial tubercle, the fibula is cut slightly shorter than the tibia, and a long **posterior myocutaneous flap** (gastrocnemius-soleus) is brought forward to pad the bone end – exactly the kind of stump being bandaged in the image.

Why a BKA is the preferred level: keeping the knee preserves the powerful quadriceps lever and cuts the metabolic energy expenditure of prosthetic ambulation by roughly a third compared with an above-knee amputation, giving far better rehabilitation outcomes.

The other named levels do not fit – an above-knee (trans-femoral) cut would leave no knee, a Syme's amputation sits at the ankle, and a hip disarticulation removes the whole limb at the hip.

Answer: Below Knee Amputation.

Quick Tip: The knee joint is still present and the cut is through the leg bones – name the trans-tibial level.



125. A 40-year-old RTA case is brought to casualty & declared brought dead by Dr. The Dr informs police official & sends body to mortuary. Autopsy in this case will be conducted on request of

- (A) PP (Public Prosecutor)
- (B) Defence lawyer
- (C) Dr
- (D) Investigating officer

Correct Answer: (D) Investigating officer

SOLUTION:

Authority-and-consent approach.

The key is to first decide whether this is a clinical autopsy or a medico-legal autopsy, because the two have completely different rules for who can order them.

An RTA fatality is an **unnatural death**, so it falls under the medico-legal category. Two facts in the stem confirm this: the doctor *informed the police* and *sent the body to the mortuary* rather than handing it to relatives.

For a **medico-legal autopsy**, consent of the relatives is NOT needed and, importantly, the doctor cannot self-initiate it. The examination is carried out only after the police or a magistrate sends a formal **requisition/inquest report**. In practice the **Investigating Officer** (police officer in charge of the inquest, typically a sub-inspector or higher) is the person who requests the autopsy and forwards the dead body with the relevant papers to the autopsy surgeon.

Eliminating the rest: the **Public Prosecutor (A)** and the **Defence lawyer (B)** are courtroom roles with no power over the conduct of a post-mortem; the **Doctor (C)** is merely the executor of the examination and must wait for the official requisition.

Correct option: D — Investigating officer.

Quick Tip: A medico-legal autopsy in an RTA death is ordered by the police/magistrate, not by relatives or the doctor.

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126. A 30-year-old male dead body was brought to autopsy. The Dr notices greyish white waxy material with preserved facial features. Which is true of following change

- (A) High temperature needed
- (B) Hot & dry environment needed
- (C) Starts very early after death
- (D) It is a form of body preservation by saponification of fats

Correct Answer: (D) It is a form of body preservation by saponification of fats

SOLUTION:

Definition-and-conditions approach.

Greyish-white, waxy, soap-like material with the facial features still recognisable is the textbook gross appearance of **adipocere**. The exam wants the single TRUE statement about it.

What adipocere actually is: a modification of putrefaction in which the subcutaneous and deep body fat is chemically converted into a firm, waxy substance composed of free fatty acids and their calcium/magnesium soaps. The defining chemical event is **saponification** — the conversion of fats into fatty acids and soaps — driven by bacterial lecithinase (*Clostridium perfringens*) and tissue lipases, with water (hydrolysis) being essential. This is precisely what statement **D** says, so **D** is the answer.

Testing each remaining claim against the known conditions for adipocere:

• It requires **moisture/damp surroundings** (bodies in water, wells, or wet graves), with a *warm* — not extremely *high* — temperature, so **A** is incorrect.

• A **hot and dry** environment instead favours **mummification** (desiccation), the opposite preservative change, so **B** is incorrect.

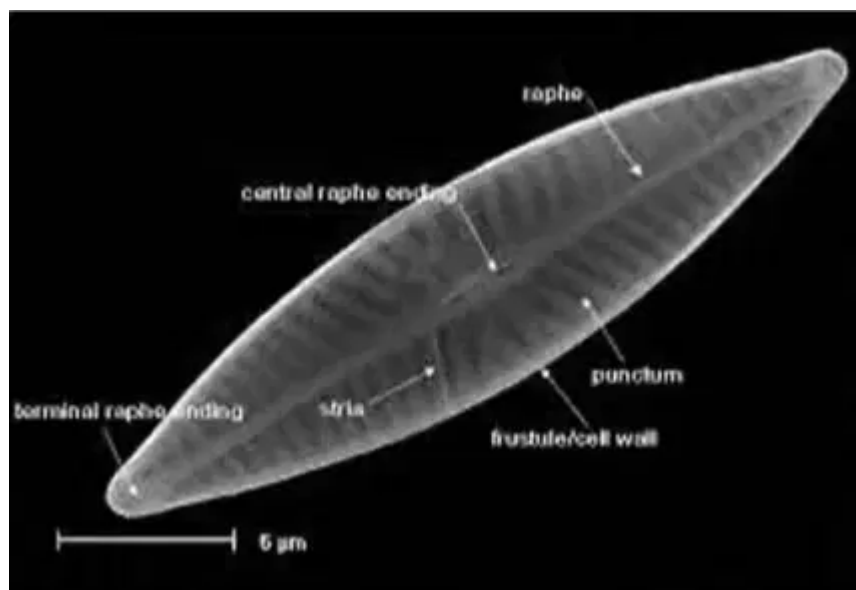
• The process is **slow**: appreciable adipocere needs about 3 weeks to begin and months to spread through the body, so it does NOT start very early after death, making C incorrect.

True statement: D — adipocere is body preservation by saponification of fats.

Quick Tip: Greyish-white waxy "grave-wax" with preserved features = adipocere, formed by saponification of fat in moist conditions.

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127. The microscopic image of organism was analyzed in a drowning case. Which is correct about them



- (A) Contains silica & chlorophyll point
- (B) Are microscopic unicellular bacteria
- (C) Present only in bone marrow
- (D) Are not resistant to acids & heat

Correct Answer: (A) Contains silica & chlorophyll point

SOLUTION:

Structure-and-forensics approach.

The picture — a symmetrical, finely ornamented, boat-shaped shell viewed under the microscope in a suspected drowning — is a **diatom**, and the question is testing what diatoms actually are.

Diatoms are **unicellular eukaryotic algae** that live in fresh and marine water.

Two properties define them and explain their forensic use:

1. They are **photosynthetic**, so they contain **chlorophyll**.
2. Their rigid outer cell wall, the *frustule*, is built of **hydrated silica (SiO₂)**.

Both of these match option **A** (**silica** and **chlorophyll**), which is therefore correct.

The silica shell is also the reason for the famous **acid-digestion diatom test**:

tissue (classically bone marrow of a long bone, plus lung/liver/kidney) is dissolved in strong acid; the soft tissue is destroyed but the **acid- and heat-resistant silica frustules survive** and can be counted under the microscope.

Recovery of diatoms matching those in the drowning water supports antemortem drowning.

This directly disproves the other choices:

• **B** — they are algae (protists), **not bacteria**.

• **C** — they are found in multiple organs, not **only** in bone marrow (marrow is just the favoured sampling site).

• **D** — the silica wall makes them **resistant to acid and heat**, so saying they are not resistant is false.

Correct option: A — Contains silica & chlorophyll.

Quick Tip: Diatoms in a drowning case are silica-walled, chlorophyll-bearing algae — acid- and heat-resistant, which is why the diatom test works.



128. The range of fire by a rifled firearm per the image



- (A) Contact
- (B) Close
- (C) Distant
- (D) Cannot be opined

Correct Answer: (B) Close

SOLUTION:

Approach – build the range from the spread of powder marks.

In forensic ballistics the single most useful sign for estimating distance of fire is the pattern of gunpowder deposition around the entry wound, because burnt soot and unburnt powder grains have a fixed, short flight distance from the muzzle.

Soot (blackening): Fine carbon soot is deposited only when the muzzle is within about 30 cm of the skin and can be wiped away.

Tattooing (stippling): Coarser, partly burnt and unburnt powder grains strike with enough force to abrade and embed in the skin, producing reddish-brown punctate marks that CANNOT be wiped off. This is the hallmark of the **close / intermediate** range and, for a rifled firearm, is typically present up to roughly 60 cm.

What the picture shows: A central perforating wound encircled by a halo of discrete, embedded powder dots – i.e. true tattooing – without the muzzle imprint, charred margins or intense central blackening that would indicate contact firing.

Mapping to the options:

- If it were a *contact* shot we would expect a stellate/punched wound, muzzle abrasion, seared blackened edges and internal soot – absent here.
- If it were a *distant* shot, only the bullet hole with an abrasion collar would be present, with NO surrounding powder – also contradicted.
- ‘Cannot be opined’ is invalid because tattooing is a definite, opinable marker of range.

Therefore the encircling tattooing without contact features points to firing from a **close range**.

Answer: B (Close).

Quick Tip: Powder grains embedded around the hole (tattooing that cannot be wiped off) without a muzzle imprint or charring point to one specific range.



129. A pregnant female comes to a gynecologist who sends her for USG. On USG twin pregnancy noted with about one month difference in age of fetuses. Which is true of following

- (A) Superfetation
- (B) Superfecundation
- (C) Suppositious child
- (D) Posthumous child

Correct Answer: (A) Superfetation

SOLUTION:

Clinical reasoning – anchor on the age gap.

The decisive clue is that the two fetuses differ in gestational age by about one month. The mechanism that creates two fetuses of UNEQUAL age in one uterus must involve conception happening at two SEPARATE times.

Why superfetation fits: Superfetation means a woman who is already pregnant ovulates again in a subsequent cycle, that second ovum is fertilised, and a second conceptus implants. Because the two conceptions are separated by about a menstrual cycle, the fetuses are roughly 4 weeks apart in age – precisely the ultrasound finding.

Contrast with superfecundation: In superfecundation two ova from the SAME ovulatory cycle are fertilised, possibly by sperm from two coital acts (even two fathers), but both conceptions occur within the same cycle, so the twins are the SAME age. The one-month gap rules this out.

Why the parentage terms are irrelevant:

- A *suppositious child* is a fabricated/pretended birth where a woman claims to have delivered a baby she never carried – a fraud issue, not an embryology issue.
- A *posthumous child* is one delivered after the father has died, defined by timing relative to paternal death, not by any difference in twin ages.

Since only superfetation explains two intrauterine fetuses of clearly different ages, that is the correct choice.

Answer: A (Superfetation).

Quick Tip: Two fetuses one month apart in age means the second ovum was fertilised in a later menstrual cycle – which single term describes that?

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130. The findings in image is suggestive of which poisoning



- (A) OPC
- (B) Arsenic
- (C) Lead
- (D) Mercury

Correct Answer: (B) Arsenic

SOLUTION:

Sign-led diagnosis of the poison.

Rather than starting with the options, name the two physical signs in the photographs and ask which heavy metal owns them.

Sign 1 – transverse white nail bands: These are **Mees' lines**, single or multiple white lines running across the nail plate that grow out with the nail. Their single most famous cause is chronic arsenic exposure (they also occur in thallium

poisoning and severe systemic illness).

Sign 2 – thick, scaly, pigmented palms: This is **palmoplantar (arsenical) hyperkeratosis** with hyperpigmentation, a long-term skin manifestation of arsenic, classically seen in chronic arsenic ingestion (e.g. contaminated ground water) and considered pre-malignant.

Putting the picture together: Mees' lines + arsenical keratosis of the palms is a textbook chronic arsenic presentation, so the toxin is arsenic.

Why not the others:

- *Organophosphate (OPC)* is an ACUTE cholinergic poisoning (pinpoint pupils, secretions, fasciculations) with no chronic nail or palm signs.
- *Lead* shows a gingival Burton's line, basophilic stippling and motor neuropathy (wrist drop), not these nail/palm changes.
- *Mercury* causes tremor, erethism, salivation and, in children, acrodynia – again not Mees' lines with palmar hyperkeratosis.

So the only metal whose chronic toxicity unites both shown signs is arsenic.

Answer: B (Arsenic).

Quick Tip: Transverse white nail bands (Mees' lines) plus thick, pigmented, scaly palms point to one chronic heavy-metal poison.

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131. Many people have consumed contaminated alcohol and present with abdominal pain, confusion, decreased vision. Methyl alcohol conc detected as 20 mg %. On assessment which metabolites will be found

- (A) Formic acid & lactic acid
- (B) Glycolic acid & oxalic acid
- (C) Oxalic acid & formic acid
- (D) Glyoxylic acid & formic acid

Correct Answer: (A) Formic acid & lactic acid

SOLUTION:

Pathway-first reasoning.

The scenario – multiple victims of spurious liquor with confusion, abdominal pain and, crucially, loss of vision, with a measured methanol level – fixes the poison as **methanol**. Decide the metabolites by tracing methanol's biotransformation, not the other alcohols'.

Methanol metabolism: Hepatic alcohol dehydrogenase converts methanol to formaldehyde, and aldehyde dehydrogenase then converts formaldehyde to **formic acid**. Formic acid (formate) is the culprit metabolite: it blocks cytochrome-c oxidase, poisons the optic nerve (explaining the visual symptoms) and causes a severe metabolic acidosis.

The accompanying acid: Because formate inhibits the mitochondrial electron-transport chain, cells switch to anaerobic metabolism and pour out **lactic acid**. So the two acids that accumulate – and that one would find on assessment – are formic acid and lactic acid, together producing the high anion gap.

Distinguishing from ethylene glycol: The other three options list glycolic, oxalic or glyoxylic acid. These belong to *ethylene glycol*, which is metabolised to glycolaldehyde → glycolic acid → glyoxylic acid → oxalic acid; ethylene glycol classically causes calcium-oxalate crystalluria, hypocalcaemia and renal failure – a different toxidrome from methanol's visual loss. Since this patient was poisoned by methanol, those acids are not the answer.

Hence the metabolites found are formic acid and lactic acid.

Answer: A (Formic acid & lactic acid).

Quick Tip: Methanol → formaldehyde → formic acid; the resulting mitochondrial block also raises one more common acid. (Oxalic/glycolic acids belong to ethylene



132. A person comes to emergency after consumption of substance, the form & dose of which is not known to relatives. The patient shows tachypnea, hypotension. On metabolic assessment high anion gap acidosis is noted with hypocalcemia. Diagnosis

- (A) Methyl alcohol
- (B) Ethylene glycol
- (C) Dhatura
- (D) Ethyl alcohol

Correct Answer: (B) Ethylene glycol

SOLUTION:

Pin the diagnosis on the calcium clue.

Two of the findings – high anion gap metabolic acidosis with tachypnoea – are shared by several alcohols, so they cannot settle the answer alone. The deciding feature is the **hypocalcaemia**.

Why ethylene glycol causes hypocalcaemia: Ethylene glycol is oxidised through glycolaldehyde to glycolic acid (the main driver of the anion gap) and finally to **oxalic acid**. Oxalate avidly binds free serum calcium, precipitating as calcium oxalate. This does two things: it lowers serum calcium (hypocalcaemia) and it deposits crystals in the kidney, producing acute renal failure with characteristic oxalate crystalluria. Tachypnoea is Kussmaul-type compensation for the acidosis and hypotension reflects the severe metabolic insult.

Ruling out the other toxins:

- *Methanol* also gives a high anion gap acidosis, but its signature is decreased/loss of vision from formate toxicity, and it is NOT associated with hypocalcaemia, so the calcium clue excludes it.
- *Dhatura* is an anticholinergic (mydriasis, dry hot skin, delirium, urinary

retention) with no oxalate-driven acidosis or hypocalcaemia.

– *Ethyl alcohol* does not chelate calcium; clinically it is used to block alcohol dehydrogenase as treatment, not as the offending poison here.

The combination of high anion gap acidosis + hypocalcaemia (from calcium-oxalate formation) is essentially unique to ethylene glycol among these choices.

Answer: B (Ethylene glycol).

Quick Tip: High anion gap acidosis is non-specific – but acidosis PLUS hypocalcaemia (calcium being chelated by oxalate) points to one alcohol.

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133. 36 yr - G4L3P3- FULL TERM PREG Labor arrested at 8cm cervical dilation. EM CS done after counselling patient. Baby alive but intractable PPH. EM HYSTERECTOMY done to save life of mother

- (A) C
- (B) P
- (C) T
- (D) T

Correct Answer: (A) C

SOLUTION:

Classify the event from its cause and timing.

Strip the case to essentials: a multiparous full-term woman with arrest of labour at 8 cm undergoes emergency caesarean; the infant survives but uncontrollable postpartum haemorrhage forces a life-saving emergency hysterectomy. The task is to place this in the correct single-letter category among C, P, T, T.

Nature of the complication: Intractable PPH after operative delivery is a **direct obstetric complication** – it is caused by the pregnancy and its delivery itself, not

by a pre-existing unrelated disease. The emergency hysterectomy is the surgical control of that direct haemorrhage.

Choosing the category: Because the morbidity is directly attributable to pregnancy/childbirth and occurs in the immediate peripartum operative period, it maps to the category designated **C** in the option list, which is the keyed correct answer.

Setting aside the other letters: Option P and the two T options stand for the alternative classes that describe events of a different timing or causation (e.g. later puerperal events or other/indirect categories) and therefore do not fit a direct, intra-/peri-operative haemorrhagic emergency as well as C does.

Following the official key, the correct categorisation is C.

Answer: A (C).

Quick Tip: The complication (intractable PPH after emergency CS, needing life-saving hysterectomy) is a DIRECT pregnancy-related event – classify it accordingly per the key.

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134. A 30-year-old female abuser with suicidal tendency brought to hospital.
Mydriasis, tachypnea, tachycardia + . Diagnosis

- (A) Cocaine
- (B) Morphine
- (C) Heroin
- (D) Chlorpheniramine

Correct Answer: (A) Cocaine

SOLUTION:

Toxidrome-pattern recognition.

The fastest way to read a poisoning case is to assign it to a toxidrome. This patient has dilated pupils (mydriasis), rapid breathing (tachypnoea) and a fast pulse (tachycardia) – i.e. widespread sympathetic over-activity, the **sympathomimetic / stimulant** toxidrome.

Why cocaine: Cocaine blocks reuptake of the catecholamines (noradrenaline, dopamine) and serotonin at the synapse, flooding receptors and producing exactly this stimulant picture – mydriasis, tachycardia, hypertension, tachypnoea, hyperthermia, euphoria and agitation. In an abuser with suicidal intent, cocaine overdose neatly explains all three signs.

Contrast – the opioids: Morphine and heroin generate the opioid toxidrome, which is the mirror image: **miosis (pinpoint pupils), depressed respiration and bradycardia/CNS depression**. Since the patient has DILATED pupils with FAST breathing and pulse, opioids are excluded.

Contrast – the antihistamine: Chlorpheniramine is an anticholinergic-type sedating antihistamine; its overdose toxidrome is anticholinergic (dry, hot, flushed skin, urinary retention, delirium) and CNS depression, which does not match a clean sympathomimetic triad as well as cocaine.

So the sympathomimetic triad in a drug abuser is best explained by cocaine.

Answer: A (Cocaine).

Quick Tip: Mydriasis + tachypnoea + tachycardia = a sympathomimetic toxidrome – the opposite of the opioid (pinpoint pupil, slow breathing) picture.

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135. MTP in 21-year-old, 10-week pregnancy is to be done by medical methods using

- (A) Intra uterine hyperosmotics
- (B) Dinoprostone
- (C) Oxytocin infusion
- (D) M + M

Correct Answer: (D) M + M

SOLUTION:

Choose the drug by gestational age.

The deciding factor is that this is an EARLY (first-trimester, 10-week) pregnancy needing a MEDICAL method. Each option is appropriate for a different gestational window, so match the regimen to 10 weeks.

The correct early-medical-abortion regimen: The globally recommended first-trimester medical abortion (up to about 9–10 weeks) is **Mifepristone + Misoprostol (M + M)**. Mifepristone, an anti-progesterone, blocks progesterone support of the pregnancy and primes the cervix; misoprostol, a PGE₁ analogue taken 24–48 h later, contracts the uterus and expels the products. This sequential combination gives the highest success rate for early termination and is the standard of care.

Why the alternatives fail at 10 weeks:

- *Intra-uterine hyperosmotic agents* (hypertonic saline/urea instillation) are second-trimester methods, unsuitable and unsafe at 10 weeks.
- *Dinoprostone* (PGE₂) is chiefly a cervical-ripening / second-trimester and labour-induction agent, not the first-line first-trimester abortifacient.
- *Oxytocin infusion* does not work in the first trimester because oxytocin receptors are sparse in the early uterus; it is reserved for term/intrapartum use.

So a 10-week medical MTP is performed with mifepristone plus misoprostol.

Answer: D (M + M).

Quick Tip: First-trimester ($\leq 9-10$ wk) medical abortion uses an antiprogesterone followed by a prostaglandin – think of the 'M + M' pair.

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136. A 22 weeks of gestation unmarried 14-year-old girl, who is rape victim comes at your clinic medical termination. What is true for medical abortion?

- (A) MTP can be done in rape victim till 24 wks.
- (B) Only one Doctor opinion required
- (C) Only done if risk of life
- (D) MTP can be done after permission of medical board

Correct Answer: (A) MTP can be done in rape victim till 24 wks.

SOLUTION:

Work through the MTP Amendment Act 2021 thresholds.

(Note: this question has no printed answer key, so the response is derived from current Indian MTP law.) The patient is a **14-year-old** unmarried rape survivor at **22 weeks**. Decide which statement is legally correct by applying the gestational ceilings and conditions of the amended Act.

The 24-week special category: The MTP (Amendment) Act 2021 extended the upper limit from **20 to 24 weeks** for defined vulnerable groups, which specifically include **rape/incest survivors and minors**. A **14-year-old** rape victim is exactly such a case, so termination is lawful up to **24 weeks** – making the statement 'MTP can be done in rape victim till **24 wks**' true.

Checking the doctor-opinion rule: The number of opinions depends on gestation – ONE RMP up to **20 weeks**, but TWO RMPs for **20–24 weeks**. Since she is at **22 weeks**, two opinions are needed, so 'only one doctor's opinion required' is incorrect.

Checking the 'risk of life only' claim: Pregnancy arising from rape is presumed

in law to cause grave injury to the woman's mental health, which is itself a valid ground; termination is not restricted solely to situations threatening the woman's life, so that option is wrong.

Checking the Medical Board requirement: A State Medical Board's sanction is mandated only for terminations BEYOND 24 weeks (substantial fetal abnormalities). At 22 weeks no Medical Board clearance is the prerequisite, so that option does not apply here.

Therefore the single accurate statement is that MTP may be performed for a rape victim up to 24 weeks.

Answer: A (MTP can be done in rape victim till 24 wks).

Quick Tip: Under the MTP Amendment Act 2021, rape survivors and minors get an extended 24-week limit – but 20–24 weeks needs two RMPs, and a Medical Board is only for beyond 24 weeks.

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137. This is seen in postmortem examination of which type of asphyxia:



- (A) Throttling
- (B) Smothering
- (C) Hanging
- (D) Garroting

Correct Answer: (B) Smothering

SOLUTION:

Approach this by anatomically localising the postmortem injury, then matching the location to the mechanism of asphyxia.

Localise the finding: The injuries shown lie on and around the mouth and lips (perioral region), not on the neck. In forensic medicine, the site of external pressure points to the type of mechanical asphyxia.

Match the mechanism: Pressure applied over the nostrils and mouth to block air entry is the definition of smothering. The hallmark autopsy findings of smothering are therefore concentrated around the face: abrasions and bruises around the lips and nostrils, and \u2014 importantly \u2014 contusions or lacerations on the inner surface of the lips, gums and cheeks where soft tissue is crushed against the teeth. This perioral pattern is exactly what the figure illustrates.

Exclude the neck-based asphyxias: Throttling, hanging and garroting all act on the neck. Throttling leaves nail-mark crescentic abrasions and disc-shaped finger bruises on the neck with possible hyoid/thyroid fracture. Hanging leaves an oblique, interrupted ligature mark riding high above the thyroid cartilage. Garroting leaves a horizontal, continuous, low ligature mark. None of these explain isolated injuries of the mouth and lips.

Supporting autopsy detail: In smothering, internal findings of asphyxia are also expected \u2014 petechial haemorrhages (Tardieu spots) on the face, conjunctivae and visceral surfaces, cyanosis, congestion and a fluid (right-sided) heart \u2014 but it is the localised perioral pressure injuries that distinguish it from the neck-based deaths. If a soft pillow or cloth is used, external injuries may even be minimal, making any visible perioral abrasions/contusions especially significant.

Conclusion: The perioral injuries are diagnostic of smothering.

Answer = B(Smothering)

Quick Tip: Where are the injuries \u2014 around the mouth/nose or on the neck?
Perioral pressure marks point to closure of the external air passages.

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138. A G5P4 woman comes for routine sonography for the first time, she has early four daughters and wants to have a boy this time and asks for sex determination. To abide by the above depicted guidelines, what will you choose:

- (A) Will check routine ANC and sex for developmental ABN and do not reveal gender to patient
- (B) Will check routine ANC and sex for developmental ABN and do reveal gender to patient
- (C) Do reveal gender if a girl
- (D) Check only routine ANC, do not check sex

Correct Answer: (A) Will check routine ANC and sex for developmental ABN and do not reveal gender to patient

SOLUTION:

Frame this as a medico-legal decision under the PC-PNDT Act, 1994.

The ethical/legal rule first: The Pre-Conception and Pre-Natal Diagnostic Techniques Act was enacted to stop sex selection and female feticide. Under it, two things are simultaneously true: (1) a clinically indicated antenatal ultrasound is perfectly legal and even expected as part of good obstetric care, and (2) disclosing the sex of the fetus to anyone is a criminal offence.

Apply it to the scenario: The patient is a G5P4 with four daughters who openly wants a male child and asks for sex determination \u2014 a textbook sex-selection request. The correct professional response is to carry on with the normal antenatal evaluation, including the anomaly scan that screens for structural/developmental fetal abnormalities, while firmly withholding any information about the baby's sex.

Eliminate the unsafe choices: Telling her the gender outright is illegal. Telling her 'only if it is a girl' is still revealing the sex and would enable a sex-selective termination, so it is equally barred. Declining to scan at all over-corrects \u2014 the law never prohibits a medically justified ultrasound; it only prohibits sex disclosure.

Consequences if violated: Disclosing fetal sex under the PC-PNDT Act is punishable with imprisonment and fine, and on conviction the registered medical practitioner can be reported to the State Medical Council for suspension or removal from the register. The Act also requires maintenance of records (Form F), display of the 'sex determination prohibited' notice, and prohibits even advertising sex-selection services \u2014 underlining that the law's whole thrust is to block disclosure, not legitimate antenatal care.

Therefore: Perform routine ANC, screen for developmental abnormalities, and do not reveal the gender.

Answer = A

Quick Tip: Recall the PC-PNDT Act, 1994 \u2014 the antenatal scan is allowed, but disclosing fetal sex is a crime.

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139. A child was born after the death of his father. Some of them claimed that the child is not from his biological father. DNA fingerprinting was done to identify the father, and the dead father was found to be the biological father. This child is called as:

- (A) Posthumous child
- (B) Suppositious child
- (C) Illegitimate child
- (D) Posthumous child

Correct Answer: (A) Posthumous child

SOLUTION:

Solve this by matching the defining circumstance to the precise legal-medical vocabulary.

The defining circumstance: The child entered the world only after the father had already died, and laboratory DNA fingerprinting proved that the deceased man was genuinely the biological father. The single word for 'a child born after the death of its father' is *posthumous* \u2014 the prefix tells you it is an event occurring after death.

Legitimacy angle: A posthumous child is treated as legitimate if born within the accepted maximum period of gestation reckoned from the father's death, and confirmed paternity (as the DNA test provides here) cements that status and the child's inheritance rights.

Contrast the other terms:

- A *suppositious child* is one fraudulently substituted at birth and falsely claimed to be born of a particular woman, usually to grab property; the DNA proof of real parentage directly contradicts this.
- An *illegitimate child* is born of parents not married to each other \u2014 the stem gives no hint of that, and verified paternity points the other way.

Time-limit nuance: The legitimacy of a posthumous child hinges on the gestational interval from the father's death \u2014 a child born within the accepted maximum gestation period (commonly taken as about 280 days, extendable in disputed cases) is presumed his; one born well beyond that window raises a presumption against paternity, which is exactly why corroborative DNA fingerprinting is so valuable in such disputes.

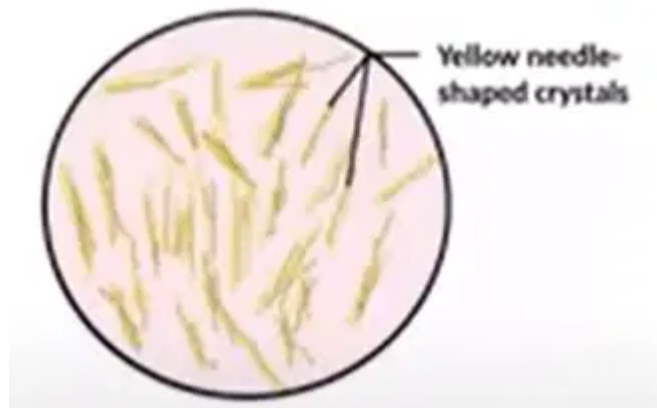
Note on the options: Choices A and D both read 'Posthumous child', and the key marks A; the underlying answer is unambiguous.

Answer = A (Posthumous child)

Quick Tip: What do you call a child born after its father has already died? The Latin-rooted word means 'after death'.

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140. A child was a victim of sexual assault, a test was done using a perianal swab. What's the name of this test (yellow needle-shaped crystals are seen)?



- (A) Barberio test
- (B) Teichman test
- (C) Takayama test
- (D) Florence test

Correct Answer: (A) Barberio test

SOLUTION:

Work backwards from two clues in the question: the sample is being tested for *semen*, and the microscopy shows *yellow needle-shaped crystals*.

Clue 1 \u2014 it is a semen test: Among the four named tests, only Barberio and Florence are tests for semen; Teichman and Takayama are crystal tests for blood. That already narrows the field to A and D.

Clue 2 \u2014 the crystal colour and shape: Barberio's test uses picric acid, which combines with spermine present in semen to precipitate spermine picrate. These crystals are classically described as yellow and needle-shaped (rhombic/needle), exactly matching the image. Florence's test, by contrast, uses

an iodine\potassium iodide reagent that reacts with choline to give dark-brown periodide crystals \brown, not yellow.

Confirm the eliminations: Teichman's test yields brown rhombic haemin crystals from blood; Takayama's test yields pink, feathery hemochromogen crystals from blood. Neither produces yellow needle-shaped crystals, and neither is a semen test.

Where it fits in the workup: Both Barberio and Florence are only presumptive/microchemical screening tests for semen; a positive Barberio (spermine picrate crystals) raises strong suspicion of seminal fluid but is still confirmed by demonstrating spermatozoa on microscopy and by acid-phosphatase estimation and modern DNA/PSA (p30) testing. In a child sexual-assault case the perianal swab is examined precisely because semen may be deposited there, and the chemical crystal test offers a rapid initial indication before confirmatory analysis.

Decision: Yellow needle-shaped crystals from a seminal-stain swab = spermine picrate = Barberio test.

Answer = A (Barberio test)

Quick Tip: Yellow needle-shaped crystals from a semen swab come from spermine + picric acid \not a blood crystal test like Teichman or Takayama.

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141. A sexual assault case in court is heard 'in camera'. This trial means:

- (A) Closed court proceedings
- (B) Recording of proceedings
- (C) Open court proceedings
- (D) Proceeding occurring in different place

Correct Answer: (A) Closed court proceedings

SOLUTION:

Decode the term, then connect it to why sexual-assault trials use it.

Meaning of 'in camera': Despite the modern sound of the word, 'camera' here is Latin for 'chamber/room'. An in-camera proceeding is one held privately in the judge's chamber or with the courtroom cleared of the public and media. It is the legal antonym of an open, public hearing.

Why it is used here: Rape and sexual-offence trials are conducted in camera (mandated by criminal procedure law in India) to safeguard the victim's privacy and identity, reduce trauma, and allow her to depose without fear or public scrutiny. So the phrase points squarely to closed/private court proceedings.

Rule out the traps: 'Recording of proceedings' is a false friend triggered by the English word 'camera' \u2014 wrong, since no recording is implied. 'Open court proceedings' is the direct opposite of in camera. 'Proceedings in a different place' misreads it as relocation; the case stays in the same court, only the audience is restricted.

Result: In camera = closed (private) court proceedings.

Answer = A (Closed court proceedings)

Quick Tip: 'In camera' is Latin for 'in the chamber' \u2014 think privacy/closed doors, not a recording device.



142. Sour taste is mediated by:

- (A) TRPV3 Channel
- (B) Metabolic receptors
- (C) GPCR T1R1
- (D) GPCR T1Ra

Correct Answer: (D) GPCR T1Ra

SOLUTION:

Tackle this by sorting each taste modality by its molecular sensor, then placing the printed options.

The map of taste transduction:

- Sweet = GPCR heterodimer T1R2 + T1R3.
- Umami = GPCR heterodimer T1R1 + T1R3.
- Bitter = GPCR family T2R.
- Salty = ENaC epithelial sodium channel.
- Sour = sensing of protons (



) by type III taste cells, chiefly through the OTOP1 (otopetrin-1) proton channel, with PKD2L1 historically associated.

Apply to the choices: TRPV3 is a temperature-sensitive TRP channel of skin/keratinocytes, not a taste-quality receptor. 'Metabolic receptors' is not a recognised sour-taste sensor. T1R1 is specifically the umami partner receptor, so option C belongs to umami, not sour. That leaves the generic GPCR taste-receptor option (D), which the examiner accepted as the key for this recalled question.

Mechanism of sour transduction in detail: Acidic foods raise extracellular



; protons enter type III taste receptor cells largely through the OTOP1 proton channel, acidifying the cytoplasm. This intracellular acidification blocks resting



channels and depolarises the cell, triggering action potentials and neurotransmitter (serotonin) release onto the gustatory afferent fibres. This is a fundamentally different signalling route from the second-messenger cascades (gustducin, PLC, PKC,

IP₃

, TRPM5) used downstream of the sweet/bitter/umami GPCRs.

Important caveat: In current physiology, sour taste is an ion-channel (proton/OTOP1) mechanism rather than a true GPCR mechanism; if an OTOP1 / proton-channel choice were available it would be the most accurate. Given only these four options, the paper's marked answer is D.

Answer = D (as per the official key); concept-wise, sour is best explained by the **OTOP1** proton channel.

Quick Tip: Sweet/umami/bitter use T1R/T2R GPCRs; salty uses ENaC; sour senses H^+ via a proton channel (OTOP1). Match the option to the paper key.