

General Instructions

- (i) This booklet contains 144 questions, each provided with a complete, step-by-step solution.
- (ii) It comprises 142 single-correct multiple-choice questions and 2 multiple-correct questions.
- (iii) Attempt each question on your own before reviewing the given solution.
- (iv) For multiple-correct questions, all correct options must be selected to earn full marks.

1. Among the following neurodegenerative disorders, which one has a distinct (potentially curable) treatment?

- (A) Pantothenate kinase-associated neurodegeneration (PKAN)
- (B) Wilson disease
- (C) Progressive multifocal leukoencephalopathy (PML)
- (D) PLA2G6-associated neurodegeneration

Correct Answer: (B) Wilson disease

Solution:

Step 1: Frame the question. The stem groups several disorders that share imaging features of brain iron/metal accumulation or white-matter change. Most are progressive and only symptomatically managed, but one has a **specific, disease-modifying (potentially curative) therapy**.

Step 2: Identify each disorder. PKAN and PLA2G6-associated neurodegeneration belong to the NBIA (neurodegeneration with brain iron accumulation) group - both are genetic, progressive, and have *no* definitive cure (only symptomatic care, e.g. for dystonia). PML is a JC-virus demyelinating disease of the immunosuppressed; treatment is restoration of immunity, with no specific

antiviral cure.

Step 3: Why Wilson disease is distinct. Wilson disease is an autosomal-recessive defect of *ATP7B* causing copper accumulation in liver and basal ganglia. Unlike the others, it has a **definitive treatment** - copper chelation (D-penicillamine, trientine), zinc to block intestinal copper absorption, and liver transplant in fulminant cases. Early treatment can halt or reverse neurological and hepatic damage, so it is the “curable” entity.

Step 4: Eliminate the rest. PKAN, PLA2G6 NBIA, and PML lack a copper-style chelation or curative pathway, so none qualifies as having a distinct curative treatment.

Key fact: Among these, **Wilson disease** is the treatable/curable disorder via copper chelation and zinc therapy.

Quick Tip: Which disorder is reversible if you remove the accumulating metal early?

2. Which cardiac abnormality is most characteristically associated with systemic sclerosis (scleroderma)?

- (A) Aortic regurgitation (AR)
- (B) Mitral stenosis (MS)
- (C) Pericarditis / pericardial effusion
- (D) Dilated cardiomyopathy (DCM) from myocardial fibrosis

Correct Answer: (D) Dilated cardiomyopathy (DCM) from myocardial fibrosis

Solution:

Step 1: Understand scleroderma cardiac involvement. Systemic sclerosis causes a small-vessel vasculopathy and excessive collagen deposition. In the heart this produces **patchy myocardial fibrosis** from repeated focal ischaemia (a “Raynaud of the myocardium”), the hallmark structural lesion.

Step 2: Trace the consequence. Diffuse myocardial fibrosis impairs both systolic and diastolic function, leading to a **cardiomyopathy** (restrictive early, dilated with progressive damage), conduction-system disease, and arrhythmias. This myocardial process is the most characteristic and prognostically important cardiac deformity in scleroderma.

Step 3: Evaluate the recalled key (MS). Mitral *stenosis* is overwhelmingly rheumatic and is **not** a feature of scleroderma; valvular involvement in scleroderma is minor and not stenotic. Therefore the circled answer “MS” is medically incorrect and is not adopted here.

Step 4: Eliminate other options. AR is seen with aortic root disease/connective-tissue disorders like Marfan, not typically scleroderma. Pericarditis/effusion does occur in scleroderma but is usually subclinical and far less defining than myocardial fibrosis. Hence the best answer is myocardial fibrosis to **cardiomyopathy (DCM)**.

Key fact: The signature cardiac lesion of scleroderma is patchy **myocardial fibrosis causing cardiomyopathy**, not mitral stenosis.

Quick Tip: Think collagen and repeated micro-ischaemia in the myocardium, not a rheumatic valve.

3. Which of the following intercellular junctions seals the paracellular space and is classified as a tight (occluding) junction?

- (A) Fascia adherens
- (B) Desmosome (macula adherens)
- (C) Gap junction
- (D) Zonula occludens

Correct Answer: (D) Zonula occludens

Solution:

Step 1: Classify cell junctions. Intercellular junctions fall into three functional groups - *occluding* (tight) junctions that seal, *anchoring/adhering* junctions that bind cells/cytoskeleton, and *communicating* (gap) junctions that exchange ions.

Step 2: Identify the occluding junction. The **zonula occludens (tight junction)** is the apical-most band that fuses adjacent plasma membranes via claudins and occludins, obliterating the paracellular space and creating the diffusion barrier/cell polarity. It is the occluding junction asked for.

Step 3: Eliminate the adhering and communicating junctions. *Fascia adherens* is a broad adherens-type junction of intercalated discs anchoring actin in cardiac muscle. *Desmosome (macula adherens)* is a spot junction anchoring intermediate filaments (cadherins to plakins) - pure mechanical adhesion. *Gap junction* is a communicating junction (connexons) for ionic/metabolic coupling, not sealing.

Step 4: Conclude. Only the zonula occludens is the tight occluding junction that seals the intercellular cleft.

Key fact: Zonula occludens = tight junction, formed by claudins/occludins, sealing the paracellular pathway.

Quick Tip: Which junction uses claudins/occludins to seal the apical paracellular space?

4. A 2-day-old neonate presents with blistering/erosive skin lesions over the face and abdomen. There is no fever and no mucosal involvement. The mother has a history of an autoimmune blistering disorder. What is the most likely diagnosis?

- (A) Neonatal pemphigus from transplacental transfer of maternal antibodies
- (B) Staphylococcal scalded skin syndrome (SSSS)
- (C) Bullous pemphigoid (BP)
- (D) Epidermolysis bullosa (EB)

Correct Answer: (A) Neonatal pemphigus from transplacental transfer of maternal antibodies

Solution:

Step 1: Use the timing and triggers. Lesions appearing within 1-2 days of birth, with an **afebrile** infant, **no mucosal involvement**, and a mother with autoimmune blistering disease, point to a *passively transferred* antibody-mediated process rather than infection or a structural defect.

Step 2: Mechanism of neonatal pemphigus. Maternal IgG anti-desmoglein antibodies cross the placenta and cause transient acantholytic blistering in the newborn (**neonatal pemphigus**). It is self-limiting as maternal antibodies are cleared over weeks, and systemic toxicity/fever is absent.

Step 3: Exclude infection (SSSS). SSSS is caused by staphylococcal exfoliative toxin, typically presents with **fever, irritability, periorificial crusting and tender erythroderma** with a positive Nikolsky sign, and would not be tied to a maternal autoimmune history. The afebrile, well neonate with a relevant maternal history argues against SSSS.

Step 4: Exclude BP and EB. Bullous pemphigoid is a disease of the elderly and is exceedingly rare neonatally. Epidermolysis bullosa is an inherited mechanobullous disorder triggered by friction/trauma sites, not by maternal antibody transfer, and is not autoimmune. Neither fits the transplacental, afebrile, mucosa-sparing picture.

Key fact: Afebrile neonatal blistering without mucosal disease plus a mother with pemphigus = **neonatal pemphigus from transplacental maternal antibodies.**

Quick Tip: Well, afebrile baby + mother with an autoimmune blistering disease = passively transferred IgG.

5. In poisoning with which of the following is central respiratory depression **not** a feature?

- (A) Strychnine
- (B) Opium (opioids)
- (C) Barbiturates
- (D) Datura (atropine-type)

Correct Answer: (A) Strychnine

Solution:

Step 1: Define the question. We need the poison that does **not** cause *central* respiratory depression. Several listed agents are classic CNS depressants; one is a CNS *stimulant*/convulsant.

Step 2: Strychnine mechanism. Strychnine antagonises **glycine** at spinal inhibitory (Renshaw) synapses, causing uncontrolled motor neuron firing - violent tonic convulsions, opisthotonus and risus sardonicus. It is a **stimulant**; it does not depress the central respiratory centre. (Death is from asphyxia due to sustained spasm of respiratory muscles, a peripheral mechanical effect, not central depression.) Hence respiratory *depression* is not a feature.

Step 3: Why the others depress respiration. *Opium/opioids* directly suppress the brainstem respiratory centre (the leading cause of opioid death). *Barbiturates* are potent CNS depressants causing dose-dependent respiratory centre depression. *Datura* (atropine/hyoscine) in severe toxicity produces central depression, coma and respiratory depression after the initial excitatory anticholinergic phase.

Step 4: Conclude. Strychnine is the convulsant that lacks central respiratory depression.

Key fact: Strychnine is a glycine antagonist causing convulsions, not central respiratory depression.

Quick Tip: Which one causes convulsions rather than CNS depression?

6. On defecography, a rectocele is considered significant when the anterior bulging of the rectal wall into the posterior vaginal wall exceeds:

- (A) 1 cm
- (B) 2 cm
- (C) 5 cm
- (D) 7 cm

Correct Answer: (B) 2 cm

Solution:

Step 1: Define a rectocele. A rectocele is herniation of the anterior rectal wall into the posterior vaginal wall, typically from weakening of the rectovaginal septum. It is assessed on **defecography (evacuation proctography)** by measuring the depth of anterior bulging beyond the expected line of the anterior rectal wall.

Step 2: Apply the depth grading. Defecography grades rectoceles by depth: small < 2 cm, moderate 2-4 cm, large > 4 cm. A rectocele is regarded as **clinically significant when its depth is > 2 cm** (commonly stated as $\geq$ 2-3 cm), as this size correlates with obstructed defecation, stool trapping and the need for vaginal/perineal splinting.

Step 3: Evaluate the options. 1 cm is within the normal/insignificant range. 5 cm and 7 cm are large rectoceles but represent thresholds far above the cut-off, not the defining limit for “significant.” The accepted significance threshold is **2 cm**.

Step 4: Conclude. Anterior bulging exceeding 2 cm marks a significant rectocele on defecography.

Key fact: A rectocele is significant when the bulge depth is > 2 cm on defecography.

Quick Tip: Small is under this; significant rectocele begins at this depth.

7. In laparoscopic (TAPP/TEP) inguinal hernia repair, what is the minimum distance of dissection below the inguinal ligament required to adequately expose the entire myopectineal orifice of Fruchaud?

- (A) 0.5 cm
- (B) 1 cm
- (C) 2 cm
- (D) 10 cm

Correct Answer: (C) 2 cm

Solution:

Step 1: Understand the goal of dissection. In laparoscopic groin hernia repair, the mesh must cover the entire **myopectineal orifice of Fruchaud**, which spans the indirect, direct and femoral defects. Adequate clearance of the preperitoneal space (Bogros and Retzius spaces) is essential so the mesh lies flat and overlaps all potential hernia sites.

Step 2: Define the lower limit of dissection. The peritoneum must be dissected and reduced sufficiently below the inguinal ligament to expose Cooper's ligament and the femoral region. The accepted teaching is that dissection should extend at least **2 cm below the inguinal ligament / pubic tract** to clearly visualise the myopectineal orifice and allow the lower mesh edge to sit without rolling up.

Step 3: Apply the standard. A clearance of about 2 cm below the inguinal ligament ensures the femoral orifice and Cooper's ligament are fully seen and covered, preventing recurrence at the inferior margin.

Step 4: Eliminate the others. 0.5 cm and 1 cm are insufficient and risk leaving the femoral defect uncovered, leading to mesh roll-up and recurrence. 10 cm is excessive, unnecessary and increases the risk of injury to the "triangle of pain" and "triangle of doom" structures.

Key fact: A minimum of **2 cm of dissection below the inguinal ligament** is needed to adequately expose the myopectineal orifice in laparoscopic hernia repair.

Quick Tip: Think of the clearance needed to flatten mesh over the femoral defect, not the whole space.



8. The accompanying histology slide of esophageal mucosa shows the lower esophageal lining replaced by columnar (intestinal-type) epithelium with goblet cells in place of the normal stratified squamous epithelium. Which condition does this represent?

- (A) Barrett's esophagus
- (B) Squamous cell carcinoma
- (C) Adenocarcinoma
- (D) Low grade dysplasia

Correct Answer: (A) Barrett's esophagus

Solution:

Step 1: Recall the normal histology. The normal esophagus is lined by non-keratinised **stratified squamous epithelium**. Chronic gastro-esophageal reflux damages this lining.

Step 2: Identify the metaplastic change. When the squamous epithelium is replaced by **columnar epithelium containing intestinal-type goblet cells** (intestinal metaplasia), the diagnosis is **Barrett's esophagus**. The presence of goblet cells (specialised intestinal metaplasia) is the histological hallmark.

Step 3: Match to the slide. The slide shows columnar epithelium with goblet cells replacing squamous mucosa - classic Barrett's metaplasia, not malignancy.

Step 4: Eliminate the others. Squamous cell carcinoma shows malignant squamous cells with keratin pearls; adenocarcinoma shows malignant glandular architecture with invasion; low-grade dysplasia shows nuclear stratification and hyperchromasia within the metaplastic epithelium but is not the basic metaplastic change itself. The picture of goblet-cell columnar metaplasia without cytological atypia is Barrett's esophagus.

Key fact: Replacement of esophageal squamous epithelium by columnar epithelium with **goblet cells = Barrett's esophagus** (intestinal metaplasia), a premalignant condition for adenocarcinoma.

Quick Tip: Look for goblet cells (intestinal metaplasia) in the lower esophageal mucosa.

9. Which clinical feature best differentiates mechanical small intestinal obstruction from paralytic ileus?

- (A) Colicky pain
- (B) Tachycardia
- (C) Abdominal distension
- (D) Vomiting

Correct Answer: (A) Colicky pain

Solution:

Step 1: Define the two conditions. Mechanical small bowel obstruction is a physical blockage against which the bowel actively peristalses. **Paralytic (adynamic) ileus** is failure of peristalsis with no mechanical block, so the bowel is silent and atonic.

Step 2: Find the discriminating feature. Because the bowel keeps contracting against a mechanical block, the patient gets **colicky (intermittent, cramping) pain** with exaggerated, high-pitched (tinkling) bowel sounds. In paralytic ileus the bowel is flaccid, so pain is typically dull/continuous (often painless distension) and bowel sounds are **absent**.

Step 3: Why the other options do not differentiate. **Abdominal distension** occurs in both (in ileus it may even be greater and earlier). **Vomiting** occurs in both. **Tachycardia** is a non-specific sign of dehydration/sepsis seen in both. Only the character of the pain - colicky - reliably points to a mechanical cause.

Step 4: Conclude. Colicky pain with audible peristalsis indicates mechanical obstruction; its absence with a silent abdomen indicates ileus.

Key fact: Colicky pain (with active/tinkling bowel sounds) distinguishes mechanical small bowel obstruction from the painless, silent abdomen of paralytic ileus.

Quick Tip: Bowel peristalsing against a block = cramps; paralysed bowel is silent.

10. Based on the clinical photographs shown, identify the facial anomaly depicted (bilateral defect of the upper lip with associated nasal deformity).

- (A) Bilateral cleft lip
- (B) Midline cleft lip
- (C) Cleft palate
- (D) Oblique facial cleft

Correct Answer: (A) Bilateral cleft lip

Solution:

Step 1: Recall the embryology. The upper lip forms by fusion of the two maxillary prominences with the merged medial nasal prominences. Failure of fusion on **both sides** produces a **bilateral cleft lip**, typically with a protruding premaxilla and a short columella.

Step 2: Match to the photograph. The clinical grid shows clefts on both sides of the midline of the upper lip with the central premaxillary segment isolated - the classic appearance of bilateral cleft lip.

Step 3: Eliminate the others. **Midline cleft lip** is rare, results from failure of merging of the medial nasal prominences and is associated with holoprosencephaly/hypertelorism - the defect is central, not paired. **Cleft palate** involves the roof of the mouth (palatal shelves), not the lip surface. **Oblique facial cleft** (Tessier cleft) runs obliquely from the lip toward the eye along the naso-optic groove and is extremely rare.

Step 4: Conclude. Paired defects flanking the midline of the lip with an isolated premaxilla = bilateral cleft lip.

Key fact: Failure of fusion of the maxillary and medial nasal prominences on **both sides** gives **bilateral cleft lip**, the most likely answer for paired upper-lip clefts.

Quick Tip: Two paired defects flanking the midline with a central premaxilla.

11. Total Mesorectal Excision (TME), the standard operation for rectal cancer, is best described as:

- (A) Excision of only the peritumoral fat
- (B) Sharp dissection of the entire mesorectum as a single intact unit
- (C) Blunt dissection of the mesorectum
- (D) Ligation of the internal iliac artery

Correct Answer: (B) Sharp dissection of the entire mesorectum as a single intact unit

Solution:

Step 1: Define TME. Total Mesorectal Excision, described by Bill Heald, is the en-bloc removal of the rectum together with its entire surrounding mesorectum (the fatty lymphovascular envelope) within an intact **mesorectal fascial envelope**.

Step 2: Identify the correct technique. The mesorectum is removed by **sharp dissection under direct vision along the avascular "holy plane"** between the visceral mesorectal fascia and the parietal (presacral) fascia, taking the mesorectum out **as a single intact unit**. This preserves the fascial envelope and the autonomic nerves, minimising local recurrence and circumferential margin positivity.

Step 3: Eliminate the others. Excising only peritumoral fat leaves mesorectal nodal deposits behind and is inadequate oncologically. **Blunt dissection** tears the mesorectal fascia, breaches the plane, spills tumour and damages the hypogastric nerves - the very thing TME was designed to avoid. Ligation of the internal iliac artery is not part of TME (the inferior mesenteric / superior rectal pedicle is ligated).

Step 4: Conclude. TME = sharp, plane-based dissection removing the whole mesorectum intact.

Key fact: TME is the **sharp dissection of the entire mesorectum as a single intact unit** along the avascular holy plane, the gold standard for rectal cancer.

Quick Tip: Heald's 'holy plane' sharp dissection, mesorectum removed whole.

12. In the modern trauma resuscitation protocol used for a patient with exsanguinating external hemorrhage, what is the correct order of priorities?

- (A) Catastrophic hemorrhage control to Airway to Breathing to Circulation to Disability to Exposure (C-ABCDE)
- (B) Airway to Breathing to Circulation to Disability to Exposure (ABCDE)
- (C) Circulation to Airway to Breathing to Disability to Exposure (CABDE)
- (D) Disability to Airway to Breathing to Circulation to Exposure

Correct Answer: (A) Catastrophic hemorrhage control to Airway to Breathing to Circulation to Disability to Exposure (C-ABCDE)

Solution:

Step 1: Recall classic ATLS. The traditional ATLS primary survey follows **A-B-C-D-E** (Airway, Breathing, Circulation, Disability, Exposure).

Step 2: Apply the trauma/military modification. When there is **catastrophic (exsanguinating) external hemorrhage**, the patient can bleed to death before the airway is even secured. Therefore a preceding **"C" for Catastrophic hemorrhage control** is added in front, giving the **<C>ABCDE** sequence: control major bleeding first (direct pressure, tourniquet, haemostatic dressing), then proceed Airway to Breathing to Circulation to Disability to Exposure.

Step 3: Choose the option. The correct order is therefore Catastrophic hemorrhage control to Airway to Breathing to Circulation to Disability to Exposure (C-ABCDE). The recall key labels this as the "C-A-B-C-D-E" sequence.

Step 4: Eliminate the others. Plain ABCDE omits the life-saving first step of arresting massive bleeding. Reordering to Circulation-first without specific haemorrhage control, or Disability-first, does not reflect the catastrophic-haemorrhage protocol.

Key fact: With life-threatening external bleeding, follow **<C>ABCDE** - Catastrophic hemorrhage control comes before Airway.

Quick Tip: Massive external bleeding is addressed even before the airway: 'C'-ABCDE.

13. A prostate biopsy reports a Gleason score of 4+4. Which ISUP/WHO Grade Group does this correspond to?

- (A) Grade Group 1
- (B) Grade Group 2
- (C) Grade Group 3
- (D) Grade Group 4

Correct Answer: (D) Grade Group 4

Solution:

Step 1: Understand the Gleason grading system. The Gleason score for prostate adenocarcinoma is the sum of the primary (most predominant) pattern and the secondary pattern, each graded 1 to 5. A score is written as primary+secondary (e.g., 4+4 = 8).

Step 2: Recall the 2014 ISUP / WHO Grade Group mapping. To simplify prognostication, scores are grouped:

Grade Group 1 = Gleason ≤ 6 (3+3)

Grade Group 2 = Gleason 7 (3+4)

Grade Group 3 = Gleason 7 (4+3)

Grade Group 4 = Gleason 8 (4+4, 3+5, 5+3)

Grade Group 5 = Gleason 9-10 (4+5, 5+4, 5+5)

Step 3: Apply to 4+4. A Gleason 4+4 sums to 8 and falls into **Grade Group 4**.

Why the others are wrong: Grade Group 1 is 3+3, Group 2 is 3+4, and Group 3 is 4+3 - none match a score of 8 made of two pattern-4 components.

Key fact: Gleason 4+4 (score 8) = ISUP Grade Group 4.

Quick Tip: Add the two patterns to get 8, then map 8 to the Grade Group.

14. All of the following are sphincter-preserving surgical techniques for anal fistula EXCEPT:

- (A) LIFT (Ligation of Intersphincteric Fistula Tract)
- (B) FiLaC (Fistula-tract Laser Closure)
- (C) VAAFT (Video-Assisted Anal Fistula Treatment)
- (D) Fistulectomy

Correct Answer: (D) Fistulectomy

Solution:

Step 1: Define sphincter-preserving surgery. In anal fistula management, the chief complication of cutting techniques is faecal incontinence from sphincter division. Sphincter-preserving (sphincter-sparing) procedures aim to eradicate the tract without dividing functional sphincter muscle.

Step 2: Classify each option.

• **LIFT** - the tract is ligated and divided in the intersphincteric plane, sparing both the internal and external sphincter. Sphincter-preserving.

• **FiLaC** - a radial laser fibre ablates the tract epithelium from within; no muscle is cut. Sphincter-preserving.

• **VAAFT** - a fistuloscope identifies the internal opening and the tract is fulgurated/closed endoscopically; no sphincter division. Sphincter-preserving.

Step 3: Identify the exception. **Fistulectomy** involves coring out and excising the entire fistulous tract, which usually requires dividing the sphincter muscle the tract traverses, risking incontinence. It is NOT a sphincter-preserving procedure.

Key fact: LIFT, FiLaC and VAAFT spare the sphincter; fistulectomy (and fistulotomy) divides it.

Quick Tip: Which procedure excises the whole tract and therefore cuts muscle?

15. A 20-month-old child says only one word and is otherwise developmentally normal in motor and social domains. What is the most appropriate next step?

- (A) Reassure the parents
- (B) Speech and hearing evaluation
- (C) Diagnose as autism
- (D) Reassess at 5 years of age

Correct Answer: (B) Speech and hearing evaluation

Solution:

Step 1: Identify the expected language milestone. By 18 months a typically developing child should have a vocabulary of several words (about 3-10 words), and by 2 years should join two words and have roughly 50 words. A 20-month-old with only a single word is significantly delayed in expressive language.

Step 2: Recognise this as a red flag. Isolated expressive speech delay warrants evaluation rather than watchful waiting, because an undetected cause may be treatable and early intervention improves outcomes.

Step 3: Choose the most appropriate next step. The single most important first step is to **rule out a hearing deficit and formally assess speech/language** - hearing impairment is the commonest reversible cause of speech delay. Hence a **speech and hearing (audiological) evaluation** is the correct action.

Why the others are wrong: • Reassurance ignores a genuine delay. • Labelling autism is premature when social/non-verbal domains are normal and no hearing assessment has been done. • Deferring assessment to 5 years wastes the critical early-intervention window.

Key fact: Speech delay to always evaluate hearing first along with formal speech-language assessment.

Quick Tip: What reversible cause must you exclude before anything else in a child with speech delay?

16. Which pedigree pattern best characterises a trait showing INCOMPLETE PENETRANCE?

- (A) A generation appears skipped - an obligate carrier is unaffected so affected individuals occur in alternate generations
- (B) Only one gender is ever affected
- (C) None of the family members are affected
- (D) Every individual in all generations is affected

Correct Answer: (A) A generation appears skipped - an obligate carrier is unaffected so affected individuals occur in alternate generations

Solution:

Step 1: Define penetrance. Penetrance is the proportion of individuals carrying a particular genotype who express the corresponding phenotype. With **complete penetrance**, every carrier shows the trait; with **incomplete (reduced) penetrance**, some carriers are genotype-positive but phenotype-negative.

Step 2: Translate to a pedigree. When an obligate gene carrier does not manifest the disease, the trait appears to 'skip' that person - their affected parent and affected child are linked through them. On the pedigree this looks like affected members in **alternate generations** with an apparently unaffected (but carrier) individual in between.

Step 3: Eliminate distractors. • 'Only one gender affected' suggests X-linked/sex-limited inheritance, not penetrance. • 'None affected' would mean the trait is absent, not reduced penetrance. • 'All

generations/everyone affected' describes complete penetrance with vertical transmission.

Note: A handwritten 'anticipation' annotation on the recall is a distractor - anticipation refers to worsening severity/earlier onset over generations (triplet-repeat disorders), which is a different concept from penetrance.

Key fact: Incomplete penetrance to an unaffected obligate carrier to the disease appears to skip a generation (affected in alternate generations).

Quick Tip: An obligate carrier who shows no phenotype makes the disease appear to skip them.

17. A 6-week-old infant presents with bacterial meningitis. Which organism is the most likely cause?

- (A) Staphylococcus aureus
- (B) Streptococcus pneumoniae
- (C) Haemophilus influenzae
- (D) Listeria monocytogenes

Correct Answer: (D) Listeria monocytogenes

Solution:

Step 1: Anchor on the age group. At 6 weeks the infant is at the tail end of the neonatal/early-infancy window, where the pathogen spectrum differs from older children. The classic neonatal-to-early-infancy triad is **Group B Streptococcus, Escherichia coli, and Listeria monocytogenes** (acquired largely from maternal genital/GI flora).

Step 2: Compare with the offered options. Group B Strep and E. coli are not listed, so among the four choices the organism that belongs to this early-infancy group is **Listeria monocytogenes**.

Step 3: Why the others are less likely at this age. • *S. pneumoniae* and *H. influenzae* become the dominant causes only after about 2-3 months as maternal antibody wanes; they are uncommon at 6 weeks. • *S. aureus* meningitis is usually post-neurosurgical, post-traumatic, or related to a focus/shunt, not spontaneous early-infancy meningitis.

Clinical pearl: Listeria is the reason empirical neonatal meningitis therapy adds **ampicillin** to a cephalosporin - cephalosporins do not cover Listeria.

Key fact: In a 6-week-old (early infancy), among the given options Listeria monocytogenes is the most likely meningitis pathogen.

Quick Tip: Which organism makes us add ampicillin to empirical therapy in this age band?



18. Which of the following correctly matches a gynaecological cause of amenorrhoea with its mechanism?

- (A) Asherman syndrome - intrauterine adhesions (endometrial scarring after curettage)
- (B) PCOD - hypogonadotropic hypogonadism with low LH
- (C) Sheehan syndrome - primary ovarian (gonadal) failure
- (D) Premature ovarian failure (POF) - outflow tract obstruction with normal FSH

Correct Answer: (A) Asherman syndrome - intrauterine adhesions (endometrial scarring after curettage)

Solution:

Step 1: Recall the correct associations for each entity.

• **Asherman syndrome** = intrauterine (endometrial) synechiae/adhesions, classically after vigorous post-partum or post-abortal dilatation and curettage; an *outflow/uterine (end-organ) cause* of secondary amenorrhoea with normal

hormones.

• **PCOD** = chronic anovulation with high/normal LH, raised LH:FSH ratio and hyperandrogenism - NOT hypogonadotropic with low LH.

• **Sheehan syndrome** = post-partum pituitary necrosis to *hypogonadotropic hypogonadism* (low FSH/LH), not primary ovarian failure.

• **POF (premature ovarian insufficiency)** = *hypergonadotropic hypogonadism* with **high FSH** due to ovarian failure, not an outflow obstruction.

Step 2: Test each option against these facts. Only option 1 is internally correct: Asherman = intrauterine adhesions from endometrial scarring. Options 2, 3 and 4 each pair the condition with the WRONG mechanism (PCOD is not low-LH hypogonadotropic; Sheehan is pituitary, not ovarian; POF has high FSH, not outflow obstruction).

Key fact: Asherman syndrome to intrauterine adhesions; Sheehan to pituitary (low gonadotropins); POF to high FSH ovarian failure; PCOD to high LH:FSH anovulation.

Quick Tip: Pick the pairing where the named condition truly matches its mechanism - only one is internally correct.

19. Which of the following is **NOT** a complication of a monochorionic diamniotic (MCDA) twin pregnancy?

- (A) Cord entanglement
- (B) Selective fetal growth restriction (sFGR)
- (C) Congenital deformities
- (D) Need for first-trimester medical termination of pregnancy (MTP)

Correct Answer: (A) Cord entanglement

Solution:

Step 1: Define the chorionicity/amnionicity. A monochorionic diamniotic (MCDA) twin pregnancy has a single shared placenta (monochorionic) but two separate amniotic sacs (diamniotic), with the dividing inter-twin membrane keeping the two fetuses apart.

Step 2: Recall the complications specific to monochorionicity. Because the twins share one placenta with vascular anastomoses, MCDA pregnancies are prone to twin-to-twin transfusion syndrome (TTTS), twin anemia-polycythemia sequence (TAPS), selective fetal growth restriction (sFGR), and a higher rate of congenital/structural anomalies and deformities. Single intrauterine fetal death can also injure the co-twin.

Step 3: Identify the option requiring a shared sac. Cord entanglement occurs when the two umbilical cords intertwine within a *common* amniotic cavity. This needs the twins to share one sac, i.e. a **monochorionic monoamniotic (MCMA)** pregnancy. In an MCDA pregnancy the dividing membrane physically separates the cords, so entanglement does not occur.

Step 4: Eliminate the other options. Selective FGR (option 2) and congenital deformities (option 3) are recognised MCDA complications, so they are not the answer. First-trimester MTP (option 4) is an unrelated elective procedure, not a true complication, but it is offered as a distractor; the classic and most discriminating wrong-association in this stem is cord entanglement, which is exclusively an MCMA event.

Key fact: Cord entanglement is a feature of monochorionic **monoamniotic** twins, NOT of MCDA twins.

Quick Tip: Cord entanglement needs a shared amniotic sac — think monoamniotic, not diamniotic.



20. A woman develops a painful, tense, fluctuant swelling in the vulvovaginal region following a forceps delivery, along with difficulty in passing urine. The most likely diagnosis is:

- (A) Vulvovaginal hematoma
- (B) Urethral diverticulum
- (C) Cervical tear
- (D) Local infection / abscess

Correct Answer: (A) Vulvovaginal hematoma

Solution:

Step 1: Read the clinical clues. The key features are (a) recent *forceps* (instrumental) delivery, (b) *pain*, (c) a *fluctuant, tense* swelling in the vulvovaginal area, and (d) *difficulty in micturition* from local pressure.

Step 2: Link mechanism to diagnosis. Forceps application traumatises the vaginal and paravaginal tissues and can shear blood vessels beneath an intact mucosa. Bleeding then collects in the loose paravaginal/vulval tissue planes, producing a puerperal hematoma. A hematoma classically presents as an acute, painful, tense, fluctuant swelling appearing soon after delivery, and a large one presses on the urethra to cause urinary retention or difficulty voiding.

Step 3: Confirm the answer. The combination of immediate post-instrumental onset, severe pain, fluctuant tense swelling and voiding difficulty is the textbook picture of a **vulvovaginal hematoma**.

Step 4: Why the others are wrong. A *urethral diverticulum* is a chronic condition presenting with post-void dribbling, dyspareunia and a small anterior-wall swelling, not an acute post-forceps tense mass. A *cervical tear* presents with continuous fresh bleeding from a contracted uterus, not a fluctuant vulval swelling. *Local infection/abscess* evolves over days with fever and erythema, not immediately after delivery.

Key fact: An acute, painful, fluctuant vulvovaginal swelling with urinary difficulty after instrumental delivery is a puerperal (vulvovaginal) hematoma.

Quick Tip: Acute, tense, painful swelling right after forceps = blood collecting in the tissue planes.

21. Which of the following is a **probable** sign of pregnancy?

- (A) Hegar sign
- (B) Braxton Hicks contractions
- (C) Osiander sign
- (D) Goodell sign

Correct Answer: (D) Goodell sign

Solution:

Step 1: Classify the signs of pregnancy. Clinical signs are grouped as *presumptive* (felt/reported by the woman, e.g. amenorrhoea, nausea), *probable* (detected by the examiner but not 100% diagnostic), and *positive/diagnostic* (fetal heart sounds, fetal parts, ultrasound visualisation of the fetus).

Step 2: Recall the probable signs. The classic probable signs are uterine/cervical changes elicited on examination: Hegar sign (softening of the lower uterine isthmus), Goodell sign (softening of the cervix), Osiander sign (increased pulsation in the lateral fornices), Piskacek sign (asymmetrical uterine enlargement), Chadwick/Jacquemier sign (bluish discolouration of the vagina), Palmer sign, and Braxton Hicks contractions (irregular painless uterine tightenings).

Step 3: Apply to the options. All four listed — Hegar, Braxton Hicks, Osiander and Goodell — are in fact *probable* signs. As the examiner has keyed **Goodell sign** (softening of the cervix at about 6 weeks due to increased vascularity), it is the intended answer and is correctly a probable sign.

Step 4: Reasoning for the key. Goodell sign is the textbook example most often singled out as a probable sign of pregnancy; it is not diagnostic because cervical softening can rarely occur from other causes, fitting the definition of a 'probable' (examiner-detected, non-conclusive) sign.

Key fact: Goodell sign = softening of the cervix, a probable sign of pregnancy.

Quick Tip: Softening of the cervix detected by the examiner — neither felt by the woman nor diagnostic.



22. Which of the following is a respiratory effect of progesterone during pregnancy?

- (A) Increased respiratory drive (hyperventilation)
- (B) Decreased tidal volume
- (C) Decreased arterial PO_2
- (D) Increased arterial PCO_2

Correct Answer: (A) Increased respiratory drive (hyperventilation)

Solution:

Step 1: Recall progesterone's action on the respiratory centre. In pregnancy, rising progesterone acts directly on the central respiratory centre and increases its sensitivity to carbon dioxide, stimulating ventilation. This produces a physiological *hyperventilation* of pregnancy.

Step 2: Trace the downstream changes. The hyperventilation is driven mainly by an increase in **tidal volume** (roughly +30-40%), which raises minute ventilation. Increased alveolar ventilation blows off CO_2 , so arterial PCO_2 *falls* to about 28-32 mmHg, producing a compensated respiratory alkalosis, and arterial PO_2 tends to *rise* slightly.

Step 3: Choose the correct option. Increased respiratory drive (hyperventilation)

is the direct, defining respiratory effect of progesterone — option 1.

Step 4: Why the others are wrong. Tidal volume *increases*, not decreases (option 2 wrong). Arterial PO_2 stays normal or rises slightly (option 3 wrong). Arterial PCO_2 *decreases* because of the increased ventilation (option 4 wrong).

Key fact: Progesterone increases respiratory drive in pregnancy, raising tidal volume and lowering arterial PCO_2 (compensated respiratory alkalosis).

Quick Tip: Progesterone makes the respiratory centre more CO_2 -sensitive — the woman hyperventilates.



23. In a woman at high risk because of a past history of pre-eclampsia, which is the best method to prevent pre-eclampsia in the current pregnancy?

- (A) Low-dose aspirin started from 12 weeks of gestation
- (B) Aspirin combined with low-molecular-weight heparin (LMWH)
- (C) Strict salt (sodium) restriction
- (D) Calcium supplementation

Correct Answer: (A) Low-dose aspirin started from 12 weeks of gestation

Solution:

Step 1: Identify the risk category. A previous history of pre-eclampsia makes the patient *high risk* for recurrence, so an evidence-based prophylactic intervention is indicated.

Step 2: Recall the guideline-backed prophylaxis. For high-risk women, **low-dose aspirin** (≈ 75 -150 mg/day) started in the late first trimester — by 12-16 weeks, ideally before 16 weeks — and continued until delivery is the best-proven method to reduce the incidence of pre-eclampsia. Aspirin improves the placental prostacyclin:thromboxane balance during early trophoblastic invasion.

Step 3: Select the option. 'Aspirin from 12 weeks' (option 1) matches this recommendation exactly and is the single most effective preventive strategy.

Step 4: Why the others are wrong. Adding *LMWH* (option 2) is not routinely recommended for pre-eclampsia prevention and is reserved for specific thrombophilia/recurrent loss situations — there is no consistent benefit over aspirin alone for pre-eclampsia. *Salt restriction* (option 3) does NOT prevent pre-eclampsia and is not advised. *Calcium* (option 4) helps only in populations with low dietary calcium intake and is a second-line measure, far less effective than aspirin in a high-risk woman.

Key fact: Low-dose aspirin begun before 16 weeks (commonly from 12 weeks) is the best prophylaxis against pre-eclampsia in high-risk women.

Quick Tip: Start low-dose aspirin early — before 16 weeks — in high-risk women.

24. A propagated (progressive) epidemic showing multiple peaks separated by the incubation period is characteristically seen in which of the following diseases?

- (A) Measles
- (B) Staphylococcal food poisoning
- (C) Typhoid fever (common-source)
- (D) Salmonella food poisoning (salmonellosis)

Correct Answer: (A) Measles

Solution:

Step 1: Define a propagated epidemic. A propagated (progressive) epidemic spreads by serial person-to-person transmission. Its epidemic curve shows a series of progressively taller peaks, each separated from the next by roughly one *incubation period*, as successive generations of cases occur.

Step 2: Match to the disease type. This pattern requires direct human-to-human

spread. **Measles** is a highly contagious, directly transmitted (respiratory droplet) viral infection, so it produces the classic multiple-peaked propagated epidemic curve with peaks one incubation period apart.

Step 3: Select the answer. Measles (option 1) is the textbook example of a propagated, person-to-person epidemic.

Step 4: Why the others are wrong. *Staphylococcal food poisoning* (option 2) and *Salmonella food poisoning* (option 4) are common-source, point-source outbreaks from contaminated food — a single sharp peak, no serial generations. *Typhoid* (option 3) is typically a common-source (water/food-borne) outbreak as well; although secondary spread can occur, it is not the classic example of a multiple-peaked propagated epidemic in the way measles is.

Key fact: A propagated epidemic with peaks one incubation period apart is characteristic of directly transmitted infections like measles.

Quick Tip: Peaks one incubation period apart = serial person-to-person spread — think a contagious virus.

25. Under the RMNCAH+ (Reproductive, Maternal, Newborn, Child and Adolescent Health) framework, districts are colour-coded for performance. Which of the following indicators, when meeting the stated criterion, would be marked as a RED card (poor performance)?

- (A) IMR less than 20%
- (B) TFR more than 20%
- (C) MMR <20%
- (D) Children receiving healthcare <20%

Correct Answer: (D) Children receiving healthcare <20%

Solution:

Step 1: Understand the RMNCAH+ colour-coding system.

Under the RMNCAH+A strategy, districts/blocks are tracked on key maternal and child health indicators and given a card colour. A **GREEN** card denotes acceptable/good performance, whereas a **RED** card flags poor performance that needs priority action.

Step 2: Interpret each indicator against its desirable direction.

For mortality indicators (IMR, MMR) a LOW value is GOOD, so <20% threshold being met = green. A lower TFR is also desirable, and a high TFR is bad - however the question frames the only indicator whose LOW value is undesirable as service coverage.

Step 3: Identify the undesirable low value.

For a service-utilisation/coverage indicator such as *children receiving healthcare*, a HIGH value is good and a LOW value is bad. If only <20% of children are receiving healthcare, coverage is grossly deficient - this is a poor-performance (RED) indicator.

Step 4: Why the others are GREEN.

IMR <20%, MMR <20% reflect low mortality (good). A low value being met is favourable for mortality indicators, hence green. Coverage falling below 20% is the lone deficiency.

Key fact: For coverage/utilisation indicators a low value (<20% children receiving healthcare) earns the RED card, while low mortality indicators earn green.

Quick Tip: For coverage indicators a LOW value is bad; for mortality indicators a LOW value is good.



26. The "lethal triad" (deadly triad) associated with massive blood transfusion classically comprises hypothermia, coagulopathy and acidosis. Which of the following is NOT a component of this classic lethal triad?

- (A) Hyperkalemia
- (B) Coagulopathy
- (C) Hypothermia
- (D) Hypocalcemia

Correct Answer: (A) Hyperkalemia

Solution:

Step 1: Define the lethal triad.

The classic *lethal/deadly triad* of massive transfusion and major trauma is **hypothermia + coagulopathy + acidosis**. These three feed back on one another, worsening bleeding and mortality.

Step 2: Map the metabolic complications of massive transfusion.

Stored blood is cold (hypothermia), citrate-anticoagulated (chelates calcium to hypocalcemia), dilutes clotting factors and platelets (coagulopathy), and leaks potassium from aged red cells (hyperkalemia), while citrate metabolism and tissue hypoperfusion contribute to acid-base shifts.

Step 3: Match options to the triad.

Coagulopathy and hypothermia are bona-fide triad members. Hypocalcemia, although a real transfusion complication, is the metabolic stand-in often paired with the triad. **Hyperkalemia** is a transfusion complication but is NOT one of the classic triad components.

Step 4: Choose the exception.

The indicator that does not belong to the classic lethal triad is **hyperkalemia**.

Key fact: Lethal triad = hypothermia, coagulopathy, acidosis; hyperkalemia is a separate transfusion-related electrolyte hazard.

Quick Tip: Triad = hypothermia, coagulopathy, acidosis - potassium is not in it.

27. Lamotrigine is a broad-spectrum antiepileptic. For which of the following seizure types is lamotrigine particularly useful and a recognised first/adjunctive choice?

- (A) Generalised tonic-clonic seizures (GTCS)
- (B) Myoclonic seizures
- (C) Absence seizures
- (D) Atonic seizures

Correct Answer: (A) Generalised tonic-clonic seizures (GTCS)

Solution:

Step 1: Recall lamotrigine's mechanism and spectrum.

Lamotrigine blocks voltage-gated sodium channels and inhibits glutamate release. It is a *broad-spectrum* agent effective in focal seizures, GTCS, and as adjunct in Lennox-Gastaut syndrome.

Step 2: Evaluate each seizure type.

• **GTCS:** Lamotrigine is effective and is a recognised choice for generalised tonic-clonic seizures.

• **Myoclonic:** Lamotrigine can *aggravate* myoclonic seizures (notably in juvenile myoclonic epilepsy), so it is not preferred.

• **Absence:** Although used, it is less effective than ethosuximide/valproate and may worsen some idiopathic generalised epilepsies.

• **Atonic:** Atonic/drop attacks are best managed within Lennox-Gastaut regimens; lamotrigine helps as adjunct but GTCS is its clearer indication.

Step 3: Select the best fit.

The seizure type for which lamotrigine is most clearly useful and a first-line generalised indication is **GTCS**.

Key fact: Lamotrigine is effective for focal seizures and GTCS but can worsen myoclonic seizures.

Quick Tip: Broad-spectrum Na-channel blocker; can aggravate myoclonus, but good



28. Cilnidipine is unique among dihydropyridine calcium channel blockers in not causing reflex tachycardia. This is because, in addition to L-type channels, it also blocks which type of voltage-gated calcium channel located on sympathetic nerve endings?

- (A) T-type
- (B) R-type
- (C) N-type
- (D) L-type

Correct Answer: (C) N-type

Solution:

Step 1: Recall the basis of reflex tachycardia with DHPs.

Conventional dihydropyridines (e.g., amlodipine, nifedipine) block *L-type* calcium channels, causing arteriolar vasodilation and a fall in blood pressure. The baroreceptor reflex then activates the sympathetic system, releasing noradrenaline and producing reflex tachycardia.

Step 2: Identify Cilnidipine's extra target.

Cilnidipine is an **L/N-type** calcium channel blocker. The **N-type** channels sit on sympathetic (postganglionic) nerve terminals and control calcium-dependent noradrenaline release.

Step 3: Link mechanism to the clinical effect.

By blocking N-type channels, Cilnidipine reduces sympathetic noradrenaline release. So even when L-type blockade lowers BP, the compensatory sympathetic surge is blunted - hence *no reflex tachycardia*.

Step 4: Eliminate the others.

T-type channels are targeted by mibefradil/efonidipine (different profile); R-type

are not the relevant sympathetic target here; L-type blockade alone is what causes the reflex problem. The answer is **N-type**.

Key fact: Cilnidipine blocks N-type Ca channels on sympathetic nerve endings, preventing reflex tachycardia.

Quick Tip: N-type channels on sympathetic terminals control noradrenaline release.

29. All of the following are ligand-gated ion channel (ionotropic) receptors EXCEPT:

- (A) 5-HT₃ receptor
- (B) Nicotinic acetylcholine receptor
- (C) GABA-A receptor
- (D) Histamine H₂ receptor

Correct Answer: (D) Histamine H₂ receptor

Solution:

Step 1: Classify receptor superfamilies.

Ligand-gated ion channels (ionotropic receptors) open an ion pore directly on ligand binding, giving fast millisecond responses. G-protein-coupled receptors (GPCRs/metabotropic) act through second messengers.

Step 2: Test each option.

• **5-HT₃:** the only serotonin receptor that is a ligand-gated cation channel - ionotropic.

• **Nicotinic ACh receptor:** a classic ligand-gated cation channel - ionotropic.

• **GABA-A:** a ligand-gated chloride channel - ionotropic.

• **Histamine H₂:** a G_s-coupled GPCR that raises cAMP (e.g., gastric acid secretion) - metabotropic, NOT a ligand-gated ion channel.

Step 3: Pick the exception.

The receptor that is not a ligand-gated ion channel is **H₂**.

Key fact: 5-HT₃, nicotinic and GABA-A are ionotropic; all histamine receptors (H₁-H₄) are GPCRs.

Quick Tip: All histamine receptors are GPCRs, not ion channels.

30. Which of the following is a classic example of a MEDIUM-vessel vasculitis?

- (A) Leukocytoclastic (cutaneous small-vessel) vasculitis
- (B) Polyarteritis nodosa (PAN)
- (C) Granulomatosis with polyangiitis (GPA)
- (D) Microscopic polyangiitis (MPA)

Correct Answer: (B) Polyarteritis nodosa (PAN)

Solution:

Step 1: Recall the Chapel Hill vasculitis classification by vessel size.

Vasculitides are grouped as large-vessel (Takayasu, giant cell), **medium-vessel** (polyarteritis nodosa, Kawasaki disease), and small-vessel (ANCA-associated and immune-complex types).

Step 2: Categorise each option.

• **Leukocytoclastic vasculitis:** a small-vessel (postcapillary venule) vasculitis - not medium.

• **PAN:** the prototypical *medium-vessel* vasculitis affecting muscular arteries, classically ANCA-negative and associated with hepatitis B.

• **GPA:** an ANCA-associated *small-vessel* vasculitis.

• **MPA:** an ANCA-associated *small-vessel* vasculitis.

Step 3: Choose the medium-vessel disease.

Among the options the medium-vessel vasculitis is **polyarteritis nodosa (PAN)**.

Key fact: PAN and Kawasaki disease are the classic medium-vessel vasculitides; GPA and MPA are small-vessel (ANCA-associated).

Quick Tip: PAN and Kawasaki = medium vessel; GPA/MPA = small-vessel ANCA-associated.

31. Which special stain is used to demonstrate fibrosis (collagen deposition) in a liver biopsy?

- (A) Masson trichrome
- (B) Periodic acid-Schiff (PAS)
- (C) Gomori methenamine silver (GMS)
- (D) Gram stain

Correct Answer: (A) Masson trichrome

Solution:

Step 1: Identify what is being asked.

The slides show liver fibrosis, and the question asks which stain best demonstrates the deposited collagen.

Step 2: Recall Masson trichrome.

Masson trichrome is a three-dye method that stains collagen and fibrous connective tissue blue or green, muscle/cytoplasm and keratin red, and nuclei dark blue/black. It is the standard stain to highlight and stage fibrosis (e.g., bridging fibrosis, cirrhosis) in liver biopsies.

Step 3: Eliminate the wrong options.

PAS stains glycogen, basement membranes and fungal walls magenta (PAS with diastase highlights alpha-1-antitrypsin globules, not fibrosis). GMS (Gomori methenamine silver) is a fungal/Pneumocystis and basement-membrane stain. Gram stain is for bacteria. None of these selectively demonstrate collagen.

Key fact: Collagen/fibrosis is best shown by **Masson trichrome** (and reticulin for the collapse pattern).

Quick Tip: Which dye method turns collagen blue/green?

32. Which of the following is the **least likely** direct cause of falls in the elderly?

- (A) Balance disturbance
- (B) Rheumatoid arthritis
- (C) Muscle weakness
- (D) Hearing impairment

Correct Answer: (D) Hearing impairment

Solution:

Step 1: Frame the question.

Falls in the elderly are multifactorial. The task is to pick the factor that is the weakest *direct* contributor among the options.

Step 2: Review the strong, direct risk factors.

Balance disturbance (vestibular/proprioceptive/cerebellar dysfunction) is one of the strongest predictors of falls. Lower-limb muscle weakness (sarcopenia) directly impairs postural recovery and is a leading modifiable risk factor. Rheumatoid arthritis causes joint deformity, pain, reduced mobility and gait instability, so it too directly raises fall risk.

Step 3: Position hearing impairment.

Hearing loss is an *associated* risk marker rather than a direct mechanical cause of falling. Its link is indirect (reduced spatial/environmental awareness, shared inner-ear pathology). Compared with balance, weakness and arthritis, it is the least direct cause.

Key fact: Among these, **hearing impairment** is the least direct cause of falls;

balance disturbance, muscle weakness and arthritis are direct, well-established risk factors.

Quick Tip: Three options directly impair posture/gait; one is only indirectly linked.

33. Frailty in the elderly is most directly characterised/affected by which of the following?

- (A) Age
- (B) Sarcopenia
- (C) Male gender
- (D) Gait alone

Correct Answer: (B) Sarcopenia

Solution:

Step 1: Define frailty.

Frailty is a state of reduced physiological reserve and increased vulnerability to stressors. The Fried phenotype defines it by five criteria: unintentional weight loss, exhaustion, weakness (low grip strength), slow walking speed and low physical activity.

Step 2: Pick the core pathophysiology.

Sarcopenia (age-related loss of skeletal muscle mass and strength) is the central biological driver of frailty; weakness, slow gait and low activity all stem from it. This makes sarcopenia the most direct determinant.

Step 3: Why not the others.

Age is a risk factor but frailty is distinct from chronological age (young-old people can be robust; some can be frail earlier). Frailty is actually *more* prevalent in women, so male gender is not the answer. Gait speed is only one of several criteria, not the underlying cause.

Key fact: Sarcopenia is the biological core of frailty.

Quick Tip: What muscle change underlies weakness, slow gait and low activity?



34. Regarding nicotine replacement therapy (NRT), which of the following statements is **correct**?

- (A) Acidic foods/beverages should be avoided for about 15 minutes before using nicotine gum or lozenge
- (B) Nicotine gum delivers more nicotine than an equivalent lozenge
- (C) Varenicline carries a black box warning for cardiovascular risk
- (D) Swallowed nicotine is well absorbed from the gastrointestinal tract

Correct Answer: (A) Acidic foods/beverages should be avoided for about 15 minutes before using nicotine gum or lozenge

Solution:

Step 1: Recall how NRT is absorbed.

Nicotine from gums and lozenges is absorbed across the buccal (oral) mucosa, which requires an alkaline pH. An acidic mouth environment ionises nicotine and blocks absorption, so patients are told to avoid coffee, juice and soft drinks for about 15 minutes before and during use. **Statement 1 is correct.**

Step 2: Gum versus lozenge.

For a given labelled dose, the lozenge actually delivers roughly 25% MORE nicotine than the gum (it dissolves completely with no chewing loss). So 'gum delivers more than lozenge' is wrong.

Step 3: Varenicline black box.

Varenicline's black box warning was for serious neuropsychiatric symptoms, and that boxed warning was removed by the FDA in 2016 after the EAGLES trial. It is not a cardiovascular black box warning, so that statement is incorrect.

Step 4: GI absorption.

Swallowed nicotine is poorly absorbed and undergoes extensive hepatic first-pass metabolism; NRT works through buccal/transdermal/nasal routes, not the GI route. Incorrect.

Key fact: The single true statement is that **acidic food/drink must be avoided ~15 minutes before oral NRT.**

Quick Tip: Buccal nicotine needs an alkaline pH to be absorbed.

35. Which of the following receptors is coupled to the stimulatory G protein (Gs)?

- (A) M2 muscarinic receptor
- (B) M1 muscarinic receptor
- (C) Beta-adrenergic receptor
- (D) Alpha-2-adrenergic receptor

Correct Answer: (C) Beta-adrenergic receptor

Solution:

Step 1: Recall G-protein coupling.

Gs (stimulatory) activates adenylyl cyclase and raises cyclic AMP. Gi (inhibitory) lowers cAMP. Gq activates phospholipase C, raising IP3/DAG and intracellular calcium.

Step 2: Classify each receptor.

Beta-adrenergic receptors (beta-1, beta-2, beta-3) are the classic **Gs**-coupled receptors, increasing cAMP. M2 muscarinic and alpha-2-adrenergic receptors are Gi-coupled (decrease cAMP). M1 (and M3/M5) muscarinic receptors are Gq-coupled.

Step 3: Choose the Gs receptor.

Only the beta-adrenergic receptor uses Gs.

Key fact: Beta-adrenergic = Gs; M2 and alpha-2 = Gi; M1 = Gq.

Quick Tip: Which catecholamine receptor raises cAMP?

36. Match each targeted anticancer drug with its molecular target. Which of the following pairings is entirely correct?

- (A) Sunitinib - c-KIT/VEGFR; Nilotinib - BCR-ABL; Trastuzumab - HER2; Gefitinib - EGFR
- (B) Sunitinib - HER2; Nilotinib - EGFR; Trastuzumab - BCR-ABL; Gefitinib - c-KIT
- (C) Sunitinib - BCR-ABL; Nilotinib - c-KIT; Trastuzumab - EGFR; Gefitinib - HER2
- (D) Sunitinib - EGFR; Nilotinib - HER2; Trastuzumab - c-KIT; Gefitinib - BCR-ABL

Correct Answer: (A) Sunitinib - c-KIT/VEGFR; Nilotinib - BCR-ABL; Trastuzumab - HER2; Gefitinib - EGFR

Solution:

Step 1: Identify each drug's target.

Sunitinib is a multi-kinase tyrosine kinase inhibitor acting on VEGFR, PDGFR and **c-KIT** (used in renal cell carcinoma and imatinib-resistant GIST).

Nilotinib is a second-generation **BCR-ABL** tyrosine kinase inhibitor for chronic myeloid leukaemia.

Trastuzumab is a monoclonal antibody against **HER2 (ErbB2)**, used in HER2-positive breast and gastric cancer.

Gefitinib is an **EGFR** tyrosine kinase inhibitor for EGFR-mutant non-small-cell lung cancer.

Step 2: Match.

Sunitinib-c-KIT, Nilotinib-BCR-ABL, Trastuzumab-HER2, Gefitinib-EGFR. This is option 1.

Step 3: Reject the rest.

Every other option scrambles at least two targets, so only option 1 is fully correct.

Key fact: Sunitinib to c-KIT/VEGFR, Nilotinib to BCR-ABL, Trastuzumab to HER2, Gefitinib to EGFR.

Quick Tip: Nilotinib treats CML; trastuzumab targets HER2 breast cancer.

37. Regarding the pathophysiology of beta-thalassemia major, which of the following correctly describes the disease mechanism?

- (A) Ineffective (defective) erythropoiesis
- (B) Hepcidin is markedly increased
- (C) Hepcidin is suppressed, causing iron overload
- (D) Predominantly intravascular hemolysis

Correct Answer: (A) Ineffective (defective) erythropoiesis

Solution:

Step 1: Basic defect. In beta-thalassemia major there is severely reduced or absent synthesis of beta-globin chains. The unpaired alpha-globin chains precipitate within erythroid precursors in the bone marrow.

Step 2: Ineffective erythropoiesis. These alpha-chain inclusions damage and cause apoptosis of red cell precursors before they mature, so erythropoiesis is markedly expanded but largely **ineffective (defective)**. This is the central, defining mechanism and the best single answer.

Step 3: Why the others are wrong. Hepcidin is **not increased** - the expanded ineffective erythropoiesis releases erythroferrone, which **suppresses hepcidin**; this drives increased gut iron absorption and (together with transfusions) causes iron overload. So option 2 is false, and although option 3 (suppressed hepcidin / iron overload) is a real downstream consequence, it is secondary to the primary

erythroid defect. Hemolysis in thalassemia is predominantly **extravascular** (splenic clearance of defective cells) plus intramedullary destruction, not chiefly intravascular, so option 4 is incorrect.

Key fact: Ineffective erythropoiesis from precipitated alpha-chains is the core pathology of beta-thalassemia major; erythroferrone-mediated hepcidin suppression and iron overload follow from it.

Quick Tip: Unpaired alpha-chains kill precursors in the marrow before they mature.

38. Regarding anti-ageing/longevity drugs and their molecular targets, which of the following pairs is **incorrectly** matched?

- (A) Rapamycin - mTOR inhibitor
- (B) Metformin - AMPK activator
- (C) Resveratrol - Sirtuin (SIRT1) activator
- (D) Spermidine - decreases autophagy

Correct Answer: (D) Spermidine - decreases autophagy

Solution:

Step 1: Identify the theme. The question tests the molecular targets of candidate longevity/anti-ageing agents that act on conserved nutrient-sensing and autophagy pathways.

Step 2: Check each true pair. Rapamycin inhibits **mTOR**, extending lifespan in models (option 1 correct). Metformin activates **AMPK** (and indirectly inhibits mTOR), an energy-sensing kinase (option 2 correct). Resveratrol activates **sirtuins (SIRT1)**, NAD⁺-dependent deacetylases (option 3 correct).

Step 3: Find the error. Spermidine is a well-known autophagy **inducer** - it **increases** autophagy (largely via inhibition of acetyltransferase EP300), promoting clearance of damaged organelles. The statement that spermidine

decreases autophagy is therefore wrong, making option 4 the incorrectly matched pair and the answer.

Key fact: Spermidine is a classic autophagy *enhancer*, not a suppressor.

Quick Tip: Spermidine is famous for switching autophagy ON, not off.

39. A child with Dravet syndrome is started on fenfluramine for seizure control. Which of the following must be specifically monitored during therapy?

- (A) Cardiovascular status (echocardiography for valvulopathy/pulmonary hypertension)
- (B) Agranulocytosis
- (C) Hepatic failure
- (D) Renal failure

Correct Answer: (A) Cardiovascular status (echocardiography for valvulopathy/pulmonary hypertension)

Solution:

Step 1: Drug and indication. Fenfluramine is approved for seizures associated with Dravet syndrome (and Lennox-Gastaut syndrome). It acts via serotonin (5-HT) release and agonism at 5-HT receptors plus sigma-1 activity.

Step 2: Class-specific toxicity. Fenfluramine was historically withdrawn as an appetite suppressant because of serotonin-mediated **cardiac valvulopathy and pulmonary arterial hypertension**. Even at antiseizure doses this risk mandates **cardiac monitoring** - baseline and periodic **echocardiography** to detect valvular regurgitation and pulmonary hypertension. This makes option 1 correct.

Step 3: Why the others are wrong. Agranulocytosis is characteristic of clozapine/carbamazepine, not fenfluramine (option 2 wrong). Idiosyncratic hepatic failure is the concern with valproate/felbamate (option 3 wrong).

Significant nephrotoxicity is not a defining fenfluramine effect (option 4 wrong).

Key fact: Echocardiographic cardiovascular monitoring (valvulopathy and pulmonary hypertension) is the mandated safety surveillance for fenfluramine.

Quick Tip: Remember why fen-phen was pulled from the diet-pill market.

40. Renin secretion from the juxtaglomerular apparatus is stimulated in response to:

- (A) Elevated sodium delivery to the macula densa
- (B) Elevated renal perfusion pressure
- (C) Reduced renal perfusion pressure
- (D) Increased extracellular fluid volume

Correct Answer: (C) Reduced renal perfusion pressure

Solution:

Step 1: Triggers of renin release. Renin is released by juxtaglomerular (granular) cells of the afferent arteriole in response to three signals: (i) the **renal baroreceptor** sensing low afferent arteriolar/perfusion pressure, (ii) **decreased NaCl** delivery sensed by the macula densa, and (iii) **sympathetic (beta₁) stimulation**.

Step 2: Apply to options. A fall in renal perfusion pressure (e.g., hypovolemia, hypotension, renal artery stenosis) reduces stretch of the JG baroreceptor and increases renin - option 3 is correct.

Step 3: Why the others are wrong. Elevated sodium at the macula densa **inhibits** renin (tubuloglomerular feedback), so option 1 is wrong. Elevated perfusion pressure and an increased ECF/blood volume both **suppress** renin, so options 2 and 4 are wrong.

Key fact: Renin rises when renal perfusion pressure, distal NaCl delivery fall, or sympathetic drive increases - here, **reduced renal perfusion pressure**.

Quick Tip: The JG baroreceptor fires renin when it senses LOW pressure/stretch.



41. A patient has Hb 9 g/dL, MCV 68 fL and a low serum ferritin. The most likely type of anemia is:

- (A) Iron deficiency anemia
- (B) Megaloblastic anemia
- (C) Aplastic anemia
- (D) Hemolytic anemia

Correct Answer: (A) Iron deficiency anemia

Solution:

Step 1: Classify by MCV. An MCV of 68 fL (normal ~80-100 fL) indicates a **microcytic** anemia. Causes include iron deficiency, thalassemia, anemia of chronic disease, and sideroblastic anemia.

Step 2: Use the ferritin. Serum ferritin is the key discriminator of iron stores. A **low ferritin** is essentially diagnostic of **depleted iron stores**, i.e., iron deficiency anemia. (Ferritin is high/normal in thalassemia and anemia of chronic disease.)

Step 3: Eliminate others. Megaloblastic anemia (B12/folate) is **macrocytic** (high MCV) - opposite picture, so option 2 is wrong. Aplastic anemia is typically **normocytic/macrocytic** with pancytopenia and normal-to-high ferritin (option 3 wrong). Hemolytic anemias are usually **normocytic** with raised reticulocytes, LDH and bilirubin, not low ferritin (option 4 wrong).

Key fact: Microcytic anemia (low MCV) with a **low ferritin** = iron deficiency anemia.

Quick Tip: Low MCV + low ferritin is the classic iron-deficiency signature.

42. A patient has symptomatic hypercalcemia due to malignancy. Which of the following is the most appropriate **first** step in acute management?

- (A) Administration of bisphosphonates
- (B) Intravenous fluids (isotonic saline volume expansion)
- (C) Loop diuretics
- (D) Find the source of exogenous calcium

Correct Answer: (B) Intravenous fluids (isotonic saline volume expansion)

Solution:

Step 1: Recognise the problem. Hypercalcemia of malignancy is usually accompanied by significant **volume depletion**, because hypercalcemia impairs renal concentrating ability and causes a nephrogenic diabetes insipidus-like polyuria, plus reduced intake from nausea/vomiting.

Step 2: First step = rehydrate. The immediate priority is **aggressive isotonic saline (IV fluid) volume expansion**. Restoring intravascular volume increases glomerular filtration and promotes calciuresis, rapidly lowering serum calcium. This makes option 2 the correct first step.

Step 3: Sequence the rest. **Bisphosphonates** (e.g., zoledronate) treat the underlying osteoclastic bone resorption but take 2-4 days to work and are given *after/with* hydration, not first (option 1). **Loop diuretics** are no longer routine and only after the patient is volume-replete (and mainly if volume overload/heart failure supervenes), so option 3 is wrong as the first step. Hypercalcemia of malignancy is endogenous (PTHrP, osteolysis, calcitriol), so hunting for exogenous calcium is irrelevant here (option 4).

Key fact: Begin treatment of malignant hypercalcemia with vigorous IV isotonic saline; add bisphosphonates (+/- calcitonin) thereafter.

Quick Tip: These patients are dry - fix the volume first, then block resorption.

43. Hyperpolarization of the receptor cell on stimulation is characteristically seen with which of the following sensory modalities?

- (A) Olfactory
- (B) Visual
- (C) Taste
- (D) Tactile

Correct Answer: (B) Visual

Solution:

Step 1: Identify the unique receptor.

Almost all sensory receptors depolarize when stimulated, generating a depolarizing receptor (generator) potential. The photoreceptors of the retina (rods and cones) are the classic exception.

Step 2: Phototransduction in the dark.

In darkness, cyclic GMP keeps cation ($\text{Na}^+/\text{Ca}^{2+}$) channels open, producing a steady inward 'dark current' that keeps the photoreceptor partially depolarized (around -40 mV) and releasing glutamate continuously.

Step 3: Effect of light.

Light activates rhodopsin $\rightarrow$ transducin $\rightarrow$ phosphodiesterase, which hydrolyses cGMP. Falling cGMP closes the cation channels, the inward current stops, and the cell **hyperpolarizes**, reducing glutamate release. Thus the visual receptor responds to its adequate stimulus with hyperpolarization.

Step 4: Why the others are wrong.

Olfactory receptors depolarize via cAMP-gated cation channels; taste receptors depolarize through various transduction routes; tactile (mechanoreceptor) endings depolarize via stretch-activated cation channels. None hyperpolarize on

stimulation.

Key fact: Photoreceptors are unique in hyperpolarizing in response to light.

Quick Tip: Think of the receptor that turns off its 'dark current' when stimulated.

44. In refeeding syndrome, which electrolyte abnormality is the most important and characteristic?

- (A) Hypocalcemia
- (B) Hypophosphatemia
- (C) Hypokalemia
- (D) Hyponatremia

Correct Answer: (B) Hypophosphatemia

Solution:

Step 1: Define refeeding syndrome.

It is the constellation of metabolic and electrolyte shifts that occur when nutrition (especially carbohydrate) is reintroduced to a chronically malnourished or starved patient.

Step 2: Mechanism.

Carbohydrate refeeding triggers an insulin surge. Insulin drives glucose, phosphate, potassium and magnesium intracellularly and stimulates synthesis of ATP and 2,3-DPG, which consume phosphate. In a body already depleted of total-body phosphate, this causes a sharp fall in serum phosphate.

Step 3: The hallmark abnormality.

Hypophosphatemia is the defining biochemical feature of refeeding syndrome and is responsible for its most dangerous complications — rhabdomyolysis, respiratory and cardiac failure, hemolysis, seizures and arrhythmias.

Step 4: Why the others are wrong.

Hypokalemia and hypomagnesemia also occur and matter, but they are not the signature defect. Hypocalcemia and hyponatremia are not central to the syndrome. Phosphate is the key monitored and replaced electrolyte.

Key fact: Hypophosphatemia is the hallmark of refeeding syndrome — monitor and replace phosphate, and start refeeding slowly.

Quick Tip: Insulin-driven ATP synthesis depletes which already-low anion?

45. Regarding Extended-Spectrum Beta-Lactamase (ESBL)-producing organisms, which of the following statements are TRUE?

- (A) Carbapenems can be used for treatment
- (B) Resistance is plasmid-transmitted
- (C) Piperacillin-tazobactam is the reliable drug of choice
- (D) Third-generation cephalosporins can be used

Correct Answer: (A) Carbapenems can be used for treatment

Solution:

Step 1: What ESBLs do.

Extended-spectrum beta-lactamases are enzymes that hydrolyse penicillins, oxyimino-cephalosporins (3rd-generation cephalosporins such as ceftriaxone, ceftazidime, cefotaxime) and aztreonam. They are inhibited by clavulanic acid in vitro but this does not translate into reliable clinical efficacy.

Step 2: Statement on carbapenems (TRUE).

Carbapenems (meropenem, imipenem, ertapenem) are the drugs of choice for serious ESBL infections — they are stable to ESBL hydrolysis.

Step 3: Statement on plasmid transmission (TRUE).

ESBL genes (e.g. CTX-M, TEM, SHV) are carried on **plasmids**, allowing

horizontal spread and co-resistance to other antibiotic classes.

Step 4: Why piptaz and 3rd-gen cephalosporins are wrong.

By definition ESBLs hydrolyse 3rd-generation cephalosporins, so these must NOT be used even if the lab reports apparent susceptibility. Piperacillin-tazobactam is not a reliable first-line agent for serious ESBL infection (inoculum effect, variable outcomes), so it is not the correct answer.

Key fact: ESBL = plasmid-mediated, hydrolyses 3rd-gen cephalosporins; treat serious infection with a carbapenem.

Quick Tip: Which enzyme spreads on plasmids and is beaten only by carbapenems?



46. In which region of the adult human brain can neurons regenerate and increase in number (adult neurogenesis)?

- (A) Ventral part of spinal cord
- (B) Dentate gyrus of the hippocampus
- (C) Substantia nigra
- (D) Locus ceruleus

Correct Answer: (B) Dentate gyrus of the hippocampus

Solution:

Step 1: The concept of adult neurogenesis.

The mammalian central nervous system was long thought to be post-mitotic, but two niches retain neural stem cells capable of producing new neurons throughout adult life.

Step 2: The two recognised niches.

These are (a) the **subgranular zone of the dentate gyrus** of the hippocampus, which generates new granule-cell neurons important for learning and memory, and (b) the subventricular zone of the lateral ventricles, which feeds the rostral

migratory stream to the olfactory bulb.

Step 3: Apply to the options.

The **dentate gyrus** is the classic answer and the only listed site of ongoing adult neurogenesis.

Step 4: Why the others are wrong.

The ventral spinal cord, substantia nigra (dopaminergic; its loss causes Parkinson disease and is not replenished) and locus ceruleus (noradrenergic) do not generate new neurons in the adult — their neuronal loss is permanent.

Key fact: Adult neurogenesis occurs in the dentate gyrus (hippocampus) and the subventricular zone.

Quick Tip: Which memory-related hippocampal structure keeps making granule cells?

47. Lesion at which site is associated with impaired emotional prosody (loss of emotional tone/melody of speech)?

- (A) Cingulate gyrus
- (B) Superior temporal gyrus
- (C) Parietal lobe
- (D) Precentral gyrus

Correct Answer: (A) Cingulate gyrus

Solution:

Step 1: Define emotional prosody.

Prosody is the melodic, rhythmic and emotional intonation of speech that conveys feeling beyond the literal words. Emotional (affective) prosody is processed largely by the non-dominant (right) hemisphere and limbic structures.

Step 2: Role of the cingulate gyrus.

The **anterior cingulate gyrus** is a core limbic region governing the emotional and motivational content of vocalization and the affective coloring of speech. Lesions here impair the generation and modulation of emotional prosody (aprosodia), causing flat, monotonous, affectless speech.

Step 3: Apply to the options.

Among the choices, the cingulate gyrus is the limbic structure tied to emotional prosody, making it the best answer for the site of lesion.

Step 4: Why the others are wrong.

The superior temporal gyrus relates to auditory/receptive language (Wernicke), the parietal lobe to somatosensation and integration, and the precentral gyrus to primary motor output of articulation — none is the primary seat of affective prosody.

Key fact: The cingulate gyrus, a limbic structure, governs the emotional tone of speech (emotional prosody).

Quick Tip: Which limbic gyrus gives speech its emotional color?

48. High serum triglycerides are seen in all of the following conditions EXCEPT:

- (A) High fructose intake
- (B) Diabetes mellitus
- (C) Nephrotic syndrome
- (D) Abetalipoproteinemia

Correct Answer: (D) Abetalipoproteinemia

Solution:

Step 1: Read the 'except' stem.

Three options raise triglycerides; one lowers them. Identify the outlier.

Step 2: Conditions that raise triglycerides.

High fructose drives hepatic de novo lipogenesis and VLDL output, raising TG.

Diabetes mellitus (insulin deficiency/resistance) reduces lipoprotein lipase activity and increases VLDL, causing hypertriglyceridemia. **Nephrotic syndrome** increases hepatic lipoprotein synthesis (including VLDL) in response to hypoalbuminemia, raising both cholesterol and triglycerides.

Step 3: The exception.

Abetalipoproteinemia is caused by a defect in microsomal triglyceride transfer protein (MTP), so the liver and intestine cannot assemble apoB-containing lipoproteins (chylomicrons, VLDL, LDL). Triglycerides and cholesterol in plasma are therefore very LOW, not high. This is the exception.

Step 4: Conclusion.

Abetalipoproteinemia is the only listed condition associated with low, not high, triglycerides.

Key fact: MTP deficiency in abetalipoproteinemia blocks apoB lipoprotein assembly → absent chylomicrons/VLDL/LDL → low plasma TG.

Quick Tip: Which lipid disorder cannot even export triglyceride from the liver/gut?

49. Ultraviolet (UV) radiation causes DNA damage primarily by which of the following mechanisms?

- (A) Formation of pyrimidine dimers
- (B) Inhibition of p53
- (C) Deamination of cytosine
- (D) Increase in cyclins

Correct Answer: (A) Formation of pyrimidine dimers

Solution:

Step 1: Identify the lesion produced by UV light.

UV-B radiation (wavelength 280-320 nm) is directly absorbed by adjacent pyrimidine bases on the same DNA strand. This induces covalent linkage between two neighbouring pyrimidines (most commonly thymine), producing **cyclobutane pyrimidine dimers** (and 6-4 photoproducts).

Step 2: Effect of the dimer.

The dimer distorts the DNA double helix and blocks both replication and transcription. If unrepaired it leads to mutations, especially C to T transitions, which underlie UV-induced skin cancers.

Step 3: Repair pathway.

Pyrimidine dimers are removed by **nucleotide excision repair (NER)**. A defect in NER causes xeroderma pigmentosum, characterised by extreme photosensitivity and early skin malignancy.

Step 4: Why the other options are wrong.

• *Inhibition of p53* - p53 mutation can result from UV damage but is a downstream consequence, not the direct lesion.

• *Deamination of cytosine* - this is a spontaneous hydrolytic event (or caused by nitrous acid), giving uracil; it is not the UV mechanism.

• *Increase in cyclins* - unrelated to the primary DNA photolesion.

Key fact: UV light damages DNA by forming pyrimidine (thymine) dimers, repaired by nucleotide excision repair.

Quick Tip: Think of the lesion repaired by nucleotide excision repair and defective in xeroderma pigmentosum.

50. Which vitamin-derived coenzyme is required for a transamination reaction?

- (A) Thiamine (Vitamin B1)
- (B) Pyridoxine (Vitamin B6)
- (C) FAD (Riboflavin / Vitamin B2)
- (D) NAD (Niacin / Vitamin B3)

Correct Answer: (B) Pyridoxine (Vitamin B6)

Solution:

Step 1: Define transamination.

Transamination is the transfer of an amino group ($-\text{NH}_2$) from an amino acid to an alpha-keto acid (usually alpha-ketoglutarate), generating a new amino acid (glutamate) and a new keto acid. It is catalysed by aminotransferases (transaminases) such as ALT and AST.

Step 2: Identify the coenzyme.

All transaminases use **pyridoxal phosphate (PLP)**, the active coenzyme form of **pyridoxine (Vitamin B6)**. PLP forms a Schiff base (aldimine) with the amino acid and shuttles the amino group via a pyridoxamine intermediate.

Step 3: Why the other options are wrong.

• *Thiamine (B1)* to thiamine pyrophosphate is needed for oxidative decarboxylation (pyruvate/alpha-ketoglutarate dehydrogenase, transketolase), not transamination.

• *FAD (B2)* to a hydrogen carrier in oxidation-reduction reactions (e.g. succinate dehydrogenase).

• *NAD (B3)* to a hydride acceptor in dehydrogenase reactions, not amino-group transfer.

Key fact: Transamination requires pyridoxal phosphate, derived from Vitamin B6 (pyridoxine).

Quick Tip: The coenzyme that forms a Schiff base with amino acids is pyridoxal phosphate.

51. Which technique is best suited for identifying a specific protein in a formalin-fixed paraffin-embedded (FFPE) tissue section?

- (A) Immunohistochemistry (IHC)
- (B) Fluorescence in situ hybridization (FISH)
- (C) Western blot
- (D) Flow cytometry

Correct Answer: (A) Immunohistochemistry (IHC)

Solution:

Step 1: Understand the substrate.

An FFPE section is a fixed, paraffin-embedded tissue slice mounted on a slide, preserving tissue architecture. The question asks specifically about detecting a **protein** while keeping it *in situ*.

Step 2: Match the technique.

Immunohistochemistry (IHC) uses antibodies directed against the target protein, with a chromogenic (e.g. DAB) or enzymatic readout, applied directly to the tissue section. It localises the protein within specific cells/compartments while preserving morphology - ideally suited to FFPE sections.

Step 3: Why the other options are wrong.

- *FISH* - detects nucleic acids (DNA/RNA sequences), not proteins.
- *Western blot* - detects proteins but requires tissue lysis/homogenisation and gel electrophoresis; it loses spatial/architectural information and is not performed on intact sections.
- *Flow cytometry* - requires a single-cell suspension, not a solid fixed section.

Key fact: IHC is the method of choice for in-situ protein identification on FFPE tissue.

Quick Tip: You need antibody-based protein detection that keeps tissue architecture

intact.

52. Which bacterium possesses two circular chromosomes?

- (A) *Escherichia coli*
- (B) *Vibrio cholerae*
- (C) *Mycobacterium tuberculosis*
- (D) *Mycoplasma genitalium*

Correct Answer: (B) *Vibrio cholerae*

Solution:

Step 1: Recall the usual bacterial genome.

Most bacteria carry a **single circular chromosome**. *E. coli*, *M. tuberculosis* and *Mycoplasma genitalium* all follow this pattern (the last having one of the smallest genomes known).

Step 2: Identify the exception.

Vibrio cholerae is the classic example of a bacterium with **two distinct circular chromosomes** - a large chromosome 1 carrying most essential genes and a smaller chromosome 2 (originally a megaplasmid that became indispensable). This divided genome is a favourite exam point.

Step 3: Why the other options are wrong.

- *E. coli* - one circular chromosome (plus optional plasmids).
- *M. tuberculosis* - single circular chromosome, GC-rich.
- *Mycoplasma genitalium* - single tiny circular chromosome, no cell wall.

Key fact: *Vibrio cholerae* uniquely has two circular chromosomes among the options.

Quick Tip: The comma-shaped cholera bacterium famously has a divided (two-chromosome) genome.

53. A middle-aged woman from Assam presents with chronic cough, fever, weight loss and cavitary lung lesions. Lung biopsy shows granulomatous inflammation with caseous necrosis and histiocyte-rich infiltrates. Which of the following should be strongly considered in the differential diagnosis?

- (A) Sarcoidosis
- (B) Aspergillosis
- (C) Histoplasmosis
- (D) Granulomatosis with polyangiitis

Correct Answer: (C) Histoplasmosis

Solution:

Step 1: Recognise the clinical syndrome.

Chronic cough, fever, weight loss and cavitary lesions in a patient from Assam (North-East India) is a classic tuberculosis-mimic picture. The histology - granulomas with **caseous necrosis** and abundant **histiocytes** - raises an endemic dimorphic fungus.

Step 2: Geographic and histological clue.

North-East India (Assam, Manipur) is an endemic focus for **histoplasmosis**. *Histoplasma capsulatum* is an intracellular dimorphic yeast that proliferates inside histiocytes/macrophages, producing histiocyte-rich, necrotising granulomas that closely mimic tuberculosis. The small intracellular yeasts are seen on GMS/PAS stains.

Step 3: Why the other options are less fitting.

• *Sarcoidosis* - gives **non-caseating** granulomas; caseous necrosis argues against it.

• *Aspergillosis* - typically an aspergilloma in a pre-existing cavity or angio-invasive disease with septate acute-angle hyphae, not histiocyte-rich caseating granulomas.

• *Granulomatosis with polyangiitis* - necrotising vasculitis with geographic necrosis and giant cells, usually with sinonasal/renal involvement, not the

histiocyte-rich endemic-fungus picture.

Key fact: An Assam patient with TB-like caseating, histiocyte-rich granulomas should prompt strong consideration of histoplasmosis.

Quick Tip: An endemic dimorphic fungus of North-East India that grows inside histiocytes and mimics TB.



54. Which of the following antifungal agents is NOT effective for the treatment of mucormycosis?

- (A) Isavuconazole
- (B) Posaconazole
- (C) Voriconazole
- (D) Amphotericin B

Correct Answer: (C) Voriconazole

Solution:

Step 1: Recall first-line therapy for mucormycosis.

Mucormycosis (caused by Mucorales - Rhizopus, Mucor, etc.) is treated primarily with **liposomal amphotericin B** plus aggressive surgical debridement and control of the predisposing condition (e.g. diabetic ketoacidosis).

Step 2: Salvage / step-down azoles.

Among the azoles, only **posaconazole** and **isavuconazole** have reliable activity against Mucorales and are used as step-down or salvage therapy.

Step 3: Identify the ineffective drug.

Voriconazole has NO activity against Mucorales. In fact, voriconazole prophylaxis (used against Aspergillus) is a recognised risk factor for breakthrough mucormycosis. Hence it is the drug that should *not* be given.

Step 4: Why the other options are wrong (they ARE effective).

• *Amphotericin B* - drug of choice.

• *Posaconazole* - effective, used for step-down/salvage.

• *Isavuconazole* - effective, FDA-approved for mucormycosis.

Key fact: Voriconazole does not cover Mucorales and is ineffective in mucormycosis.

Quick Tip: The azole used against *Aspergillus* that actually predisposes to breakthrough Mucorales infection.

55. The trabecular meshwork of the eye is composed of which of the following components? (Select all that apply)

- (A) Uveal meshwork
- (B) Corneoscleral meshwork
- (C) Juxtacanalicular (pericanalicular) meshwork
- (D) Canal of Schlemm

Correct Answer: (A) Uveal meshwork ; (B) Corneoscleral meshwork ; (C) Juxtacanalicular (pericanalicular) meshwork

Solution:

Step 1: Recall the structure of the trabecular meshwork.

The trabecular meshwork (TM) is a sieve-like structure located in the anterior chamber angle (at the iridocorneal junction). It is the principal route of conventional aqueous humour outflow, draining aqueous toward the canal of Schlemm.

Step 2: Identify the three classic layers of the TM.

From inside (anterior chamber) outward, the TM has three histological parts:

1. **Uveal meshwork** - the innermost portion, adjacent to the anterior chamber, with large irregular spaces.

2. **Corneoscleral meshwork** - the middle and largest portion, extending from the scleral spur to the anterior wall of the scleral sulcus.

3. **Juxtacanalicular (pericanalicular / cribriform) meshwork** - the outermost portion lying immediately adjacent to the inner wall of Schlemm canal; this offers the **greatest resistance** to aqueous outflow.

Step 3: Evaluate each option.

• Uveal meshwork - correct, it is the innermost layer.

• Corneoscleral meshwork - correct, it is the middle layer (note: the recall “uveo-scleral mesh” is a typo for this layer; the uveoscleral pathway is a separate outflow route, but the meshwork layer meant is the corneoscleral mesh).

• Juxtacanalicular/pericanalicular meshwork - correct, outermost layer.

• Canal of Schlemm - this is the collecting channel that **receives** aqueous after it passes through the TM; it is NOT a part of the meshwork itself.

Step 4: Key fact.

The trabecular meshwork has three layers - uveal, corneoscleral and juxtacanalicular - while the canal of Schlemm is a separate downstream structure.

Quick Tip: Three layers of trabecular meshwork; Schlemm canal is a downstream channel, not part of the mesh.

56. On entering a dark room (dark adaptation), which of the following physiological changes occur in the eye? (Select all that apply)

- (A) Conversion of all-trans retinol towards regeneration of rhodopsin photopigment
- (B) Mydriasis (pupillary dilatation)
- (C) Photopsin and retinal recombine to form rhodopsin (not the reverse)
- (D) Increased sensitivity of the retina to light

Correct Answer: (A) Conversion of all-trans retinol towards regeneration of rhodopsin photopigment ; (B) Mydriasis (pupillary dilatation) ; (D) Increased sensitivity of the retina to light

Solution:

Step 1: Understand dark adaptation.

When a person moves from bright light into a dark room, the retina gradually becomes more sensitive over ~20-30 minutes. This is mediated by regeneration of visual pigments (rhodopsin in rods) and pupillary dilatation.

Step 2: Analyse each statement.

- **Mydriasis** - correct. In darkness the pupil dilates to allow more light onto the retina. This is an early, rapid component of dark adaptation.

- **Increased sensitivity to light** - correct. Regenerated rhodopsin and the shift to rod-dominated (scotopic) vision markedly increase retinal sensitivity.

- **Retinol channelled to regenerate rhodopsin** - correct. In the visual cycle, 11-cis retinal (derived from vitamin A / retinol) recombines with opsin to re-form rhodopsin. So vitamin A metabolism feeds pigment regeneration in the dark.

- **Rhodopsin/opsin to photopsin conversion** - INCORRECT as written.

Bleaching of rhodopsin into opsin + all-trans retinal happens in LIGHT, not in the dark. In darkness the reverse occurs (rhodopsin is re-formed).

“Photopsin” refers to cone opsins, not the rod bleaching product, so option 3 is the wrong/false statement.

Step 3: Key fact.

Dark adaptation = pupillary dilatation + regeneration of rhodopsin (using vitamin A) + heightened retinal sensitivity. Bleaching of pigment is a light-phase event, so option 3 is false.

Quick Tip: Dark = pupil dilates, rhodopsin regenerates, sensitivity rises; bleaching to opsin happens in light.

57. A young adult sustains chest trauma with haemodynamic instability and bilateral femur and tibia fractures. Once the patient has been resuscitated and is haemodynamically stabilized, what is the most appropriate definitive management of the fractures?

(Figure: X-ray of the limb showing the long-bone fracture.)

- (A) Intramedullary nailing of both femurs and external fixation of the tibiae
- (B) Plating of the tibiae and external fixation of the femurs
- (C) Nailing / plating / external fixation chosen by whichever procedure the surgeon can perform fastest
- (D) External fixation of both femurs and both tibiae

Correct Answer: (A) Intramedullary nailing of both femurs and external fixation of the tibiae

Solution:

Step 1: Recognise the clinical setting.

A polytrauma patient with chest injury, haemodynamic instability and multiple long-bone fractures is at high risk for fat embolism, ARDS and the ‘second hit’ of major surgery. Management follows damage-control orthopaedics (DCO) initially, then conversion to definitive fixation once the patient is physiologically stable.

Step 2: Apply the early-total-care vs damage-control principle.

The question specifies that the patient is **already haemodynamically stabilized**. A stable patient tolerates definitive fixation. For long-bone shaft fractures the definitive treatment of choice is **intramedullary (IM) nailing**, which gives early mobilisation, low infection rates and excellent union.

Step 3: Choose the correct fixation per bone.

- **Femur shaft** - closed IM nailing is the gold standard definitive treatment.
- **Tibia** - in a polytrauma patient who has just been stabilized, with chest injury and the systemic insult already sustained, the tibiae are best controlled with **external fixation** as a staged/definitive option, especially where soft-tissue

status is uncertain.

This combination (nail the femurs, ex-fix the tibiae) prioritises the most beneficial procedure for the femoral fractures while limiting the surgical burden on the tibiae.

Step 4: Why the other options are wrong.

• Option 2 (plate tibia, ex-fix femur) - femoral shaft fractures are best nailed, not externally fixed, in a stable patient; plating tibia adds soft-tissue insult.

• Option 3 (fastest procedure by surgeon preference) - speed-based choice is a damage-control concept for an UNSTABLE patient; here the patient is stabilized, so definitive evidence-based fixation is indicated.

• Option 4 (ex-fix everything) - appropriate only as temporary damage control in an unstable patient, not as definitive care once stabilized.

Step 5: Key fact.

In a stabilized polytrauma patient, definitive care of femoral shaft fractures is IM nailing; the tibiae can be managed with external fixation - hence nailing of the femurs with external fixation of the tibiae.

Quick Tip: Stabilized polytrauma: nail the femurs (gold standard), ex-fix the tibiae.



58. A young adult presents with chest trauma, haemodynamic instability and fractures of bilateral femur and tibia. After the patient has been resuscitated and rendered haemodynamically stable, what is the most appropriate definitive management of the long-bone fractures?

- (A) Intramedullary nailing of the femurs and external fixation of the tibiae
- (B) Plating of the tibiae and external fixation of the femurs
- (C) Nailing / plating / external fixation as per whichever procedure the surgeon can perform fastest
- (D) External fixation of both femurs and both tibiae

Correct Answer: (A) Intramedullary nailing of the femurs and external fixation of the tibiae

Solution:

Step 1: Define the scenario.

This is essentially the same polytrauma case - chest trauma, initial haemodynamic instability, and bilateral femoral and tibial fractures - with management decided AFTER the patient is haemodynamically stabilized.

Step 2: Recall the staged-management framework.

An unstable polytrauma patient is first managed by **damage-control orthopaedics** (temporary external fixation, minimal physiological burden). Once **stabilized**, definitive fixation is carried out using the optimal construct for each fracture.

Step 3: Pick the optimal definitive fixation.

- **Femoral shaft fractures** - the definitive treatment of choice is closed **intramedullary nailing**; it gives load-sharing, early mobilisation and the highest union rates.

- **Tibial fractures** - in this multiply-injured patient, **external fixation** is the safe definitive/staged choice given the prior systemic insult and potential soft-tissue compromise.

Therefore: nail the femurs, externally fix the tibiae.

Step 4: Eliminate the distractors.

- Option 2 reverses the logic - femurs should be nailed, not externally fixed.

- Option 3 (fastest method) describes the damage-control approach appropriate to an unstable patient, not a stabilized one.

- Option 4 (ex-fix all bones) is only a temporary measure, not definitive care once the patient is stable.

Step 5: Key fact.

Definitive care of femoral shaft fractures in a stabilized polytrauma patient is IM nailing, while the tibiae are managed with external fixation.

Quick Tip: Once stable: IM nailing is definitive for femur shaft; tibiae get external fixation.

59. What is the most likely cause of **unilateral** genu varum (bow-leg deformity of one leg) in a patient with a history of a previous injury to that limb?

(Figure: AP X-ray of both lower limbs showing bowing of one leg.)

- (A) Malunion of a previous fracture
- (B) Rickets
- (C) Non-ossifying fibroma
- (D) Osteofibrous dysplasia

Correct Answer: (A) Malunion of a previous fracture

Solution:

Step 1: Focus on the two key clues - UNILATERAL deformity and PREVIOUS INJURY.

The deformity is confined to one leg and there is a history of trauma to that limb. This combination points strongly to a post-traumatic structural cause rather than a systemic or developmental one.

Step 2: Reason from the cause of unilateral bowing.

A fracture that heals in an angulated position - **malunion** - or growth-plate injury producing asymmetric growth, will result in a **localised, unilateral** angular deformity such as genu varum. The history of previous injury makes malunion the natural explanation.

Step 3: Eliminate the distractors.

• **Rickets** - a metabolic (vitamin-D deficiency) disorder that classically causes **bilateral, symmetrical** bowing of the legs; it does not fit a unilateral, trauma-related deformity.

• **Non-ossifying fibroma** - usually an incidental, asymptomatic benign

cortical lesion; it can occasionally cause pathological fracture, but it is not the typical cause of a unilateral varus deformity following injury.

• **Osteofibrous dysplasia** - a rare benign fibro-osseous lesion of the tibia in young children causing anterior/anterolateral tibial bowing, not a varus deformity linked to prior trauma.

Step 4: Key fact.

Unilateral genu varum with a history of injury is most consistent with malunion of a previous fracture (or post-traumatic growth disturbance); systemic rickets produces bilateral symmetric bowing.

Quick Tip: Unilateral + prior injury = malunion; rickets is bilateral and symmetric.



60. An athlete presents with sudden pain in the back of the ankle after a push-off, with weakness of plantarflexion. Imaging suggests Achilles tendon rupture. Which of the following is the relevant clinical finding/sign for this condition?

(Figure: Sagittal fat-saturated ankle MRI showing discontinuity of the Achilles tendon.)

- (A) Positive Thompson (Simmonds) test
- (B) Positive McMurray test
- (C) Steroid injection
- (D) Plaster cast immobilisation

Correct Answer: (A) Positive Thompson (Simmonds) test

Solution:

Step 1: Identify the condition.

The history (sudden posterior ankle pain, weak plantarflexion) and the MRI showing Achilles tendon discontinuity indicate an **Achilles tendon rupture**.

Step 2: Recall the diagnostic clinical test.

The bedside test for Achilles rupture is the **Thompson (Simmonds) test**: with

the patient prone and the foot hanging off the table, the examiner squeezes the calf muscle. Normally this produces passive plantarflexion of the foot. If the Achilles tendon is ruptured, squeezing the calf produces **no plantarflexion** - a **positive (abnormal) Thompson test**.

Step 3: Evaluate the options.

• **Positive Thompson test** - correct; it is the specific clinical sign of Achilles tendon rupture.

• **McMurray test** - this examines for a **meniscal tear of the knee**, irrelevant to the Achilles tendon.

• **Steroid injection** - not a clinical finding; moreover steroids around the Achilles are contraindicated as they promote tendon rupture.

• **Plaster cast** - a treatment option, not a clinical sign/finding.

Step 4: Key fact.

Achilles tendon rupture is clinically confirmed by a positive Thompson (calf-squeeze) test - absence of passive plantarflexion on squeezing the calf.

Quick Tip: Achilles rupture = positive Thompson (calf-squeeze) test; McMurray is for the knee meniscus.

61. A patient with HIV and a CD4 count <40 cells/mm³ presents with a ring-enhancing lesion in the brain on contrast imaging. What is the most likely diagnosis?

- (A) Lymphoma
- (B) Toxoplasma
- (C) Tuberculosis
- (D) Progressive multifocal leukoencephalopathy (PML)

Correct Answer: (B) Toxoplasma

Solution:

Step 1: Identify the clinical setting.

This is an HIV patient with a CD4 count <40 cells/mm³, i.e. profound immunosuppression (AIDS-defining range). In this group, opportunistic CNS infections become highly likely.

Step 2: Apply the imaging clue.

A contrast ring-enhancing lesion in the brain in advanced HIV most classically points to **cerebral toxoplasmosis** (reactivation of *Toxoplasma gondii*). It typically produces *multiple* ring-enhancing lesions with a predilection for the basal ganglia and grey-white junction, with surrounding oedema.

Step 3: Eliminate the other options.

- **Primary CNS lymphoma** tends to be a single, more solid/homogeneously enhancing periventricular lesion (though it can ring-enhance, it is usually solitary and shows restricted diffusion); thallium-SPECT/PET uptake helps distinguish it.
- **Tuberculoma** is possible but less characteristic at this very low CD4 count as the lead answer.
- **PML** classically causes *non-enhancing*, asymmetric white-matter demyelination (JC virus) and does *not* produce ring enhancement.

Step 4: Conclusion.

Multiple ring-enhancing lesions in an HIV patient with CD4 <40 are most likely toxoplasmosis; empirical anti-toxoplasma therapy with response on follow-up imaging is standard. **Key fact: Toxoplasmosis is the commonest cause of ring-enhancing brain lesions in advanced HIV/AIDS.**

Quick Tip: CD4 <100 plus ring enhancement in HIV → think the commonest opportunistic CNS infection first.

62. A patient presents with right iliac fossa (RIF) pain of 48 hours' duration. The ultrasound (colour-Doppler) image is shown. What is the most appropriate next step in management?

- (A) Surgery (appendicectomy)
- (B) CECT abdomen
- (C) USG-guided drainage
- (D) Conservative management with antibiotics only

Correct Answer: (A) Surgery (appendicectomy)

Solution:

Step 1: Interpret the clinical picture.

Right iliac fossa pain of 48 hours' duration with a diagnostic ultrasound points to **acute appendicitis**. The classic USG signs are a non-compressible, blind-ending tubular structure >6 mm in diameter, target/bull's-eye appearance, peri-appendiceal fluid and increased colour-Doppler flow in the wall.

Step 2: Decide on the next step.

When ultrasound confirms uncomplicated acute appendicitis, the definitive treatment is **surgery (appendicectomy)**, ideally laparoscopic. A confident sonographic diagnosis within 48 hours does not warrant delaying definitive treatment.

Step 3: Eliminate the other options.

- **CECT** is reserved for equivocal cases or where the appendix is not visualised on USG; here the USG is already diagnostic, so further imaging is unnecessary.
- **USG-guided drainage** is appropriate only for a well-formed appendicular *abscess* (a later complication), not for acute appendicitis without a drainable collection.
- **Antibiotics alone** is not the standard definitive answer in an exam setting once appendicitis is confirmed and the patient is fit for surgery.

Step 4: Conclusion.

USG-confirmed acute appendicitis presenting acutely → proceed to **surgery**. **Key fact: an inflamed, non-compressible blind-ending tubule >6 mm with hyperaemic wall on Doppler is diagnostic of acute appendicitis and mandates appendicectomy.**

Quick Tip: USG already shows an inflamed appendix — no extra imaging is needed; treat definitively.

63. According to ACR guidelines, a Group II gadolinium-based contrast agent (lowest risk of nephrogenic systemic fibrosis) can be safely administered in which of the following?

- (A) A patient with GFR <50 mL/min/1.73m²
- (B) A 30-year-old with no renal function test done
- (C) A 60-year-old with no renal function test done
- (D) A child with normal renal function

Correct Answer: (A) A patient with GFR <50 mL/min/1.73m²

Solution:

Step 1: Recall the ACR classification of gadolinium agents.

The ACR groups gadolinium-based contrast agents (GBCAs) by their risk of causing nephrogenic systemic fibrosis (NSF): **Group I** = highest NSF risk (linear agents, e.g. gadodiamide); **Group II** = very low/negligible NSF risk (macrocyclic agents, e.g. gadobutrol, gadoteridol, gadoterate); **Group III** = limited data (e.g. gadoxetate).

Step 2: Apply the safety rule for Group II.

For Group II agents the NSF risk is so low that ACR states routine eGFR screening before administration is *not* mandatory, and they may be given even to patients with significantly **reduced renal function**. Therefore a patient with a known low GFR is the scenario where a Group II agent is specifically the safe choice that this question targets.

Step 3: Evaluate the options.

- **GFR <50** — a Group II agent is considered safe here because of its negligible NSF risk; this is the intended answer as it tests the unique selling point of Group II agents (use despite renal impairment).

- A 30-year-old or 60-year-old **with no RFT** are distractors about screening rather than about the agent's intrinsic safety in renal impairment.
- A child with normal RFT is safe but does not illustrate the special Group II advantage.

Step 4: Conclusion.

Group II GBCAs carry negligible NSF risk and can be given even when GFR is low.

Key fact: Group II macrocyclic gadolinium agents do not require eGFR screening and are safe in renal impairment, including GFR <50.

Quick Tip: Group II agents = macrocyclic, negligible NSF risk → usable even when renal function is poor.



64. A 12-year-old child presents with an uncomplicated upper respiratory tract infection (URTI). What is the most appropriate next radiological investigation?

- (A) No radiological investigation
- (B) HRCT chest
- (C) CECT chest
- (D) Chest X-ray

Correct Answer: (A) No radiological investigation

Solution:

Step 1: Recognise the nature of the illness.

An uncomplicated upper respiratory tract infection in a child is a **clinical diagnosis**. It is overwhelmingly viral and self-limiting (common cold, pharyngitis, rhinosinusitis).

Step 2: Apply imaging appropriateness principles.

Routine imaging is *not* indicated for an uncomplicated URTI. Investigations are reserved for warning features such as suspected lower respiratory tract involvement (pneumonia), persistent high fever, respiratory distress or failure to

improve. In their absence, the correct answer is **no radiological investigation**, in keeping with ALARA (avoiding unnecessary radiation in children).

Step 3: Eliminate the other options.

- **Chest X-ray** would only be justified if pneumonia or lower-tract disease were suspected; a simple URTI does not warrant it.
- **HRCT** is for interstitial lung disease/bronchiectasis evaluation — far beyond a URTI and a high radiation burden.
- **CECT** adds contrast and even more radiation, entirely inappropriate here.

Step 4: Conclusion.

An uncomplicated URTI in a 12-year-old needs no imaging. **Key fact: URTI is a clinical diagnosis; radiology is unnecessary unless red-flag features of lower-tract disease appear, especially given radiation concerns in children.**

Quick Tip: Simple URTI is clinical — imaging children without red flags only adds needless radiation.



65. A Group II gadolinium-based contrast agent (per ACR/RCR guidance, lowest risk of nephrogenic systemic fibrosis) can be safely administered in which of the following patients?

- (A) A patient with GFR <50 mL/min/1.73m²
- (B) A 30-year-old with no renal function test done
- (C) A 60-year-old with no renal function test done
- (D) A patient with normal renal function tests

Correct Answer: (A) A patient with GFR <50 mL/min/1.73m²

Solution:

Step 1: Recall the gadolinium agent groups.

Gadolinium-based contrast agents (GBCAs) are stratified by nephrogenic systemic fibrosis (NSF) risk: **Group I** = highest risk (linear, e.g. gadodiamide);

Group II = very low/negligible risk (macrocyclic, e.g. gadoterate, gadobutrol, gadoteridol); **Group III** = limited data.

Step 2: Identify the unique property of Group II.

Because Group II agents have negligible NSF risk, guidance allows their use even in patients with **impaired renal function**, and routine pre-contrast eGFR screening is not mandatory for them. The scenario that specifically tests this is a patient with a documented low GFR.

Step 3: Evaluate the options.

- **GFR <50** — a Group II macrocyclic agent remains safe here owing to negligible NSF risk; this is the intended answer because it highlights safe use despite renal impairment.
- The two "**no RFT**" options concern screening, not the agent's intrinsic safety in renal disease.
- A patient with **normal RFT** can of course receive any GBCA, but this does not demonstrate the special advantage of Group II.

Step 4: Conclusion.

Group II GBCAs may be given even when GFR is reduced. **Key fact: macrocyclic (Group II) gadolinium agents carry negligible NSF risk and are safe in renal impairment, including GFR <50, without mandatory eGFR screening.**

Quick Tip: The whole point of Group II macrocyclic agents — negligible NSF risk, so usable even with low GFR.

66. A 12-year-old child presents with an uncomplicated upper respiratory tract infection (URTI). What should be the next step regarding radiological imaging?

- (A) No radiological investigation
- (B) HRCT chest
- (C) CECT chest
- (D) Chest X-ray

Correct Answer: (A) No radiological investigation

Solution:

Step 1: Characterise the presentation.

A 12-year-old with an uncomplicated URTI has a self-limiting, predominantly viral illness diagnosed clinically. There is no indication of lower respiratory tract disease.

Step 2: Apply imaging appropriateness and radiation safety.

Guidelines do not recommend any radiological investigation for an uncomplicated URTI. Children are particularly radiosensitive, so the ALARA principle reinforces avoiding unnecessary imaging. Hence the next step is **no radiological investigation**.

Step 3: Eliminate the other options.

- **Chest X-ray** is appropriate only if pneumonia or lower-tract involvement is suspected — not for a simple URTI.
- **HRCT** evaluates interstitial lung disease/bronchiectasis and delivers high radiation; it is irrelevant here.
- **CECT** adds contrast and still more radiation, and is clearly inappropriate.

Step 4: Conclusion.

No imaging is warranted for an uncomplicated paediatric URTI. **Key fact: URTI is a clinical diagnosis; image only if red-flag features of lower respiratory tract disease develop, sparing the child unnecessary radiation.**

Quick Tip: Uncomplicated URTI = clinical call; no red flags means no scan and no radiation.

67. A 70-year-old man with a past history of pulmonary tuberculosis, currently on anti-tubercular therapy (ATT), presents with back pain. An MRI of the spine is performed and shows a vertebral lesion. What is the next investigation of choice to reach a definitive diagnosis?

- (A) Mantoux test
- (B) Biopsy
- (C) Serum protein electrophoresis
- (D) CT chest

Correct Answer: (B) Biopsy

Solution:

Step 1: Frame the clinical problem.

A 70-year-old patient with a known history of pulmonary TB (already on ATT) now has back pain and an MRI-detected vertebral lesion. The key teaching point is that an MRI can suggest, but cannot definitively distinguish, the cause of a destructive vertebral lesion in an elderly patient.

Step 2: List the differentials.

In an elderly patient, a destructive spinal lesion can be tuberculous spondylitis (Pott's spine), metastasis, multiple myeloma, or primary bone tumour. Despite the TB history, malignancy must be actively excluded because TB and malignancy can coexist and have overlapping imaging features.

Step 3: Decide the next step.

Imaging (MRI/CT) characterises the lesion but is not diagnostic. To establish tissue diagnosis - to differentiate TB from malignancy and to obtain material for culture, drug-sensitivity and histopathology - a **biopsy** (CT-guided or open) is the definitive next investigation. It both confirms the aetiology and guides therapy.

Step 4: Why the other options are wrong.

Mantoux only indicates prior TB exposure; it is already positive in most adults from an endemic area and cannot confirm spinal TB versus malignancy. Serum protein electrophoresis screens for myeloma but is non-specific and would not characterise a focal vertebral lesion definitively. CT chest evaluates the lungs, not the painful vertebra.

Key fact: A destructive vertebral lesion needs tissue - biopsy is the definitive next step to differentiate TB from malignancy.

Quick Tip: Imaging suggests; tissue confirms - the painful vertebral lesion needs a tissue diagnosis.

68. Match the clinical sign with the correct dermatological condition and select the correctly matched pair(s):

- (i) Positive Nikolsky sign - Pemphigus vulgaris
 - (ii) Negative/absent Nikolsky sign - Bullous pemphigoid
 - (iii) Pseudo-Nikolsky sign - Stevens-Johnson syndrome / Toxic epidermal necrolysis
 - (iv) Cerebriform tongue - Pemphigus vegetans
- (A) Positive Nikolsky - Pemphigus vulgaris
 - (B) Negative/absent Nikolsky - Bullous pemphigoid
 - (C) Pseudo-Nikolsky - Stevens-Johnson syndrome
 - (D) Cerebriform tongue - Pemphigus vegetans

Correct Answer: (D) Cerebriform tongue - Pemphigus vegetans

Solution:

Step 1: Understand the Nikolsky sign.

Nikolsky sign is dislodgement of the superficial epidermis on tangential pressure, indicating loss of epidermal cohesion (acantholysis). It is positive in pemphigus group disorders.

Step 2: Evaluate each pair.

- (i) Pemphigus vulgaris has intra-epidermal (suprabasal) acantholysis to **positive Nikolsky**. Correct.
- (ii) Bullous pemphigoid has a sub-epidermal split (anti-BP180/BP230, hemidesmosomes) with intact epidermal cohesion to **Nikolsky negative**. Correct.
- (iii) In SJS/TEN the epidermal-dermal separation gives a **pseudo-Nikolsky**

(Asboe-Hansen) appearance - the sign is positive only over already-affected erythematous skin, not normal skin. Correct as a pseudo sign.

(iv) Pemphigus vegetans classically shows a **cerebriform (brain-like) tongue** with sulci and gyri. Correct.

Step 3: Identify the intended answer.

This is a match-the-following item and all four pairs are individually correct associations. The examiner's key emphasises the highlighted/most classic association: **Cerebriform tongue - Pemphigus vegetans**, which is the distinctive, frequently tested pair. The answer marked is option 4.

Key fact: Cerebriform (brain-like) tongue is the hallmark of pemphigus vegetans; Nikolsky is positive in pemphigus, negative in bullous pemphigoid, and pseudo-positive in SJS/TEN.

Quick Tip: Brain-like (cerebriform) tongue is the signature of pemphigus vegetans.

69. A patient presents with a black pigmented lesion on the heel/sole (as shown). Which is the investigation of choice to establish the diagnosis?

- (A) Biopsy
- (B) KOH mount
- (C) Gram stain
- (D) Tzanck smear

Correct Answer: (A) Biopsy

Solution:

Step 1: Recognise the lesion.

A black/dark pigmented lesion on an acral site such as the heel or sole is highly suspicious of **acral lentiginous melanoma**, the commonest melanoma subtype in dark-skinned and Asian populations. Any new, irregular, pigmented acral lesion must be assumed malignant until proven otherwise.

Step 2: Choose the investigation.

The definitive diagnosis of melanoma is **histopathology**. An excisional biopsy (ideally full-thickness, removing the entire lesion with a narrow margin) is preferred because it allows accurate measurement of Breslow thickness, which determines prognosis and further management.

Step 3: Why the other options are wrong.

KOH mount detects fungal elements (e.g., tinea nigra), but a frankly suspicious black acral lesion in this setting demands tissue diagnosis, not just a fungal screen. Gram stain identifies bacteria and is irrelevant to a pigmented neoplastic lesion. Tzanck smear detects acantholytic/multinucleate giant cells in vesiculobullous and herpetic lesions, not pigmented tumours.

Key fact: A suspicious pigmented acral lesion (possible acral lentiginous melanoma) requires biopsy with histopathology and Breslow thickness - biopsy is the investigation of choice.

Quick Tip: A suspicious black acral lesion is melanoma until biopsy proves otherwise.

70. A patient presents with painful grouped vesicles over the penile/genital region associated with fever and malaise. What is the most likely causative organism?

- (A) Herpes simplex virus (HSV)
- (B) Haemophilus ducreyi
- (C) Human papillomavirus (HPV)
- (D) Treponema pallidum (Syphilis)

Correct Answer: (A) Herpes simplex virus (HSV)

Solution:

Step 1: Identify the lesion morphology.

The combination of **painful, grouped vesicles** on an erythematous base over the

genitalia, with systemic features (fever, malaise), is the classic picture of **primary genital herpes** caused by herpes simplex virus (usually HSV-2).

Step 2: Match to the organism.

Primary HSV infection characteristically produces multiple painful vesicles that rupture into shallow ulcers, accompanied by tender inguinal lymphadenopathy and constitutional symptoms - exactly as described.

Step 3: Why the other options are wrong.

Haemophilus ducreyi causes chancroid - painful, ragged, undermined *ulcers*, not vesicles, and usually without prominent systemic vesicular eruption. HPV causes painless warts (condylomata acuminata), not painful vesicles. *Treponema pallidum* (syphilis) causes a *painless*, indurated chancre with painless adenopathy - the opposite of this painful vesicular presentation.

Key fact: Painful grouped genital vesicles with fever and malaise = primary genital herpes (HSV).

Quick Tip: Painful grouped genital vesicles + systemic symptoms = herpes.

71. The lesion shown is consistent with tinea capitis. What is the treatment of choice?

- (A) Oral antifungals
- (B) Antibiotics
- (C) Methotrexate
- (D) Topical steroids

Correct Answer: (A) Oral antifungals

Solution:

Step 1: Define the condition.

Tinea capitis is a **dermatophyte infection of the scalp and hair shafts**. Because the fungus invades the hair follicle and shaft, topical agents cannot penetrate

adequately.

Step 2: Choose the treatment.

The treatment of choice is **systemic (oral) antifungals** - classically griseofulvin, and increasingly terbinafine or itraconazole. Oral therapy reaches the hair follicle and eradicates the infection, which topical antifungals alone cannot achieve.

Step 3: Why the other options are wrong.

Antibiotics treat bacterial infection and have no role unless there is secondary bacterial superinfection (e.g., kerion). Methotrexate is an immunosuppressant for inflammatory/proliferative disorders such as psoriasis - it would worsen a fungal infection. Topical steroids suppress inflammation but promote fungal spread (tinea incognito) and never cure tinea capitis.

Key fact: Tinea capitis requires oral antifungals because topical drugs cannot penetrate the infected hair shaft.

Quick Tip: Hair-shaft fungal infection needs systemic, not topical, antifungal.



72. Which of the following are advantages/benefits of non-treponemal tests (e.g., VDRL, RPR) in the diagnosis and follow-up of syphilis? (Select all that apply)

- (A) They can be used to monitor response to treatment
- (B) A standard dilution is used
- (C) They remain positive for life
- (D) They are quantitative

Correct Answer: (A) They can be used to monitor response to treatment

Solution:

Step 1: Recall non-treponemal tests.

Non-treponemal tests (VDRL, RPR) detect antibodies against cardiolipin-lecithin-cholesterol antigen. Their titres rise and fall with disease activity, unlike

treponemal tests which usually stay positive for life.

Step 2: Evaluate each statement.

(A) **Response to treatment** - CORRECT. A four-fold (two-dilution) drop in titre indicates successful therapy, and a four-fold rise indicates relapse/re-infection.

This monitoring ability is the principal advantage.

(B) Standard dilution - not an advantage; non-treponemal tests are reported as serial dilutions/titres (1:2, 1:4, 1:8 …), not a single standard dilution.

(C) Lifetime positivity - this is a feature of *treponemal* tests, not non-treponemal ones; non-treponemal titres decline/revert after treatment, so this is not their benefit.

(D) **Quantitative** - CORRECT. Because results are expressed as titres, the test is quantitative, which is precisely what enables treatment monitoring.

Step 3: Conclusion.

The benefits are that the tests are **quantitative** and therefore allow **monitoring of response to treatment** (options A and D). The primary single best answer is response to treatment, which directly derives from the quantitative titre.

Key fact: Non-treponemal tests are quantitative (titre-based), letting you monitor treatment response; treponemal tests, by contrast, stay positive for life.

Quick Tip: Titre-based = quantitative, so they track treatment response.

73. A divorced, alcoholic man with major depression presents stating, "What is the point in living?" What is the most appropriate next step in management?

- (A) Hospitalize
- (B) Cognitive behavioural therapy (CBT)
- (C) Antidepressant
- (D) Reassure that it is a normal part of life

Correct Answer: (A) Hospitalize

Solution:

Step 1: Identify the clinical priority. This patient has major depression plus several high-risk suicide factors - male sex, divorced (lack of social support), alcohol use, and active passive suicidal ideation ("what is the point in living"). Safety always comes first in psychiatric emergencies.

Step 2: Assess suicide risk. The mnemonic SAD PERSONS (Sex-male, Age, Depression, Previous attempt, Ethanol/alcohol, Rational thinking loss, Social support lacking, Organised plan, No spouse, Sickness) flags multiple risk factors here. A patient verbalising suicidal ideation with depression, alcohol dependence and no social support is at imminent risk.

Step 3: Choose management. The immediate priority for an actively suicidal high-risk patient is to ensure safety, which means inpatient hospitalisation for observation, stabilisation and initiation of treatment.

Step 4: Why the others are wrong. CBT and antidepressants are essential parts of definitive treatment but take days to weeks to act and do not address immediate risk - they are started after the patient is in a safe setting. Calling it a normal part of life is dismissive and dangerous. **Hence, hospitalise the patient first.**

Quick Tip: Active suicidal ideation + multiple risk factors = ensure safety first.

74. Which of the following statements best describes classical conditioning?

- (A) Unconditioned stimulus producing an unconditioned response
- (B) Conditioned stimulus producing a conditioned response
- (C) Association between a conditioned stimulus and a conditioned response
- (D) Association between an unconditioned stimulus and an unconditioned response

Correct Answer: (B) Conditioned stimulus producing a conditioned response

Solution:

Step 1: Define classical conditioning. Classical (Pavlovian) conditioning is a form of associative learning in which a previously neutral stimulus, after repeated pairing with an unconditioned stimulus (UCS), comes to elicit a response on its own. That neutral stimulus then becomes the conditioned stimulus (CS), and the response it now evokes is the conditioned response (CR).

Step 2: Apply Pavlov's experiment. Food (UCS) naturally produces salivation (unconditioned response, UCR). A bell (neutral) repeatedly paired with food becomes the CS; eventually the bell alone produces salivation, now called the conditioned response (CR).

Step 3: Select the best descriptor. The hallmark of established classical conditioning is that the conditioned stimulus now produces a conditioned response. Option 2 captures this learned end-point precisely.

Step 4: Why the others are wrong. A UCS producing a UCR (option 1) is the innate reflex, not learning. Options 3 and 4 misstate the associations - conditioning forms an association between a CS and a UCS (stimulus-stimulus), not CS-CR or UCS-UCR. **Therefore, the conditioned stimulus producing a conditioned response best describes classical conditioning.**

Quick Tip: Think of Pavlov's bell alone making the dog salivate.

75. The clinical photograph shows a scalp lesion consistent with tinea capitis. What is the treatment of choice?

- (A) Oral antifungals
- (B) Antibiotics
- (C) Methotrexate
- (D) Topical steroids

Correct Answer: (A) Oral antifungals

Solution:

Step 1: Identify the condition. Tinea capitis is a dermatophyte infection of the scalp and hair shafts, commonly caused by *Trichophyton* and *Microsporum* species. It presents with scaly patches, broken hairs, alopecia and sometimes a boggy inflammatory mass (kerion).

Step 2: Understand why topical therapy fails. The fungus invades the hair shaft (endothrix/ectothrix). Topical agents cannot penetrate the hair follicle adequately, so they alone cannot eradicate the infection.

Step 3: Choose treatment. Systemic (oral) antifungals are mandatory. Griseofulvin and oral terbinafine are first-line; itraconazole and fluconazole are alternatives. A topical antifungal shampoo (e.g. selenium sulfide, ketoconazole) may be added to reduce spore shedding, but it is adjunctive.

Step 4: Why the others are wrong. Antibiotics treat bacterial infection, not fungi (used only for secondary bacterial superinfection). Methotrexate is an immunosuppressant with no antifungal role and would worsen infection. Topical steroids suppress local immunity and can cause tinea incognito. **Hence, oral antifungals are the treatment of choice.**

Quick Tip: Fungus inside the hair shaft cannot be reached by topical drugs.

76. Match the poison with its characteristic feature. Which of the following is correctly matched?

- (A) Cyanide - bitter almond smell
- (B) Carbon monoxide - cherry red discolouration
- (C) Phosphorus - garlic odour
- (D) Organophosphate - excessive secretions

Correct Answer: (A) Cyanide - bitter almond smell

Solution:

Step 1: Understand the question. Each option pairs a poison with a classic forensic/toxicological sign. All four pairings listed are, in fact, individually correct, but the conventionally "circled"/single-best answer in this recall is the cyanide pairing, which is the most iconic and frequently tested association.

Step 2: Verify cyanide. Cyanide poisoning classically produces a bitter almond odour on the breath/at autopsy (detectable by roughly 40-60% of people genetically). It also causes histotoxic hypoxia with bright red venous blood.

Step 3: Verify the others (all true). Carbon monoxide binds haemoglobin to form carboxyhaemoglobin, giving cherry-red skin/lividity. Phosphorus poisoning (and arsenic, thallium) classically gives a garlicky odour; phosphorus also causes "smoking stool" and luminescence. Organophosphate poisoning produces a cholinergic crisis with marked secretions (SLUDGE/DUMBELS).

Step 4: Select the answer. As the single most characteristic and exam-favoured match, **Cyanide - bitter almond smell** is the correct/circled answer.

Quick Tip: The most iconic odour in toxicology - bitter almonds.

77. Which of the following is a type of blunt-force mechanical injury produced without a break in the continuity of the skin?

- (A) Stab wound of the abdomen
- (B) Laceration
- (C) Bomb blast injury
- (D) Contusion

Correct Answer: (D) Contusion

Solution:

Step 1: Classify mechanical injuries. Mechanical injuries are broadly abrasions, contusions (bruises), lacerations, incised/stab (sharp-force) wounds, and firearm/blast injuries. The question asks for a blunt-force injury where the skin surface remains intact (a closed injury).

Step 2: Define contusion. A contusion (bruise) is an extravasation of blood into the tissues due to rupture of small vessels following blunt impact, with the overlying skin remaining unbroken. It is a closed blunt-force injury.

Step 3: Why the others are wrong. A stab wound is a sharp-force penetrating injury that breaks the skin. A laceration is a blunt-force tear of the skin/tissues - it does break the continuity of the skin (so it is open). A bomb blast produces complex injuries (blast, penetrating, burns) with skin disruption. **Therefore, contusion is the blunt-force injury without a breach of the skin.**

Quick Tip: A closed bruise from blunt trauma with intact overlying skin.

78. A 16-year-old girl raped by a neighbour presents at 16 weeks of gestation. Which of the following is NOT a part of her management?

- (A) Inform the police
- (B) Preserve fetal tissue for DNA analysis
- (C) Take consent from the parents/guardian
- (D) MTP can be done on the opinion of a single doctor

Correct Answer: (D) MTP can be done on the opinion of a single doctor

Solution:

Step 1: Frame the medico-legal scenario. This is a survivor of sexual assault who is a minor (16 years) and pregnant at 16 weeks, presumably seeking termination. Several statutory obligations apply.

Step 2: Apply the MTP Act (as amended 2021). For a pregnancy beyond 12

weeks and up to 20 weeks, termination requires the opinion of **two** registered medical practitioners, not one. Only up to 12 weeks does a single doctor's opinion suffice. At 16 weeks, two doctors' opinions are mandatory - so "MTP on one doctor's opinion" is the incorrect/NOT-included step.

Step 3: Validate the other steps. Under the POCSO Act, sexual assault of a minor is a mandatory reportable offence, so the police must be informed. The aborted fetal/products of conception should be preserved for DNA profiling as forensic evidence. As the patient is a minor, consent of the parent/guardian is required for the MTP procedure (the minor's own assent plus guardian consent).

Step 4: Select the answer. Since at 16 weeks two practitioners' opinions are needed, performing MTP on a single doctor's opinion is NOT part of correct management. **Hence the answer is "MTP on one doctor's opinion."**

Quick Tip: Beyond 12 weeks the MTP Act needs two doctors' opinions, not one.



79. In organophosphate (OP) poisoning, which drug reverses the inhibited activity of acetylcholinesterase (AChE)?

- (A) Atropine
- (B) Pralidoxime
- (C) Flumazenil
- (D) Naloxone

Correct Answer: (B) Pralidoxime

Solution:

Step 1: Understand OP toxicity. Organophosphates inhibit acetylcholinesterase (AChE) by phosphorylating its active (esteratic) site. This stops breakdown of acetylcholine, causing accumulation at muscarinic, nicotinic and CNS synapses (SLUDGE/DUMBELS features).

Step 2: Identify the drug that reactivates the enzyme. Pralidoxime (2-PAM) is an oxime that binds the phosphorus atom on the inhibited enzyme and cleaves the OP-enzyme bond, thereby **reactivating AChE** - especially restoring neuromuscular (nicotinic) function. It must be given early, before "aging" (irreversible dealkylation) of the enzyme occurs.

Step 3: Why the others are wrong. Atropine is the physiological antidote that blocks muscarinic receptors to counter secretions/bradycardia, but it does *not* reactivate AChE. Flumazenil reverses benzodiazepines (GABA-A). Naloxone reverses opioids (mu receptor).

Key fact: Only Pralidoxime reactivates the phosphorylated acetylcholinesterase enzyme in OP poisoning.

Quick Tip: Think of the oxime that cleaves the OP-enzyme bond, not the muscarinic blocker.



80. When should a prophylactic antibiotic be administered before elective entry into the GI tract (e.g., elective cholecystectomy)?

- (A) Within 1 hour before incision
- (B) 4 hours before incision
- (C) 6 hours before incision
- (D) At the time of skin closure

Correct Answer: (A) Within 1 hour before incision

Solution:

Step 1: Principle of surgical antibiotic prophylaxis. The aim is to have an adequate **tissue and serum drug concentration at the moment of incision** and throughout the period of contamination.

Step 2: Recommended timing. The prophylactic antibiotic should be given

within 60 minutes (30-60 min) before skin incision. For vancomycin or fluoroquinolones, infusion may begin up to 120 minutes before because of slower administration.

Step 3: Why the others are wrong. Giving the drug 4 or 6 hours earlier means levels fall before contamination occurs, defeating the purpose. Administering it only at closure is too late, as bacterial seeding has already happened during the procedure.

Key fact: Surgical antibiotic prophylaxis is best given within 30-60 minutes before incision, i.e. within 1 hour.

Quick Tip: Peak drug level must coincide with the moment of incision.



81. A neonate presents with prolonged jaundice and an umbilical hernia. The most likely diagnosis is:

- (A) Down syndrome
- (B) Congenital hypothyroidism
- (C) Cerebral palsy
- (D) Biliary atresia

Correct Answer: (B) Congenital hypothyroidism

Solution:

Step 1: Interpret the clinical clues. *Prolonged jaundice* (unconjugated, persisting beyond 2-3 weeks) plus an *umbilical hernia* in a neonate is the classic combination pointing to **congenital hypothyroidism**.

Step 2: Mechanism. Thyroid hormone deficiency slows hepatic glucuronyl transferase activity and bowel transit, prolonging unconjugated hyperbilirubinaemia. Reduced muscle tone and connective-tissue laxity of the abdominal wall produce the umbilical hernia. Other features include lethargy,

poor feeding, constipation, large fontanelle, macroglossia, hypothermia and hoarse cry.

Step 3: Why the others are wrong. Down syndrome can have an umbilical hernia and even coexisting hypothyroidism, but the specific dyad of prolonged jaundice + umbilical hernia classically denotes hypothyroidism. Cerebral palsy is a motor disorder and does not cause jaundice. Biliary atresia gives *conjugated* jaundice with pale stools, not this picture.

Key fact: Prolonged neonatal jaundice with umbilical hernia = congenital hypothyroidism; screen with TSH/T4.

Quick Tip: Prolonged unconjugated jaundice plus umbilical hernia and constipation in a sluggish neonate.



82. Differential cyanosis (pink upper limbs with cyanosed lower limbs) is characteristically seen in:

- (A) Patent ductus arteriosus (PDA) with reversal of shunt / Eisenmenger
- (B) Osler-Weber-Rendu syndrome
- (C) Peripheral arterial disease
- (D) Transposition of great arteries (simple)

Correct Answer: (A) Patent ductus arteriosus (PDA) with reversal of shunt / Eisenmenger

Solution:

Step 1: Define differential cyanosis. Differential cyanosis means the **lower limbs are cyanosed (and clubbed) while the upper limbs / head remain pink**. It indicates that deoxygenated blood is reaching the descending aorta while the upper body still receives oxygenated blood.

Step 2: Identify the lesion. This occurs in a **large PDA with pulmonary**

hypertension and reversal of the shunt (Eisenmenger physiology).

Desaturated blood from the pulmonary artery crosses the duct into the descending aorta (distal to the left subclavian), supplying the lower body, while the head and right arm get pre-ductal oxygenated blood.

Step 3: Why the others are wrong. Osler-Weber-Rendu (hereditary haemorrhagic telangiectasia) causes mucocutaneous telangiectasias and AVMs, not a regional cyanosis gradient. Peripheral arterial disease gives limb ischaemia/acrocyanosis, not the classic upper-pink/lower-blue pattern of a cardiac shunt. Simple TGA causes *reversed* differential cyanosis only in special combinations.

Key fact: Differential cyanosis (pink arms, blue legs) = PDA with right-to-left shunt / Eisenmenger syndrome.

Quick Tip: Blue legs, pink arms means desaturated blood enters the aorta distal to the subclavian via a duct.



83. In the management of shoulder dystocia, after calling for help the first/next manoeuvre to be performed is:

- (A) McRoberts manoeuvre (with suprapubic pressure)
- (B) Zavanelli manoeuvre
- (C) Wood's corkscrew manoeuvre
- (D) Symphysiotomy

Correct Answer: (A) McRoberts manoeuvre (with suprapubic pressure)

Solution:

Step 1: Recognise the emergency. Shoulder dystocia is failure of the anterior shoulder to deliver after the head, due to impaction behind the pubic symphysis. The HELPER drill is followed.

Step 2: First-line manoeuvre. The initial step (after calling for help) is the

McRoberts manoeuvre - hyperflexion and abduction of the maternal hips onto the abdomen. This straightens the lumbosacral angle and rotates the symphysis cephalad, freeing the anterior shoulder. It is combined with **suprapubic pressure** to dislodge the shoulder. McRoberts alone or with suprapubic pressure resolves the majority of cases.

Step 3: Why the others are wrong. Wood's corkscrew and delivery of the posterior arm are second-line internal rotational manoeuvres used if McRoberts fails. The Zavanelli manoeuvre (cephalic replacement followed by caesarean) and symphysiotomy are last-resort procedures for intractable dystocia.

Key fact: McRoberts manoeuvre with suprapubic pressure is the first step in managing shoulder dystocia.

Quick Tip: Hyperflex the maternal hips first; it is simple, non-invasive and resolves most cases.



84. The Pearl Index, used to express contraceptive failure rate, is defined as:

- (A) Number of accidental pregnancies per woman year
- (B) Number of accidental pregnancies per 100 woman-years of exposure
- (C) Number of pregnancies over number of losses per 100 woman-years
- (D) Number of accidental pregnancies per 1000 women per year

Correct Answer: (B) Number of accidental pregnancies per 100 woman-years of exposure

Solution:

Step 1: Purpose of the Pearl Index. The Pearl Index is the standard measure of the **effectiveness (failure rate) of a contraceptive method**.

Step 2: Definition and formula. It is the **number of accidental pregnancies per 100 woman-years of exposure (HWY):**

$$\text{Pearl Index} = \frac{\text{Total accidental pregnancies} \times 1200}{\text{Total months of exposure}}$$

The factor 1200 converts to 100 woman-years (100 women x 12 months). A lower Pearl Index means a more effective method.

Step 3: Why the others are wrong. "Per woman year" omits the standard denominator of 100 woman-years. The option dividing pregnancies by losses is not the definition. "Per 1000 women" uses the wrong denominator.

Key fact: Pearl Index = accidental pregnancies per 100 woman-years of exposure; lower value = better contraceptive.

Quick Tip: Denominator is 100 woman-years of exposure, and lower is better.

85. If X percent of stroke in a population is attributable to hypertension, and removing hypertension from the population would prevent this proportion of strokes, this scenario is an example of:

- (A) Relative risk
- (B) Attributable risk
- (C) Population attributable risk
- (D) Odds ratio

Correct Answer: (C) Population attributable risk

Solution:

Step 1: Identify the measure of association in question.

The question asks about the proportion (percent) of disease (stroke) in the *whole population* that is due to an exposure (hypertension) and could be removed if the exposure were eliminated. This is the definition of **Population Attributable Risk (PAR)** / Population Attributable Fraction.

Step 2: Define the terms.

• **Relative risk (RR)** = incidence in exposed / incidence in unexposed. It measures the *strength* of association, not a population burden.

• **Attributable risk (AR)** = incidence in exposed − incidence in unexposed. It is the excess risk in the *exposed group only*.

• **Population attributable risk (PAR)** = incidence in total population − incidence in unexposed. The *Population Attributable Fraction* is the proportion of disease in the *whole population* attributable to the exposure, i.e. the percent that would be prevented by removing the exposure.

Step 3: Map to the scenario.

X% of all strokes in the population being preventable by removing hypertension is precisely the population attributable fraction, derived from PAR. Hence the answer is **Population attributable risk**.

Step 4: Why the others are wrong.

RR and odds ratio are ratios that quantify association strength, not a removable population burden. AR applies only to the exposed subgroup, not the entire population.

Key fact: PAR (and the population attributable fraction) is used by public-health planners to estimate the impact of removing a risk factor from the entire population.

Quick Tip: Burden in the whole population removable by eliminating the exposure to think population-level attributable measure.

86. In a meta-analysis, assessing heterogeneity (e.g. using the I^2 statistic or Cochran's Q test):

- (A) Evaluates the variation between the included studies
- (B) Detects publication bias directly
- (C) Measures confounding within a single study
- (D) Quantifies the precision of an individual study

Correct Answer: (A) Evaluates the variation between the included studies

Solution:

Step 1: Understand what heterogeneity means in a meta-analysis.

A meta-analysis statistically pools the results of several independent studies.

Heterogeneity refers to the variability or differences in effect estimates *between* the included studies, beyond what is expected by chance alone.

Step 2: Tools used to assess it.

- **Cochran's Q test** - tests whether observed differences in results are due to chance.

- **I^2 statistic** - quantifies the percentage of total variation across studies due to heterogeneity rather than chance (25% low, 50% moderate, 75% high).

Step 3: Map to the options.

Heterogeneity directly **evaluates the variation between the included studies** - option 1 is correct.

Step 4: Why the others are wrong.

- Publication bias is assessed separately, by a *funnel plot* or Egger's test, not by heterogeneity statistics.

- Confounding is a within-study validity issue, addressed at the individual-study level, not by between-study heterogeneity.

- The precision of an individual study is shown by its confidence interval / weight, not by heterogeneity.

Key fact: Heterogeneity (I^2 , Q) measures between-study variation; a high value favours a random-effects model.

Quick Tip: Heterogeneity is about how much the studies differ from one another, not about bias.

87. Which of the following statements about ticagrelor is TRUE?

- (A) It is a reversible P2Y₁₂ receptor inhibitor
- (B) It is a prodrug requiring hepatic activation
- (C) It causes more cerebral bleeding than clopidogrel
- (D) It is given once daily

Correct Answer: (A) It is a reversible P2Y₁₂ receptor inhibitor

Solution:

Step 1: Classify ticagrelor.

Ticagrelor is an antiplatelet agent of the **cyclopentyltriazolopyrimidine (CPTP)** class. It blocks the platelet **P2Y₁₂** ADP receptor, reducing ADP-mediated platelet aggregation.

Step 2: Key pharmacological feature.

Unlike the thienopyridines (clopidogrel, prasugrel) which bind the P2Y₁₂ receptor *irreversibly*, ticagrelor binds **reversibly**. Hence option 1 is correct.

Step 3: Why the other statements are false.

• **Prodrug** - FALSE. Ticagrelor is directly active and does NOT require hepatic CYP activation (its active metabolite also contributes, but the parent drug is itself active). Clopidogrel and prasugrel are the prodrugs.

• **More cerebral bleed than clopidogrel** - In the PLATO trial, total major bleeding was similar; ticagrelor showed slightly more non-CABG/spontaneous bleeding but it is not characterised as causing distinctly more intracranial bleeding as a defining true feature. The unequivocally correct statement is reversibility.

• **Once daily** - FALSE. Because binding is reversible and its half-life is short, ticagrelor is given **twice daily**.

Key fact: Ticagrelor = directly-acting, **reversible** P2Y₁₂ inhibitor, dosed twice daily; it also causes dyspnea (adenosine effect) and bradyarrhythmias.

Quick Tip: The feature that separates ticagrelor from clopidogrel/prasugrel is reversible, direct binding.



88. From the quantal dose-response curves shown, the median toxic dose TD_{50} is 400 mg and the median effective dose ED_{50} is 100 mg. Calculate the therapeutic index. (Refer to the figure.)

- (A) 4
- (B) 0.25
- (C) 1
- (D) 500

Correct Answer: (A) 4

Solution:

Step 1: Recall the definition of therapeutic index (TI).

The therapeutic index is a measure of drug safety, defined from quantal dose-response curves as:

$$TI = \frac{TD_{50} \text{ (or } LD_{50})}{ED_{50}}$$

where TD_{50} is the dose toxic in 50% of the population and ED_{50} the dose effective in 50%.

Step 2: Read the values from the curves.

From the figure: $ED_{50} = 100$ mg (efficacy curve) and $TD_{50} = 400$ mg (toxicity curve).

Step 3: Substitute.

$$TI = \frac{400}{100} = 4$$

Step 4: Interpret and eliminate.

A larger TI means a wider (safer) margin between effective and toxic doses. A value of 4 is the answer.

• 0.25 is the inverted ratio (ED_{50}/TD_{50}) - wrong direction.

• 1 would imply effective and toxic doses are identical - not the case here.

• 500 is the simple difference of the doses, not a ratio.

Key fact: $TI = TD_{50}/ED_{50}$; the higher the value, the safer the drug.

Quick Tip: Therapeutic index = toxic dose for 50% divided by effective dose for 50%.

89. The graph shows the relative filterability of molecules across the glomerular capillary wall plotted against effective molecular radius, for three curves A, B and C carrying different charges. Based on charge, which molecule is filtered most readily? (Refer to the figure; for a given molecular radius, filterability order $A > B > C$, with C marked as anions.)

- (A) A (cation)
- (B) B (neutral)
- (C) C (anion)
- (D) All are filtered equally

Correct Answer: (A) A (cation)

Solution:

Step 1: Recall the glomerular filtration barrier and charge selectivity.

The glomerular capillary wall (endothelium, basement membrane, podocyte slit diaphragm) carries fixed **negative charges**, largely due to heparan sulfate proteoglycans and the sialoprotein coat (e.g. podocalyxin).

Step 2: Apply electrostatics.

Because the barrier is negatively charged, it *repels* negatively charged (anionic) molecules and *attracts/permits* positively charged (cationic) molecules.

Therefore, for the **same molecular size**:

Filterability: Cation > Neutral > Anion

Step 3: Map to the curves.

Curve A (highest filterability) = **cation**; curve B = neutral; curve C (lowest, labelled anions) = anion. So the most filterable molecule based on charge is **A, the cation**.

Step 4: Why others are wrong.

• B (neutral) is filtered less than the cation.

• C (anion) is filtered least because of charge repulsion - this is the basis of selective albuminuria when the negative charge barrier is lost (e.g. minimal change disease).

• They are not filtered equally - charge clearly discriminates.

Key fact: The negatively charged glomerular barrier favours filtration of cations over neutral molecules over anions; albumin (anionic) is normally restricted partly by charge.

Quick Tip: The filtration barrier is negatively charged, so it lets positive molecules through most easily.

90. Which intestinal membrane transporter is exploited in the therapeutic management (oral rehydration) of secretory diarrhea?

- (A) CFTR chloride channel
- (B) SGLT (sodium-glucose cotransporter)
- (C) Na^+/K^+ ATPase pump
- (D) Aquaporin-2 water channel

Correct Answer: (B) SGLT (sodium-glucose cotransporter)

Solution:

Step 1: Understand the basis of oral rehydration therapy (ORT).

In secretory diarrhea (e.g. cholera), the toxin activates the CFTR chloride channel, driving massive secretion of Cl^- , Na^+ and water into the gut lumen. CFTR is the *culprit*, not the therapeutic target.

Step 2: Identify the channel used for treatment.

The cornerstone of treatment is **oral rehydration solution (ORS)**, which works through the **sodium-glucose cotransporter, SGLT1**, in the brush border of enterocytes. SGLT1 absorbs Na^+ coupled to glucose, and water follows osmotically.

Step 3: Why SGLT is the answer.

Crucially, SGLT1-mediated Na^+ -glucose absorption remains *intact* even when cholera toxin is active. By supplying glucose and sodium together, ORS harnesses this surviving pathway to reabsorb Na^+ and water, reversing dehydration. This coupling is the physiological rationale for adding glucose to ORS.

Step 4: Why the others are wrong.

• **CFTR** - mediates the secretion causing the diarrhea; we do not use it to treat.

• **Na^+/K^+ ATPase** - a basolateral pump that maintains the Na^+ gradient, but it is not the directly exploited luminal absorptive route in ORS.

• **Aquaporin-2** - an ADH-regulated water channel in the renal collecting

duct, irrelevant to intestinal ORT.

Key fact: ORS works by SGLT1-mediated Na^+ -glucose coupled absorption, which stays functional in secretory diarrhea.

Quick Tip: Why does ORS contain both salt AND glucose? The cotransporter that links them is the answer.



91. Which of the following enzymes is active in its **dephosphorylated** state?

- (A) Glycogen synthase
- (B) Glycogen phosphorylase
- (C) PFK-2 (phosphofructokinase-2)
- (D) Fructose-2,6-bisphosphatase

Correct Answer: (A) Glycogen synthase

Solution:

Step 1: Understand the covalent regulation.

Many metabolic enzymes are regulated by reversible phosphorylation. Phosphorylation (driven by cAMP-dependent protein kinase A in the fasting/adrenaline state) and dephosphorylation (driven by protein phosphatase-1 in the fed/insulin state) switch enzymes ON or OFF depending on whether the body needs to store or mobilise fuel.

Step 2: Apply it to glycogen metabolism.

In the well-fed state, insulin promotes dephosphorylation. **Glycogen synthase** is active (a) in its *dephosphorylated* form, favouring glycogen synthesis. Conversely, glycogen phosphorylase is active (a) in its *phosphorylated* form, favouring glycogen breakdown. The two are reciprocally regulated so synthesis and degradation do not occur simultaneously.

Step 3: Eliminate the other options.

Glycogen phosphorylase to active when phosphorylated. The bifunctional PFK-2/fructose-2,6-bisphosphatase enzyme: when *dephosphorylated* the kinase (PFK-2) domain is active (raising fructose-2,6-bisphosphate, promoting glycolysis), and when *phosphorylated* the phosphatase (F-2,6-BPase) domain is active. So PFK-2 active dephosphorylated is also true in the liver, but the single classical 'dephosphorylated = active' storage enzyme tested here is glycogen synthase.

Key fact: Glycogen synthase is the storage-promoting enzyme that is switched ON by dephosphorylation in the insulin-driven fed state.

Quick Tip: Fed-state insulin dephosphorylates and switches ON the glycogen-storing enzyme.

92. Which of the following bacterial toxins acts by inhibiting elongation factor 2 (EF-2)?

- (A) Diphtheria toxin
- (B) Cholera toxin
- (C) Tetanospasmin
- (D) Heat-labile toxin of *E. coli*

Correct Answer: (A) Diphtheria toxin

Solution:

Step 1: Identify the molecular target.

Elongation factor 2 (EF-2) is required for the translocation step of protein synthesis on the ribosome. Inhibiting EF-2 halts host-cell protein synthesis and kills the cell.

Step 2: Recall the toxin mechanism.

Diphtheria toxin (from *Corynebacterium diphtheriae*) is an A-B exotoxin. Its A subunit catalyses *ADP-ribosylation* of the unique amino acid diphthamide on EF-2,

inactivating it. *Pseudomonas aeruginosa* exotoxin A works by the identical mechanism, but diphtheria toxin is the classic answer.

Step 3: Eliminate the distractors.

Cholera toxin and the heat-labile toxin of *E. coli* ADP-ribosylate the Gs alpha subunit, locking adenylate cyclase ON and raising cAMP (watery diarrhoea) - they do not touch EF-2. Tetanospasmin is a protease that cleaves synaptobrevin, blocking inhibitory neurotransmitter release - again not EF-2.

Key fact: Diphtheria toxin inhibits EF-2 by ADP-ribosylating diphthamide, arresting host protein synthesis.

Quick Tip: The classic EF-2 ADP-ribosylating A-B exotoxin is from *Corynebacterium*.



93. A patient presents with septic arthritis. Culture grows a Gram-positive coccus that is catalase positive and **cefoxitin sensitive**. What is the drug of choice?

- (A) Cloxacillin
- (B) Penicillin
- (C) Vancomycin
- (D) Linezolid

Correct Answer: (A) Cloxacillin

Solution:

Step 1: Identify the organism.

A Gram-positive coccus that is *catalase positive* is a *Staphylococcus* (streptococci are catalase negative). In septic arthritis, the commonest cause is *Staphylococcus aureus*.

Step 2: Interpret the cefoxitin result.

Cefoxitin is the surrogate disc used to detect the *mecA*-mediated methicillin resistance (MRSA). **Cefoxitin sensitive** means the isolate is *methicillin-sensitive S.*

aureus (MSSA), not MRSA.

Step 3: Choose the drug.

For MSSA, the drugs of choice are the anti-staphylococcal penicillinase-resistant penicillins - **cloxacillin** / flucloxacillin / nafcillin / oxacillin. These are more effective than vancomycin against MSSA.

Step 4: Eliminate the distractors.

Plain penicillin G is destroyed by staphylococcal beta-lactamase, so >90% of *S. aureus* are resistant - not reliable. Vancomycin and linezolid are reserved for MRSA (cefoxitin-resistant) isolates and are actually inferior to cloxacillin for MSSA.

Key fact: Cefoxitin-sensitive (MSSA) staphylococcal infection to treat with an anti-staphylococcal penicillin such as cloxacillin.

Quick Tip: Cefoxitin sensitive = MSSA, so use a penicillinase-resistant penicillin, not vancomycin.



94. A poorly controlled diabetic patient presents with facial pain and a **black eschar** over the nasal/palatal region. Which organism is most likely responsible?

- (A) *Rhizopus* species (mucormycosis)
- (B) *Aspergillus* species
- (C) *Candida* species
- (D) *Cryptococcus neoformans*

Correct Answer: (A) *Rhizopus* species (mucormycosis)

Solution:

Step 1: Recognise the classic clinical picture.

A diabetic (especially in ketoacidosis) with rapidly progressing facial pain and a **black necrotic eschar** on the palate or nose is the textbook presentation of

rhino-orbito-cerebral mucormycosis.

Step 2: Identify the fungus.

Mucormycosis is caused by the order Mucorales - chiefly *Rhizopus* (also *Mucor*, *Rhizomucor*, *Lichtheimia*). On microscopy these show broad, aseptate (or sparsely septate) ribbon-like hyphae branching at wide ($\sim 90^\circ$) angles. They are angioinvasive, causing thrombosis and the characteristic black eschar from tissue necrosis.

Step 3: Why diabetics?

Hyperglycaemia and acidosis impair neutrophil function and increase free iron availability; *Rhizopus* also has a ketone reductase that lets it thrive in the acidotic, glucose-rich milieu.

Step 4: Eliminate the distractors.

Aspergillus has narrow septate hyphae branching at acute ($\sim 45^\circ$) angles and typically causes invasive disease in neutropenic/immunosuppressed hosts - not the diabetic black-eschar picture. *Candida* causes mucocutaneous/invasive candidiasis, not an invasive facial eschar. *Cryptococcus* causes meningitis in HIV/immunosuppressed patients.

Key fact: Diabetic + black palatal/nasal eschar = mucormycosis, classically due to *Rhizopus*.

Quick Tip: Diabetic ketoacidosis + black eschar = mucormycosis (broad aseptate hyphae).

95. What is the approximate amplitude (power) of accommodation in a 7-year-old child?

- (A) +14 D
- (B) +10 D
- (C) +5 D
- (D) +2 D

Correct Answer: (A) +14 D

Solution:

Step 1: Recall the concept.

The amplitude of accommodation is the maximum increase in dioptric power the eye can generate by contracting the ciliary muscle to focus on near objects. It is highest in childhood and falls progressively with age as the crystalline lens loses elasticity (eventually causing presbyopia).

Step 2: Apply Hofstetter's formula.

A standard estimate of the average (probable) amplitude of accommodation is:

$$A = 18.5 - (0.30 \times \text{age})$$

For a 7-year-old:

$$A = 18.5 - (0.30 \times 7) = 18.5 - 2.1 = 16.4 \text{ D}$$

The closest standard textbook value quoted for a young child is about **+14 D**, so option (a) is correct (+10 D and +5 D correspond to much older ages).

Step 3: Cross-check with the age trend.

Accommodation is roughly 14 D in early childhood, ~7-8 D in early adulthood (about age 20), ~4 D by 40 years, and falls to ~1 D by 60 years. A value of +14 D fits a 7-year-old far better than +10 D or +5 D.

Key fact: A young child (~7 years) has an accommodative amplitude of about +14 D; it declines steadily with age.

Quick Tip: Hofstetter: amplitude = $18.5 - 0.3 \times \text{age}$; young children have the highest value.



96. Neuropraxia (the mildest grade of nerve injury) is best described as:

- (A) A reversible physiological conduction block with intact axon
- (B) Cut/disruption of the endoneurium
- (C) Cut/disruption of the nerve sheath (epineurium)
- (D) Complete transection of the whole nerve trunk

Correct Answer: (A) A reversible physiological conduction block with intact axon

Solution:

Step 1: Recall Seddon's classification of nerve injury.

Seddon graded nerve injury into three types of increasing severity: *neuropraxia*, *axonotmesis* and *neurotmesis*.

Step 2: Define neuropraxia.

Neuropraxia is the mildest injury. There is a *physiological (functional) conduction block* - typically from segmental demyelination - but the axon and all connective tissue layers (endoneurium, perineurium, epineurium) remain *anatomically intact*. Because the axon is not severed, there is *no* Wallerian degeneration, and recovery is complete and usually rapid (days to weeks).

Step 3: Contrast with the other grades.

In *axonotmesis* the axon and myelin are disrupted (Wallerian degeneration occurs) but the endoneurial tube/connective tissue is preserved, allowing axonal regrowth. In *neurotmesis* the entire nerve including its sheath (endoneurium/perineurium/epineurium) is divided, so spontaneous recovery is poor and surgical repair is usually needed.

Step 4: Match the options.

'Endoneurium cut' corresponds to axonotmesis/neurotmesis, and

“sheath cut” corresponds to neurotmesis. Only “physiological block” describes neuropraxia.

Key fact: Neuropraxia = reversible physiological conduction block with an intact axon and no Wallerian degeneration.

Quick Tip: Mildest Seddon grade - conduction blocked but axon and sheath stay intact.

97. Neurapraxia is best described as:

- (A) Physiological conduction block with intact axon
- (B) Endoneurium and axon cut
- (C) Nerve sheath (perineurium) cut
- (D) Complete transection of the whole nerve trunk

Correct Answer: (A) Physiological conduction block with intact axon

Solution:

Step 1: Recall Seddon's classification of nerve injury, which has three grades - neurapraxia, axonotmesis and neurotmesis.

Step 2: Neurapraxia is the mildest injury. It is a **physiological (functional) conduction block** usually due to segmental demyelination. The **axon remains in continuity** and the connective tissue sheaths (endoneurium, perineurium, epineurium) are intact.

Step 3: Because the axon is not divided, there is **no Wallerian degeneration**. Recovery is complete and relatively rapid (days to weeks) once the demyelinated segment remyelinates. EMG shows no denervation/fibrillation.

Step 4: Why the others are wrong - "Endoneurium/axon cut" describes **axonotmesis** (axon and myelin disrupted, sheath preserved, Wallerian

degeneration occurs). "Sheath cut" or complete transection describes **neurotmesis**, the most severe injury where the nerve including its sheaths is divided, requiring surgical repair.

Key fact: Neurapraxia = reversible physiological conduction block with an intact axon and no Wallerian degeneration.

Quick Tip: Mildest Seddon grade - axon intact, only conduction temporarily blocked.



98. An infant presents with intensely itchy erythematous papules and vesicles distributed over the palms, soles and umbilical region, with similar lesions in family members. The most likely diagnosis is:

- (A) Scabies
- (B) Psoriasis
- (C) Atopic dermatitis
- (D) Seborrhoeic dermatitis

Correct Answer: (A) Scabies

Solution:

Step 1: Identify the classic distribution. In infants, **scabies** characteristically involves the **palms, soles, axillae, umbilicus, genitalia and scalp/face** - sites that are typically spared in older children and adults.

Step 2: The morphology fits - intense **pruritus (worse at night)** with erythematous papules, vesicles, pustules and sometimes burrows or nodules. Vesiculation on palms and soles is especially typical of infantile scabies.

Step 3: The history of **similar lesions in family members** is a strong clue, since scabies (*Sarcoptes scabiei*) is highly contagious and spreads among close contacts.

Step 4: Why the others are wrong - **Atopic dermatitis** in infants favours the

cheeks and extensor surfaces and spares the umbilicus; it is not classically vesicular on palms/soles nor transmitted to contacts. **Psoriasis** shows well-demarcated scaly plaques, is uncommon and non-pruritic-vesicular in infants. **Seborrhoeic dermatitis** gives greasy yellow scales on scalp/face/folds and is non-pruritic.

Key fact: Itchy palmoplantar and umbilical vesicles in an infant with affected contacts = scabies.

Quick Tip: Palms, soles, umbilicus + nocturnal itch + affected family contacts.

99. A patient has an irregular black pigmented lesion on the heel/sole suspicious for acral melanoma. The most appropriate confirmatory investigation is:

- (A) Excisional / full-thickness biopsy with histopathology
- (B) KOH mount / Gram stain
- (C) Tzanck smear
- (D) Wood's lamp examination

Correct Answer: (A) Excisional / full-thickness biopsy with histopathology

Solution:

Step 1: A new or changing **black pigmented lesion on the heel/sole** in an adult must be treated as possible **acral lentiginous melanoma** until proven otherwise - the most common melanoma subtype in dark-skinned and Asian populations.

Step 2: Definitive diagnosis of any suspected melanoma requires **histopathology**. The gold standard is an **excisional (full-thickness) biopsy** that allows measurement of **Breslow thickness**, which guides staging and management. (A full-thickness incisional/punch biopsy of the thickest area is acceptable when the lesion is large.)

Step 3: Why the others are wrong - **KOH/Gram stain** detects fungi/bacteria and

is useless for a pigmented neoplasm. **Tzanck smear** looks for multinucleated giant cells in vesicular viral lesions (herpes/varicella) or pemphigus, not melanoma. **Wood's lamp** highlights pigment depth and some infections but cannot diagnose malignancy.

Key fact: Suspected acral melanoma to biopsy with histopathology for diagnosis and Breslow thickness.

Quick Tip: Pigmented lesion suspicious of melanoma - only tissue (biopsy) confirms it.



100. An Afghan national arriving at the airport is found to have swallowed multiple well-sealed, professionally prepared condoms/packets filled with narcotic drugs for the purpose of international trafficking. This practice is termed:

- (A) Body packing
- (B) Body stuffing
- (C) Masking
- (D) Body pushing

Correct Answer: (A) Body packing

Solution:

Step 1: Distinguish the two main forms of internal drug concealment. **Body packing** ("mules") is the **premeditated** swallowing/insertion of **large numbers of well-prepared, tightly wrapped packets** of drugs for **cross-border smuggling**.

Step 2: The vignette fits body packing exactly - an international traveller, multiple **professionally sealed condom packets**, intent to traffic. Each packet carries a potentially lethal dose, so rupture can cause massive overdose; bowel obstruction is also a risk.

Step 3: Why the others are wrong - **Body stuffing** is the **hurried, impulsive**

swallowing of **poorly wrapped** drugs to **destroy evidence** during imminent arrest (smaller amounts, higher rupture risk). **Masking** refers to disguising/adulterating a drug or its detection, not internal concealment. "Body pushing" is not a recognised term.

Key fact: Planned smuggling of many well-wrapped drug packets in the GI tract = body packing.

Quick Tip: Pre-planned smuggling, many well-sealed packets - not the panic act of stuffing.



101. An infant girl shows normal early development followed by regression of acquired milestones at around 6-18 months, with deceleration of head growth and loss of purposeful hand use replaced by stereotyped hand-wringing movements. The most likely diagnosis is:

- (A) Rett syndrome
- (B) Childhood disintegrative disorder (Heller syndrome)
- (C) Classic autism spectrum disorder
- (D) Attention-deficit/hyperactivity disorder

Correct Answer: (A) Rett syndrome

Solution:

Step 1: Key feature is **regression of milestones beginning early (around 6-18 months)** after a period of normal development, in a **girl**.

Step 2: This points to **Rett syndrome**, an X-linked neurodevelopmental disorder (*MECP2* mutation) seen almost exclusively in girls. Hallmarks: **deceleration of head growth (acquired microcephaly)**, loss of purposeful hand skills with **stereotyped hand-wringing/washing movements**, gait abnormalities and loss of language.

Step 3: Timing is the discriminator. **Childhood disintegrative disorder (Heller syndrome)** shows normal development for **at least 2 years (usually >3-4 years)** before a marked regression - consistent with the examiner's own note ">2 yrs / Heller". Regression as early as 6 months therefore favours Rett, not Heller.

Step 4: Why the others are wrong - classic **autism** involves persistent social-communication deficits without the characteristic acquired microcephaly and hand-wringing of Rett; **ADHD** is a disorder of attention/hyperactivity with no milestone regression.

Key fact: Early regression + acquired microcephaly + hand-wringing stereotypies in a girl = Rett syndrome; late regression (>2 yrs) = Heller/childhood disintegrative disorder.

Quick Tip: Girl, early regression, shrinking head, hand-wringing - think MECP2.

102. Which adverse effect is most characteristically associated with the antiepileptic drug lamotrigine?

- (A) Severe cutaneous reactions including Stevens-Johnson syndrome / dizziness
- (B) Marked dose-independent hepatotoxicity
- (C) Gingival hyperplasia
- (D) Irreversible visual field constriction

Correct Answer: (A) Severe cutaneous reactions including Stevens-Johnson syndrome / dizziness

Solution:

Step 1: Recall the signature toxicity of **lamotrigine**, a broad-spectrum antiepileptic that blocks voltage-gated sodium channels and reduces glutamate release.

Step 2: Its most feared and most characteristic adverse effect is a **rash**, ranging

from benign maculopapular eruptions to life-threatening **Stevens-Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN)**. The risk is highest with **rapid dose escalation** and with **co-administration of valproate** (which inhibits lamotrigine metabolism) - hence the mandated slow titration. Common dose-related CNS effects include **dizziness**, diplopia, headache and ataxia.

Step 3: Why the other options are wrong - serious **hepatotoxicity** is the hallmark of **valproate** (and felbamate), not lamotrigine. **Gingival hyperplasia** is classic for **phenytoin**. **Irreversible visual field constriction** is the signature toxicity of **vigabatrin**.

Key fact: Lamotrigine to skin rash / SJS-TEN (titrate slowly, risk increased by valproate); dizziness is a common dose-related effect.

Quick Tip: Slow-titrated AED feared for skin rash and SJS, especially with valproate.



103. In *Bacillus anthracis*, the poly-D-glutamic acid (poly- γ -D-glutamate) capsule is encoded by which plasmid?

- (A) pXO1 (toxin plasmid)
- (B) pXO2 (capsule plasmid)
- (C) Chromosomal genes only
- (D) pXO1 and pXO2 jointly

Correct Answer: (B) pXO2 (capsule plasmid)

Solution:

Step 1: Identify the virulence factors of *Bacillus anthracis*.

Full virulence of *B. anthracis* requires two plasmids: **pXO1** and **pXO2**. Loss of either renders the organism attenuated.

Step 2: Map each plasmid to its product.

• **pXO1** (~182 kb) carries the genes for the tripartite anthrax **toxin** -

protective antigen (PA, *pagA*), lethal factor (LF, *lef*) and edema factor (EF, *cya*).
• **pXO2** (~95 kb) carries the *capBCADE* operon that synthesises the antiphagocytic **poly-D-glutamic acid capsule**.

Step 3: Apply to the question.

The capsule is therefore encoded by **pXO2**. The poly-glutamate capsule is unusual because most bacterial capsules are polysaccharide; this protein/peptide capsule resists phagocytosis and is poorly immunogenic, which is why the live attenuated Sterne vaccine strain (pXO1⁺, pXO2⁻) is non-capsulated.

Step 4: Why the other options are wrong.

pXO1 codes for toxin, not capsule. The capsule is plasmid-borne, not chromosomal. Both plasmids are needed for full virulence, but the capsule specifically maps to pXO2 alone.

Key fact: pXO1 = toxin, **pXO2 = capsule** (poly-D-glutamate).

Quick Tip: Two plasmids: one for toxin, one for the protein capsule - capsule is on pXO2.

104. Which of the following is a **novel (newer) antidepressant** that acts as a serotonin partial agonist and reuptake inhibitor (SPARI)?

- (A) Vilazodone
- (B) Trazodone
- (C) Imipramine
- (D) Amitriptyline

Correct Answer: (A) Vilazodone

Solution:

Step 1: Read what is being asked.

The question wants the **novel** (most recently introduced) antidepressant among

the choices.

Step 2: Classify each drug.

• **Vilazodone** - a newer agent (FDA-approved 2011) and the prototype

SPARI: it combines selective serotonin reuptake inhibition with 5-HT_{1A} receptor *partial agonism*. Vortioxetine belongs to the same modern class.

• **Trazodone** - an older (1980s) serotonin antagonist/reuptake inhibitor (SARI), widely used for its sedative effect in depression with insomnia.

• **Imipramine** and **Amitriptyline** - classic *tricyclic antidepressants*, among the oldest agents.

Step 3: Choose the novel drug.

By recency and mechanism, **Vilazodone** is the novel antidepressant.

Step 4: Why the others are wrong.

Trazodone, imipramine and amitriptyline are all long-established drugs, so none qualifies as “novel”;

Key fact: Vilazodone = SPARI = a newer antidepressant (SSRI action + 5-HT_{1A} partial agonism).

Quick Tip: Think SPARI: SSRI plus 5-HT_{1A} partial agonism - the newest of the four.

105. A phenotypic female presents with primary amenorrhoea, an **absent uterus**, **normally developed breasts** and **sparse/absent pubic and axillary hair**. What is the most likely diagnosis?

- (A) Androgen insensitivity syndrome (complete AIS)
- (B) Mayer-Rokitansky-Küster-Hauser (MRKH) syndrome
- (C) Turner syndrome (45,XO)
- (D) Kallmann syndrome

Correct Answer: (A) Androgen insensitivity syndrome (complete AIS)

Solution:

Step 1: List the clues.

Primary amenorrhoea + absent uterus + **well-developed breasts** + **scanty pubic/axillary hair** in a phenotypic female.

Step 2: Interpret the hair and breast findings.

• **Normal breasts** imply adequate oestrogen action (here from peripheral aromatisation of testosterone).

• **Sparse sexual hair** indicates the tissues cannot respond to androgens, because pubic/axillary hair is androgen-dependent. This pairing - oestrogenised but androgen-unresponsive - is the hallmark of **complete androgen insensitivity syndrome (CAIS)**.

Step 3: Explain the absent uterus.

In CAIS the karyotype is **46,XY** with functioning testes that secrete anti-Müllerian hormone (AMH); AMH causes regression of the Müllerian ducts, so the uterus, cervix and upper vagina are absent. Testosterone is made but cannot act (defective androgen receptor), giving female external genitalia.

Step 4: Why the other options are wrong.

• **MRKH** also has absent uterus and normal breasts, but pubic/axillary hair is *normal* (46,XX, normal androgen action) - here the hair is sparse, favouring AIS.

• **Turner syndrome** typically has a uterus present but poor breast development (streak ovaries, low oestrogen).

• **Kallmann syndrome** shows hypogonadism with anosmia and poor breast development; uterus is present.

Key fact: Absent uterus + good breasts + **scanty sexual hair** to complete androgen insensitivity syndrome (46,XY).

Quick Tip: Good breasts but absent uterus and sparse pubic hair - androgens cannot act despite male genotype.

106. The clinical photograph and slit-lamp image show iris hamartomas (Lisch nodules) with skin findings in a child with neurofibromatosis type 1 (NF1). Which cutaneous lesion is the earliest and most characteristic feature of NF1?

- (A) Café-au-lait macules
- (B) Ash-leaf (hypopigmented) macules
- (C) Port-wine stain
- (D) Shagreen patch

Correct Answer: (A) Café-au-lait macules

Solution:

Step 1: Recognise the condition.

Lisch nodules (iris hamartomas) on slit-lamp plus the clinical skin photo point to **neurofibromatosis type 1 (NF1)**, an autosomal dominant neurocutaneous disorder caused by mutations in the *NF1* gene (neurofibromin, chromosome 17).

Step 2: Recall the diagnostic criteria.

NF1 diagnosis needs ≥2 of: ≥6 café-au-lait macules, ≥2 neurofibromas or 1 plexiform neurofibroma, axillary/inguinal freckling (Crowe sign), ≥2 Lisch nodules, optic glioma, a distinctive bony lesion, and a first-degree relative with NF1.

Step 3: Identify the earliest cutaneous sign.

Café-au-lait macules (CALM) are flat, well-demarcated light-brown patches that usually appear first, often at birth or in infancy, before neurofibromas develop.

Step 4: Why the others are wrong.

• **Ash-leaf macules** and **Shagreen patch** are features of *tuberous sclerosis*, not NF1.

• **Port-wine stain** is seen in Sturge-Weber syndrome.

Key fact: The hallmark and earliest skin lesion of NF1 is the **café-au-lait macule** (≥ 6 of ≥ 15 mm is a diagnostic criterion).

Quick Tip: Lisch nodules + skin patches = NF1; its earliest skin marker is a flat light-brown macule.

107. The photograph shows a permanent tattoo on the skin. In which layer of the skin is the tattoo pigment chiefly deposited and retained?

- (A) Dermis
- (B) Stratum corneum of the epidermis
- (C) Subcutaneous fat
- (D) Deep fascia

Correct Answer: (A) Dermis

Solution:

Step 1: Understand how a tattoo is made.

A tattoo needle introduces insoluble pigment particles by puncturing through the epidermis and depositing ink in the **dermis**.

Step 2: Why the dermis retains the pigment.

Epidermal cells are constantly shed and renewed (turnover ~ 4 weeks), so any pigment left in the epidermis would be lost. In the dermis, pigment is engulfed by dermal **macrophages** and fibroblasts and held within a stable connective-tissue matrix, giving the tattoo its **permanence**.

Step 3: Forensic relevance.

Because pigment lies in the dermis, tattoos are useful for **identification** of the dead and can persist even after superficial decomposition; the pigment may also be traced to regional lymph nodes.

Step 4: Why the other options are wrong.

• The **stratum corneum/epidermis** sheds continually, so pigment there would not persist.

• **Subcutaneous fat** and **deep fascia** are deeper than the needle's working depth and are not the site of deposition.

Key fact: Tattoo pigment is permanently lodged in the **dermis**.

Quick Tip: The epidermis keeps shedding - permanence means the ink must sit one layer deeper.

108. What is the specific antidote used in acute **opioid** (e.g. heroin, morphine) poisoning?

- (A) Naloxone
- (B) Flumazenil
- (C) Atropine
- (D) N-acetylcysteine

Correct Answer: (A) Naloxone

Solution:

Step 1: Recall the opioid toxidrome.

Opioid overdose produces the triad of **pinpoint (miotic) pupils, respiratory depression and CNS depression/coma**. Death is from respiratory failure.

Step 2: Identify the antidote and its mechanism.

Naloxone is a pure, competitive **μ -opioid receptor antagonist**. Given IV/IM/intranasal, it rapidly reverses respiratory depression and coma. Because it is short-acting, repeated doses or an infusion may be needed when a long-acting opioid (e.g. methadone) is involved.

Step 3: Match antidotes to their poisons (why the others are wrong).

• **Flumazenil** to benzodiazepine reversal.

• **Atropine** to organophosphate / carbamate (cholinergic) poisoning and bradycardia.

• **N-acetylcysteine** to paracetamol (acetaminophen) overdose.

Key fact: The antidote for opioid poisoning is **naloxone**, an opioid-receptor antagonist that reverses respiratory depression.

Quick Tip: Pinpoint pupils + slow breathing - reverse with the opioid-receptor blocker.



109. According to the AAST (American Association for the Surgery of Trauma) Colon Injury Scale, a laceration involving more than 50% of the bowel circumference (without transection) corresponds to which grade?

- (A) Grade I
- (B) Grade II
- (C) Grade III
- (D) Grade IV

Correct Answer: (C) Grade III

Solution:

Step 1: Recall the AAST Colon Injury Scale.

The AAST organ injury scaling for the colon grades injury by extent of wall involvement and devascularisation:

• Grade I: Contusion/haematoma without devascularisation, or partial-thickness laceration.

• Grade II: Laceration involving <50% of the circumference.

• Grade III: Laceration involving >50% of the circumference (without transection).

• Grade IV: Transection of the colon.

• Grade V: Transection with segmental tissue loss / devascularised segment.

Step 2: Apply to the stem.

The injury described is >50% circumferential laceration without full transection, which maps exactly to **Grade III**.

Step 3: Eliminate others.

Grade II is <50% circumference, Grade IV is complete transection, and Grade I is contusion/partial-thickness. Hence the answer is Grade III.

Key fact: >50% circumferential colon laceration = AAST Grade III.

Quick Tip: Less than 50% circumference is Grade II; more than 50% (no transection) bumps it up one grade.



110. A trauma patient opens his eyes only to painful stimulus, makes incomprehensible sounds, and localises to pain. What is his total Glasgow Coma Scale (GCS) score?

- (A) 8
- (B) 9
- (C) 10
- (D) 11

Correct Answer: (B) 9

Solution:

Step 1: Score Eye opening (E).

Eye opening to pain = E2 (spontaneous 4, to sound/speech 3, to pain 2, none 1).

Step 2: Score Verbal response (V).

Incomprehensible sounds = V2 (oriented 5, confused 4, inappropriate words 3, incomprehensible sounds 2, none 1).

Step 3: Score Motor response (M).

Localises to pain = M5 (obeys commands 6, localises 5, withdraws 4, abnormal

flexion 3, extension 2, none 1).

Step 4: Add up.

$$\text{GCS} = \text{E2} + \text{V2} + \text{M5} = 9.$$

Key fact: Eye-to-pain (2) + incomprehensible sounds (2) + localises (5) = GCS 9, placing the patient in the moderate head-injury range (9-12).

Quick Tip: E2 + V2 + M5 - just add the three best responses.

111. An elderly man with a history of atrial fibrillation presents with sudden severe abdominal pain that is out of proportion to physical findings. Which laboratory finding most strongly supports the diagnosis of acute mesenteric ischemia?

- (A) Raised serum lactate (and LDH)
- (B) Raised serum amylase only
- (C) Hypocalcaemia
- (D) Raised serum bilirubin

Correct Answer: (A) Raised serum lactate (and LDH)

Solution:

Step 1: Identify the clinical scenario.

Atrial fibrillation predisposes to embolism; an embolus lodging in the superior mesenteric artery causes acute mesenteric ischemia. The hallmark is *pain out of proportion* to the examination findings.

Step 2: Understand the biochemistry.

Ischaemic bowel undergoes anaerobic metabolism and cell death, releasing lactate and lactate dehydrogenase (LDH). A raised serum lactate (metabolic acidosis with high anion gap) is the most useful and characteristic biochemical marker, and LDH rises with tissue necrosis.

Step 3: Eliminate the distractors.

Amylase may rise modestly but is non-specific (also raised in pancreatitis). Hypocalcaemia and hyperbilirubinaemia are not features of early mesenteric ischemia.

Key fact: AF + pain out of proportion + raised lactate/LDH = acute (embolic) mesenteric ischemia; CT angiography confirms it.

Quick Tip: Dying gut means anaerobic metabolism - look at the lactate.

112. Which of the following is NOT a feature of Kawasaki disease?

- (A) Purulent conjunctivitis
- (B) Bilateral non-exudative conjunctival injection
- (C) Strawberry tongue and cracked lips
- (D) Cervical lymphadenopathy

Correct Answer: (A) Purulent conjunctivitis

Solution:

Step 1: Recall the diagnostic criteria of Kawasaki disease.

Fever $\geq$ 5 days plus $\geq$ 4 of: (1) bilateral *non-purulent (non-exudative) bulbar conjunctival injection*, (2) oral mucosal changes - cracked red lips, strawberry tongue, (3) polymorphous rash, (4) extremity changes - erythema/oedema then periungual peeling, (5) cervical lymphadenopathy ($\geq$ 1.5 cm, usually unilateral).

Step 2: Pick the odd one out.

The conjunctivitis of Kawasaki disease is characteristically **non-purulent / non-exudative**, with limbal sparing and no discharge. **Purulent conjunctivitis is NOT a feature** - its presence should prompt a search for an alternative infective diagnosis.

Step 3: Confirm the others.

Bilateral non-exudative conjunctival injection, strawberry tongue/cracked lips, and cervical lymphadenopathy are all genuine criteria.

Key fact: Kawasaki conjunctivitis is bilateral, bulbar, and non-purulent; pus/discharge argues against the diagnosis.

Quick Tip: Kawasaki conjunctivitis has no discharge - pus points away from it.



113. A term neonate has a blood glucose of 36 mg/dL. The infant is feeding well and has no clinical signs. Which characteristic of this neonate favours continued breastfeeding (with monitoring) rather than immediate intravenous dextrose infusion?

- (A) The neonate is asymptomatic
- (B) The neonate is symptomatic with jitteriness
- (C) The neonate is having seizures
- (D) The neonate is lethargic and not feeding

Correct Answer: (A) The neonate is asymptomatic

Solution:

Step 1: Frame the management of neonatal hypoglycaemia.

Management hinges on whether the baby is *symptomatic* and on the absolute glucose value. Symptoms (jitteriness, lethargy, poor feeding, apnoea, seizures) mandate immediate IV dextrose (e.g., a mini-bolus of 2 mL/kg of 10% dextrose followed by infusion).

Step 2: Apply to this neonate.

This term infant is **asymptomatic** with a glucose of 36 mg/dL and is feeding well. An asymptomatic infant with mild hypoglycaemia in this range can be managed by encouraging/continuing breastfeeding (or oral feeds) with a glucose recheck after the feed, reserving IV dextrose for failure to respond or development of symptoms.

Step 3: Eliminate the others.

Jitteriness, seizures and lethargy/poor feeding are all symptomatic states that require IV dextrose, not conservative feeding.

Key fact: Asymptomatic mild neonatal hypoglycaemia to feed and recheck; symptomatic hypoglycaemia to IV dextrose.

Quick Tip: No symptoms and feeding well? Feed and recheck rather than reach for the IV.

114. Regarding the HPV vaccine, which of the following statements is INCORRECT?

- (A) It guarantees 100% complete protection against cervical cancer
- (B) The primary target group for vaccination is girls aged 9-14 years
- (C) A single-dose schedule is now endorsed by WHO and national guidelines for the targeted age group
- (D) It is highly immunogenic, with high seroconversion rates in the target age group

Correct Answer: (A) It guarantees 100% complete protection against cervical cancer

Solution:

Step 1: Review HPV vaccine facts.

The HPV vaccine is recommended primarily for girls aged **9-14 years** (before sexual debut). The WHO SAGE and national programmes now accept a **single-dose** schedule for this age group, and the vaccine is highly immunogenic.

Step 2: Spot the incorrect statement.

No vaccine provides **100% complete protection**. HPV vaccines cover the high-risk genotypes (e.g., 16 and 18, responsible for ~70% of cervical cancers, plus additional types in nonavalent vaccines) but do not protect against every oncogenic HPV type. Therefore the claim of complete/100% protection is **incorrect**, and cervical screening must continue.

Step 3: Confirm the valid statements.

The 9-14 target group, single-dose schedule (WHO/national), and high immunogenicity are all correct.

Key fact: HPV vaccine markedly reduces but does not eliminate cervical cancer risk - it is not 100% protective, so screening remains essential.

Quick Tip: No vaccine is 100% - HPV jabs cover the major genotypes, not all of them.



115. A researcher plots the average per-capita alcohol intake of 10 different countries against the cirrhosis mortality rate of each of those countries, using one data point per country, to look for an association. Which type of epidemiological study design does this represent?

- (A) Ecological study
- (B) Cohort study
- (C) Case-control study
- (D) Cross-sectional study

Correct Answer: (A) Ecological study

Solution:

Step 1: Identify the unit of observation. In this study each data point is an entire **country** (a population/group), not an individual person. Per-capita alcohol intake and cirrhosis mortality are both group-level (aggregate) measures averaged over the whole country.

Step 2: Apply the definition of an ecological study. An ecological (correlational) study examines the relationship between an exposure and an outcome at the level of **populations or groups** rather than individuals. Comparing the mean exposure of a group with the mean outcome of that same group across several groups is the hallmark of this design. Here, 10 countries serve as the 10 groups.

Step 3: Why the others are wrong. A **cohort study** follows exposed and unexposed *individuals* over time for incidence — not the case here. A **case-control study** starts from individuals with and without disease and looks back at exposure — again individual-level. A **cross-sectional study** measures exposure and outcome in *individuals* at one point in time. All three use the individual as the unit of analysis, whereas this study uses the country.

Step 4: Note the limitation. Because data are aggregated, an ecological study is prone to the **ecological fallacy** — a group-level association may not hold for individuals. It is best used for hypothesis generation.

Key fact: Comparing average alcohol intake versus cirrhosis across 10 countries (group as the unit) is an **ecological study**.

Quick Tip: The unit of analysis is the whole country (a group), not the individual.



116. In a case-control study, among the cases there are 60 exposed and 20 unexposed individuals, while among the controls there are 40 exposed and 80 unexposed individuals. What is the odds ratio of exposure?

- (A) 6
- (B) 3
- (C) 1.5
- (D) 0.17

Correct Answer: (A) 6

Solution:

Step 1: Lay out the 2×2 table. Let a = exposed cases = 60, b = exposed controls = 40, c = unexposed cases = 20, d = unexposed controls = 80.

Step 2: Recall the odds ratio formula. For a case-control study the odds ratio (OR) is the cross-product ratio:

$$OR = \frac{a \times d}{b \times c}$$

Step 3: Substitute the values.

$$OR = \frac{60 \times 80}{40 \times 20} = \frac{4800}{800} = 6$$

Step 4: Interpret. An OR of 6 means the odds of exposure among cases are 6 times the odds among controls, indicating a strong positive association between the exposure and the disease ($OR > 1$).

Step 5: Why the others are wrong. 3, 1.5 and 0.17 arise from mis-pairing the cells (e.g. using $a/c \div b/d$ incorrectly or inverting the cross-product). The correct diagonal cross-product is $(60 \times 80)/(40 \times 20)$.

Key fact: $OR = (ad)/(bc) = (60 \times 80)/(20 \times 40) = 6$.

Quick Tip: Odds ratio = cross-product ad/bc of the 2×2 table.

117. A new screening test is applied. It yields 90 true positives and 50 false positives. What is the positive predictive value (PPV) of the test?

- (A) 64.3%
- (B) 90%
- (C) 50%
- (D) 35.7%

Correct Answer: (A) 64.3%

Solution:

Step 1: Recall the definition of PPV. The positive predictive value is the probability that a person who tests positive truly has the disease. It uses only those who tested positive:

$$PPV = \frac{\text{True Positives}}{\text{True Positives} + \text{False Positives}}$$

Step 2: Identify the numbers. True positives (TP) = 90 and false positives (FP) = 50, so the total number testing positive = $90 + 50 = 140$.

Step 3: Substitute.

$$PPV = \frac{90}{140} = 0.643 = 64.3\%$$

Step 4: Interpret. About 64% of those flagged positive by the test actually have the disease; the remaining ~36% are false alarms.

Step 5: Why the others are wrong. 90% wrongly treats all 90 TP as the whole positive group. 50% has no valid derivation. 35.7% is the complement (50/140), i.e. the false-positive proportion among positives, not the PPV.

Key fact: $PPV = TP / (TP + FP) = 90/140 \approx 64.3\%$.

Quick Tip: $PPV = \text{true positives} \div \text{all who tested positive (TP + FP)}$.



118. An influenza vaccine shows 90% efficacy in a randomized controlled trial but only 85% performance when used in the routine community programme. Which value represents the **effectiveness** of the vaccine?

- (A) 85% (performance in real-world field conditions)
- (B) 90% (performance in the ideal trial setting)
- (C) 5% (difference between the two values)
- (D) 87.5% (average of the two values)

Correct Answer: (A) 85% (performance in real-world field conditions)

Solution:

Step 1: Distinguish efficacy from effectiveness. *Efficacy* is how well a vaccine works under **ideal, controlled conditions** such as a well-run randomized controlled trial. *Effectiveness* is how well it works under **real-world, routine field conditions** in the actual population programme.

Step 2: Map the numbers. The 90% comes from the controlled clinical trial → that is the **efficacy**. The 85% comes from routine community use → that is the **effectiveness**.

Step 3: Why effectiveness is usually lower. Real-world use introduces cold-chain breaks, missed/late doses, a broader and less healthy population, and variable circulating strains — all of which reduce the observed benefit below the trial efficacy.

Step 4: Why the others are wrong. 90% is the efficacy (trial), not effectiveness. 5% is merely the arithmetic gap between the two. 87.5% is a meaningless average; effectiveness is a directly measured field value, not an average of efficacy and itself.

Key fact: Effectiveness = real-world field performance = **85%** (efficacy = 90% is the trial value).

Quick Tip: Effectiveness = real-world/field; efficacy = ideal trial.

119. An influenza vaccine is evaluated in a well-conducted randomized controlled trial and again during routine community use. The trial value is 90% and the field value is 85%. Which value represents the **efficacy** of the vaccine?

- (A) 90% (performance in the ideal trial setting)
- (B) 85% (performance in real-world field conditions)
- (C) 5% (difference between the two values)
- (D) 87.5% (average of the two values)

Correct Answer: (A) 90% (performance in the ideal trial setting)

Solution:

Step 1: Define efficacy. Vaccine *efficacy* is the proportionate reduction in disease among the vaccinated group under **ideal, controlled conditions** — classically measured in a randomized controlled trial.

Step 2: Define effectiveness for contrast. *Effectiveness* is the performance under **routine field conditions** in the real population programme.

Step 3: Map the numbers. The 90% is derived from the randomized controlled trial → that is the **efficacy**. The 85% is the routine/community value → that is the effectiveness.

Step 4: Why efficacy is the higher value. Trials enforce strict dosing schedules, a controlled cold chain, and a selected healthier study population, so the measured protection is at its maximum. Field use loses some of this benefit, so effectiveness (85%) is usually lower than efficacy (90%).

Step 5: Why the others are wrong. 85% is the effectiveness, not efficacy. 5% is merely the gap; 87.5% is a meaningless average. Efficacy is the directly measured trial value.

Key fact: Efficacy = ideal trial performance = **90%**.

Quick Tip: Efficacy = ideal RCT value (the higher one, 90%).

120. Regarding the pattern of coronary blood flow, how does flow in the right coronary artery (RCA) differ from that in the left coronary artery (LCA) during the cardiac cycle?

- (A) RCA receives appreciable flow in **both** systole and diastole (biphasic), whereas LCA flow occurs predominantly in diastole
- (B) RCA flow occurs almost entirely in diastole, while LCA flow is biphasic
- (C) Both RCA and LCA are perfused only during systole
- (D) Both RCA and LCA are perfused only during diastole

Correct Answer: (A) RCA receives appreciable flow in **both** systole and diastole (biphasic), whereas LCA flow occurs predominantly in diastole

Solution:

Step 1: Understand why coronary flow is phasic. During systole the contracting myocardium compresses the intramural coronary vessels, throttling flow; in diastole the muscle relaxes and flow resumes. The degree of systolic throttling depends on the intramural (chamber) pressure each artery's bed is exposed to.

Step 2: Left coronary artery. The LCA supplies the thick, high-pressure **left ventricle**. During systole the LV develops very high wall tension (~120 mmHg), which strongly compresses the vessels and nearly abolishes left coronary inflow. Hence LCA flow is **predominantly diastolic**.

Step 3: Right coronary artery. The RCA mainly supplies the thin-walled **right ventricle**, whose systolic pressure is low (~25 mmHg). This generates little compressive force, so the RCA continues to receive substantial flow during systole as well as diastole — giving a **biphasic** pattern (flow in both systole and diastole).

Step 4: Match the recall key. The annotation “d + s” (diastole + systole) refers to

the RCA receiving flow in both phases due to the low RV pressure — exactly the biphasic pattern.

Step 5: Why the others are wrong. It is the LCA (not RCA) that is mainly diastolic, so option 2 is reversed. Neither artery is perfused exclusively in systole or exclusively in diastole, eliminating options 3 and 4.

Key fact: RCA flow is biphasic (systole + diastole) because RV pressure is low, whereas LCA flow is mainly diastolic because high LV systolic pressure compresses its vessels.

Quick Tip: Low RV pressure lets the RCA flow in systole too — biphasic; LCA is mainly diastolic.

121. An ECG shows ST-segment elevation in leads II, III and aVF in a patient presenting with acute chest pain. Which coronary artery is most likely occluded?

- (A) Right coronary artery (RCA)
- (B) Left anterior descending artery (LAD)
- (C) Left circumflex artery (LCx)
- (D) Left main coronary artery

Correct Answer: (A) Right coronary artery (RCA)

Solution:

Step 1: Localise the infarct from the ECG leads. Leads II, III and aVF are the **inferior leads**, so ST elevation in these leads indicates an acute **inferior wall myocardial infarction**.

Step 2: Correlate the inferior wall with its arterial supply. In about 80-90% of people the circulation is **right dominant**, meaning the posterior descending artery (PDA) arises from the right coronary artery. The RCA therefore supplies the inferior wall of the left ventricle (and usually the SA and AV nodes).

Step 3: Choose the answer. Occlusion of the **RCA** produces inferior wall MI (II, III, aVF), and may be accompanied by bradyarrhythmias/AV block due to nodal ischaemia and by right ventricular infarction.

Step 4: Why the others are wrong - the **LAD** supplies the anterior wall and septum (V1-V4 changes); the **LCx** supplies the lateral wall (I, aVL, V5-V6) and the inferior wall only in left-dominant hearts; **left main** occlusion causes extensive anterolateral involvement, not isolated inferior changes.

Key fact: Inferior wall MI (II, III, aVF) = RCA occlusion in a right-dominant circulation.

Quick Tip: Leads II, III, aVF point to the inferior wall - think dominant artery.



122. A patient with long-standing rheumatoid arthritis develops nephrotic-range proteinuria. Renal biopsy with Congo red stain shows apple-green birefringence under polarised light. Which type of amyloid protein is most likely deposited?

- (A) AA (serum amyloid A-derived) amyloid
- (B) AL (immunoglobulin light chain) amyloid
- (C) Abeta2-microglobulin amyloid
- (D) ATTR (transthyretin) amyloid

Correct Answer: (A) AA (serum amyloid A-derived) amyloid

Solution:

Step 1: Identify the clinical setting. The amyloidosis here is secondary (reactive) to a **chronic inflammatory disease** - rheumatoid arthritis. Other classic causes are chronic infections (TB, osteomyelitis, bronchiectasis), IBD and familial Mediterranean fever.

Step 2: Link inflammation to the precursor protein. In chronic inflammation the

liver produces **serum amyloid A (SAA)** as an acute-phase reactant. Persistent elevation leads to its proteolytic processing and deposition as **AA amyloid**.

Step 3: Select the answer. Chronic inflammation to **AA amyloidosis** (secondary/reactive systemic amyloidosis), which characteristically affects the kidneys.

Step 4: Why the others are wrong - **AL** amyloid derives from immunoglobulin light chains and is seen in plasma cell dyscrasias/multiple myeloma (primary amyloidosis); **Abeta2-microglobulin** accumulates in patients on long-term haemodialysis; **ATTR** is from transthyretin (senile cardiac or hereditary amyloidosis).

Key fact: Chronic inflammation produces SAA to AA amyloid (secondary amyloidosis).

Quick Tip: Acute-phase reactant SAA is the precursor in reactive amyloidosis.



123. The plasma drug-concentration versus time curve shown declines such that the half-life remains constant regardless of the starting concentration, and a constant fraction of drug is eliminated per unit time. This pattern best represents which type of elimination kinetics?

- (A) First-order kinetics
- (B) Zero-order kinetics
- (C) Michaelis-Menten (saturation) kinetics at saturating dose
- (D) Mixed-order kinetics

Correct Answer: (A) First-order kinetics

Solution:

Step 1: Read the curve. A semilog or simple decay curve in which a **constant fraction** of the drug is removed per unit time, and the **half-life ($t_{1/2}$) stays the**

same at high and low concentrations, is the hallmark of **first-order kinetics**.

Step 2: Apply the concept. In first-order elimination the rate of elimination is *proportional* to the plasma concentration: as concentration falls, the absolute amount eliminated per unit time also falls, but the proportion (and hence $t_{1/2}$) is fixed. Mathematically, $C = C_0 e^{-kt}$ and $t_{1/2} = 0.693/k$, independent of C_0 .

Step 3: Choose the answer. A constant $t_{1/2}$ with concentration-proportional elimination = **first-order kinetics**, which applies to most drugs at therapeutic doses.

Step 4: Why the others are wrong - **zero-order** kinetics removes a constant *amount* per unit time (straight-line linear decline, saturated enzymes, e.g. ethanol, phenytoin, aspirin at high dose) and has no fixed $t_{1/2}$; **Michaelis-Menten at saturating dose** behaves as zero-order; **mixed-order** shifts from zero- to first-order only as concentration falls below saturation.

Key fact: Constant $t_{1/2}$ + concentration-proportional elimination = first-order kinetics (exponential decay).

Quick Tip: Constant half-life regardless of dose to exponential, concentration-proportional elimination.

124. In the Ann Arbor staging of Hodgkin lymphoma, a mediastinal mass is designated “bulky” (the X modifier) when its maximum width exceeds what proportion of the intrathoracic diameter on chest radiograph?

- (A) Greater than one-third ($>1/3$) of the maximum intrathoracic diameter
- (B) Greater than one-half ($>1/2$) of the maximum intrathoracic diameter
- (C) Greater than one-quarter ($>1/4$) of the maximum intrathoracic diameter
- (D) Greater than two-thirds ($>2/3$) of the maximum intrathoracic diameter

Correct Answer: (A) Greater than one-third ($>1/3$) of the maximum intrathoracic diameter

Solution:

Step 1: Recall the staging modifier. In Hodgkin lymphoma, the suffix **“X”** appended to the Ann Arbor stage denotes **bulky disease**, which carries prognostic and treatment significance (often requires combined chemo-radiotherapy).

Step 2: Apply the definition for a mediastinal mass. A mediastinal mass is called bulky when its maximal width is **$>1/3$ (greater than one-third) of the maximum intrathoracic transverse diameter** on an erect PA chest radiograph (at the level of T5-T6).

Step 3: Choose the answer. The threshold is **$>1/3$ of the intrathoracic diameter** (some references also use a single nodal mass ≥ 10 cm as bulky).

Step 4: Why the others are wrong - one-half, one-quarter and two-thirds are not the accepted radiographic cut-offs; the conventional and most widely tested value for mediastinal bulk is one-third.

Key fact: Bulky (X) mediastinal mass in Hodgkin lymphoma = width $>1/3$ of the intrathoracic diameter on chest X-ray.

Quick Tip: The X modifier for a mediastinal mass uses a one-third radiographic ratio.

125. A child develops cola-coloured urine, periorbital oedema and hypertension two weeks after a sore throat. Urine microscopy shows red blood cell casts. On electron microscopy of the renal biopsy, which finding is characteristic of this condition?

- (A) Subepithelial electron-dense "humps";
- (B) Subendothelial deposits with "tram-track"; basement membrane splitting
- (C) Diffuse effacement of podocyte foot processes with no deposits
- (D) Linear ribbon-like deposits along the glomerular basement membrane

Correct Answer: (A) Subepithelial electron-dense "humps";

Solution:

Step 1: Identify the disease. A child with cola-coloured urine, oedema and hypertension **1-2 weeks after a streptococcal infection**, with **RBC casts** (a nephritic picture), has **post-streptococcal glomerulonephritis (PSGN)**.

Step 2: Recall the ultrastructure. PSGN is an immune-complex disease. On electron microscopy the deposits sit on the epithelial (outer) side of the glomerular basement membrane as discrete **subepithelial electron-dense "humps"**. Light microscopy shows diffuse proliferative GN and immunofluorescence shows a granular "starry-sky" IgG and C3 pattern.

Step 3: Choose the answer. The characteristic EM finding is **subepithelial humps**.

Step 4: Why the others are wrong - **subendothelial deposits with tram-track** splitting suggest membranoproliferative GN; **diffuse foot-process effacement with no deposits** is minimal change disease (nephrotic, not nephritic); **linear GBM deposits** indicate anti-GBM (Goodpasture) disease.

Key fact: PSGN with RBC casts shows subepithelial humps on EM.

Quick Tip: Post-strep nephritic syndrome - think where the immune deposits sit on EM.

126. Transplacental transfer of maternal anti-Ro (SS-A) and anti-La (SS-B) antibodies is most characteristically associated with which neonatal condition?

- (A) Neonatal lupus erythematosus (with congenital complete heart block)
- (B) Neonatal myasthenia gravis
- (C) Haemolytic disease of the newborn
- (D) Neonatal Graves's (thyrotoxicosis)

Correct Answer: (A) Neonatal lupus erythematosus (with congenital complete heart block)

Solution:

Step 1: Identify the antibodies. **Anti-Ro (SS-A)** and **anti-La (SS-B)** are IgG autoantibodies found in mothers with SLE or Sjögren syndrome (sometimes asymptomatic). Being IgG, they cross the placenta.

Step 2: Link to the neonatal effect. These antibodies cause **neonatal lupus erythematosus**, whose hallmarks are an annular/photosensitive rash (transient, resolves as maternal antibody clears) and the serious, often **permanent congenital complete heart block** from immune-mediated damage to the developing AV node/conduction system. Cytopenias and hepatitis may also occur.

Step 3: Choose the answer. Anti-Ro/anti-La to **neonatal lupus** with congenital heart block.

Step 4: Why the others are wrong - **neonatal myasthenia** is from maternal anti-acetylcholine-receptor antibodies; **haemolytic disease of the newborn** is from anti-Rh(D)/blood group antibodies; **neonatal Graves** is from maternal TSH-receptor stimulating antibodies.

Key fact: Maternal anti-Ro/anti-La to neonatal lupus (rash + congenital complete heart block).

Quick Tip: SS-A/SS-B antibodies crossing the placenta damage the fetal conduction



127. A histology slide of myocardium is taken from a patient who died 1-3 days after an acute myocardial infarction. Which of the following cellular changes is MOST characteristic of this time interval?

- (A) Dense neutrophilic (polymorphonuclear) infiltration of necrotic myocardium
- (B) Coagulative necrosis with wavy fibres and contraction bands only
- (C) Macrophage infiltration with phagocytosis of dead myocytes
- (D) Dense collagenous scar with few cells

Correct Answer: (A) Dense neutrophilic (polymorphonuclear) infiltration of necrotic myocardium

Solution:

Step 1: Recall the timeline of histological changes after MI.

The microscopic evolution of a myocardial infarct follows a well-defined sequence. In the first 0-4 hours there are usually no light-microscopic changes. From 4-12 hours, early coagulative necrosis, oedema and haemorrhage begin, and wavy fibres and contraction bands may appear. From 12-24 hours, coagulative necrosis becomes prominent with marked nuclear changes.

Step 2: Identify the 1-3 day window.

Between roughly 1 and 3 days (24-72 hours), there is established coagulative necrosis accompanied by a brisk acute inflammatory response - the necrotic myocardium is heavily infiltrated by **neutrophils (polymorphonuclear leukocytes)**. This is the hallmark of the slide shown.

Step 3: Why the other options are wrong.

Option 2 (wavy fibres/contraction bands alone) is an earlier finding (4-24 hours). Option 3 (macrophage infiltration clearing debris) typically peaks at day 3-7 onward, after the neutrophilic phase wanes. Option 4 (dense collagenous scar) is the late healed phase, occurring after about 2-8 weeks.

Key fact: Neutrophilic infiltration of necrotic myocardium is the characteristic histology of a 1-3 day old myocardial infarct.

Quick Tip: Acute inflammation dominates the first few days - think which white cell arrives first.



128. Hand-eye coordination, integration of visual and somatosensory information, and spatial awareness are mainly functions of which lobe of the cerebral cortex?

- (A) Parietal lobe
- (B) Frontal lobe
- (C) Occipital lobe
- (D) Temporal lobe

Correct Answer: (A) Parietal lobe

Solution:

Step 1: Understand the functional question.

Hand-eye coordination requires the brain to combine incoming visual information with proprioceptive/somatosensory information about limb position and to construct a spatial map of the body in relation to objects. The lobe that integrates these multiple sensory streams is the key.

Step 2: Localise the function.

The **parietal lobe** contains the primary somatosensory cortex (postcentral gyrus) and large posterior parietal association areas. The posterior parietal cortex integrates visual and somatosensory inputs to guide reaching, grasping and visually-directed movement, and mediates spatial awareness. Hence hand-eye coordination is principally a parietal function.

Step 3: Why the other options are wrong.

The frontal lobe executes motor commands (primary motor cortex) and higher

executive control but is not the main integrator of visuospatial input. The occipital lobe processes primary vision only. The temporal lobe handles hearing, memory and object recognition (the "what" stream), not visuomotor spatial guidance.

Key fact: The posterior parietal cortex integrates visual and somatosensory data for hand-eye coordination and spatial awareness; lesions here cause apraxia and neglect.

Quick Tip: Which lobe houses somatosensory cortex and posterior visuospatial association areas?

129. The inguinal ligament (Poupart's ligament), forming the floor of the inguinal canal, is a modification of the lower border of the aponeurosis of which muscle?

- (A) External oblique muscle of the abdomen
- (B) Internal oblique muscle of the abdomen
- (C) Transversus abdominis muscle
- (D) Rectus abdominis muscle

Correct Answer: (A) External oblique muscle of the abdomen

Solution:

Step 1: Define the inguinal ligament.

The inguinal (Poupart's) ligament is a thickened, in-rolled band stretching between the anterior superior iliac spine (ASIS) laterally and the pubic tubercle medially. It forms the floor of the inguinal canal.

Step 2: Identify its origin.

The inguinal ligament is the folded-under (rolled-in) **lower free border of the aponeurosis of the external oblique muscle**. As the external oblique aponeurosis reaches its lower margin between the ASIS and pubic tubercle, it

curves backward and upward on itself to create this ligament.

Step 3: Related derivatives and why other options are wrong.

From the external oblique aponeurosis are also derived the lacunar ligament, pectineal ligament and reflected inguinal ligament. The internal oblique and transversus abdominis form the conjoint tendon (falx inguinalis) and contribute to the roof/cremaster, not the inguinal ligament itself. The rectus abdominis is enclosed in the rectus sheath and does not form this ligament.

Key fact: The inguinal ligament is a modification of the lower border of the external oblique aponeurosis.

Quick Tip: The most superficial of the three flat abdominal muscles rolls under to form this ligament.



130. An inhaled foreign body more commonly lodges in the right main bronchus than the left. Which set of features of the right main bronchus BEST explains this tendency?

- (A) It is wider, shorter and more vertical (in line with the trachea), and the right lung has three lobes
- (B) It is narrower, longer and more horizontal, and the right lung has two lobes
- (C) It is wider but more horizontal, and the right lung has two lobes
- (D) It is narrower and more vertical, and the right lung has three lobes

Correct Answer: (A) It is wider, shorter and more vertical (in line with the trachea), and the right lung has three lobes

Solution:

Step 1: Compare the two main bronchi.

The right main (principal) bronchus differs from the left in three key ways: it is **wider** in calibre, **shorter** in length, and runs **more vertically** - that is, it is more nearly in a straight line with the trachea.

Step 2: Relate this to foreign body aspiration.

Because the right bronchus is wider and almost continues the line of the trachea, an aspirated foreign body or aspirated material tends to fall into the right bronchus and then into the right lung more often than the left. The left bronchus is narrower, longer and more horizontal, so it diverts material less readily.

Step 3: Lobar anatomy.

The right lung has **three lobes** (upper, middle, lower) separated by oblique and horizontal fissures, whereas the left lung has two lobes (upper and lower) plus the lingula. Aspirated material in the upright patient classically reaches the posterior or basal segments of the right lower lobe.

Key fact: The right main bronchus is wider, shorter, more vertical (straighter from the trachea), and the right lung has three lobes - explaining preferential right-sided aspiration.

Quick Tip: Which bronchus most nearly continues the straight line of the trachea?

131. Which of the following carbohydrates is a NON-reducing sugar and therefore gives a NEGATIVE Benedict's (and Fehling's) test?

- (A) Sucrose
- (B) Maltose
- (C) Lactose
- (D) Glucose

Correct Answer: (A) Sucrose

Solution:

Step 1: Understand the basis of Benedict's test.

Benedict's test detects reducing sugars - carbohydrates with a free aldehyde or ketone group (a free anomeric carbon) that can reduce cupric (Cu^{2+}) ions to cuprous oxide, giving a brick-red precipitate. A sugar with no free

reducing group gives a NEGATIVE test.

Step 2: Examine each sugar.

Sucrose is glucose linked to fructose through an alpha-1 to 2 glycosidic bond that joins both anomeric carbons. Because both reducing groups are tied up in the glycosidic linkage, sucrose has no free reducing group and is a **non-reducing sugar - Benedict negative**.

Step 3: Why the others are positive.

Maltose (glucose-alpha-1,4-glucose) and lactose (galactose-beta-1,4-glucose) each retain one free anomeric carbon and are reducing sugars. Glucose is a free monosaccharide with an aldehyde group and is strongly reducing. All three give a positive Benedict's test.

Key fact: Sucrose is the classic non-reducing sugar (both anomeric carbons engaged) and gives a negative Benedict's test, while the rest reduce.

Quick Tip: Which disaccharide locks BOTH of its anomeric carbons in the glycosidic bond?



132. Pretomanid, a nitroimidazole used in the BPaL/BPaLM regimen for drug-resistant tuberculosis, acts mainly by which mechanism?

- (A) Inhibition of mycolic acid synthesis (and generation of reactive nitrogen species), impairing cell-wall formation and anaerobic respiration
- (B) Inhibition of the RNA polymerase beta-subunit (rpoB)
- (C) Inhibition of mycobacterial ATP synthase
- (D) Inhibition of DNA gyrase (topoisomerase II)

Correct Answer: (A) Inhibition of mycolic acid synthesis (and generation of reactive nitrogen species), impairing cell-wall formation and anaerobic respiration

Solution:

Step 1: Classify the drug.

Pretomanid (PA-824) is a **nitroimidazo-oxazine** prodrug, structurally related to delamanid. It is activated inside *Mycobacterium tuberculosis* by the deazaflavin (F420)-dependent nitroreductase (Ddn), which requires the cofactor F420 generated by the *fgd1/fbiABC* pathway.

Step 2: Mechanism of action.

Once activated, pretomanid has a dual action. Against actively replicating (aerobic) bacilli it **inhibits the synthesis of mycolic acids**, key components of the mycobacterial cell wall, by blocking oxidation of hydroxymycolate to ketomycolate. Against non-replicating (anaerobic/dormant) bacilli, its reductive activation releases **reactive nitrogen species, including nitric oxide**, which poison the respiratory chain and exert anaerobic killing.

Step 3: Why the other options are wrong.

RNA polymerase (*rpoB*) inhibition is the mechanism of rifampicin. ATP synthase inhibition is the mechanism of bedaquiline (the "B" in BPaL). DNA gyrase inhibition is the mechanism of fluoroquinolones such as moxifloxacin/levofloxacin.

Key fact: Pretomanid inhibits mycolic acid/cell-wall synthesis and releases reactive nitrogen species, killing both replicating and dormant *M. tuberculosis*.

Quick Tip: A nitroimidazole prodrug activated by Ddn that blocks cell-wall (mycolic acid) synthesis.

133. What is the mode of infection (route of entry into the human host) of *Necator americanus* (New World hookworm)?

- (A) Skin penetration by filariform larvae
- (B) Ingestion of embryonated eggs
- (C) Bite of an infected female sandfly
- (D) Ingestion of undercooked pork containing cysts

Correct Answer: (A) Skin penetration by filariform larvae

Solution:

Step 1: Identify the parasite. *Necator americanus* is one of the two common human hookworms (the other being *Ancylostoma duodenale*). Both belong to the nematodes (roundworms) and are major causes of iron-deficiency anaemia in the tropics.

Step 2: Recall the life cycle and infective stage. Hookworm eggs passed in faeces hatch in warm, moist soil to release rhabditiform larvae, which mature into the infective **filariform (L3) larvae**. These filariform larvae actively **penetrate intact skin** (classically the web of the toes/feet of barefoot individuals).

Step 3: Trace the migration. After skin penetration, the larvae enter venules, travel to the lungs, ascend the bronchial tree, are swallowed, and mature into adults in the small intestine where they attach and feed on blood. For *Necator*, **skin penetration is the exclusive route** (unlike *Ancylostoma*, which can also infect by oral ingestion of larvae).

Step 4: Why the other options are wrong. Ingestion of embryonated eggs is the route for *Ascaris*, *Trichuris* and *Enterobius*, not hookworm. The sandfly bite transmits *Leishmania*. Undercooked pork with cysts causes *Taenia solium*/*Trichinella* infection. None of these apply to *Necator*.

Key fact: *Necator americanus* infects man almost exclusively by **filariform larvae penetrating the skin**.

Quick Tip: Think barefoot in soil: hookworm filariform larvae bore through skin.

134. A post-splenectomy patient is at greatest risk of overwhelming sepsis from which organism?

- (A) *Streptococcus pneumoniae*
- (B) *Staphylococcus aureus*
- (C) *Escherichia coli*
- (D) *Pseudomonas aeruginosa*

Correct Answer: (A) *Streptococcus pneumoniae*

Solution:

Step 1: Understand the immune role of the spleen. The spleen is the principal site for clearing **encapsulated bacteria** from the blood and for mounting opsonising IgM/complement responses. Loss of the spleen (asplenia) markedly impairs clearance of capsular organisms.

Step 2: Recall the encapsulated organisms. The classic asplenia/OPSI triad is *Streptococcus pneumoniae*, *Haemophilus influenzae* type b, and *Neisseria meningitidis*. Among these, ***Streptococcus pneumoniae* is by far the most common cause** of overwhelming post-splenectomy infection (OPSI), responsible for roughly 50-90% of cases.

Step 3: Clinical relevance. Because of this risk, asplenic patients receive **pneumococcal, Hib, and meningococcal vaccination** (ideally before elective splenectomy) plus often prophylactic penicillin.

Step 4: Why the others are wrong. *S. aureus*, *E. coli* and *Pseudomonas* are not predominantly capsule-dependent for splenic clearance and are not the signature OPSI pathogens; they cause sepsis through different, non-asplenia-specific mechanisms.

Key fact: The commonest cause of overwhelming post-splenectomy sepsis is ***Streptococcus pneumoniae***.

Quick Tip: No spleen = no clearance of encapsulated bugs; the top one is pneumococcus.

135. Which class of antibiotics characteristically shows **concentration-dependent killing**?

- (A) Aminoglycosides
- (B) Beta-lactams (penicillins)
- (C) Glycopeptides (vancomycin)
- (D) Macrolides (erythromycin)

Correct Answer: (A) Aminoglycosides

Solution:

Step 1: Define the pharmacodynamic patterns. Antibacterial killing follows two broad patterns. In **concentration-dependent killing**, the rate and extent of bacterial kill rise as drug concentration increases well above the MIC; the predictive parameters are **peak/MIC** ($C_{\max}/\text{MIC}$) and **AUC/MIC**. In **time-dependent killing**, kill plateaus once concentration exceeds about 4 x MIC, and efficacy depends on the **time above MIC** ($T > \text{MIC}$).

Step 2: Classify aminoglycosides. **Aminoglycosides** (gentamicin, amikacin, tobramycin) are the prototype **concentration-dependent** bactericidal drugs and also have a prolonged **post-antibiotic effect**. This is the rationale for **once-daily (extended-interval) dosing** - a high peak maximises killing while the trough falls low enough to limit nephro- and ototoxicity.

Step 3: Why the others are wrong. Beta-lactams and glycopeptides (vancomycin) are **time-dependent**; their efficacy depends on $T > \text{MIC}$ (vancomycin is best tracked by AUC/MIC but is not concentration-dependent in the aminoglycoside sense). Macrolides are largely time-dependent/bacteriostatic.

Key fact: Aminoglycosides (with fluoroquinolones, metronidazole and daptomycin) are classic **concentration-dependent** killers, justifying once-daily dosing.

Quick Tip: Higher peak = more kill, plus a long post-antibiotic effect: that means aminoglycosides.

136. The posterior cricoarytenoid (PCA) muscle of the larynx is the only muscle that performs which action on the vocal cords?

- (A) Abduction (opening) of the vocal cords
- (B) Adduction (closing) of the vocal cords
- (C) Tensing (lengthening) of the vocal cords
- (D) Relaxation (shortening) of the vocal cords

Correct Answer: (A) Abduction (opening) of the vocal cords

Solution:

Step 1: Recall the intrinsic laryngeal muscles. The intrinsic muscles of the larynx control the position and tension of the vocal cords and are (except the interarytenoid) supplied by the recurrent laryngeal nerve.

Step 2: Action of the posterior cricoarytenoid (PCA). The **PCA is the sole abductor of the vocal cords** - it rotates the arytenoid cartilages so as to **open the rima glottidis**, which is essential for inspiration. It is sometimes called the “safety muscle of the larynx” because it keeps the airway open.

Step 3: Contrast with other muscles. The lateral cricoarytenoid, interarytenoid (transverse/oblique) and thyroarytenoid are **adductors** (close the cords). The **cricothyroid tenses/lengthens** the cords (supplied by the external branch of the superior laryngeal nerve), and the thyroarytenoid/vocalis relaxes/shortens them.

Step 4: Clinical correlation. Because the PCA is the only abductor, bilateral recurrent laryngeal nerve palsy leaves the cords near the midline and can cause **airway obstruction and stridor**.

Key fact: The **posterior cricoarytenoid** is the **only abductor** of the vocal cords.

Quick Tip: Only one muscle opens the airway at the larynx - the posterior cricoarytenoid.

137. Sudden sensorineural hearing loss (SSNHL) is defined as ≥ 30 dB of SNHL over at least three contiguous frequencies developing within 3 days. What is the mainstay of treatment?

- (A) Corticosteroids (systemic, with intratympanic steroids as salvage)
- (B) Oral antiviral therapy alone
- (C) Immediate cochlear implantation
- (D) Broad-spectrum intravenous antibiotics

Correct Answer: (A) Corticosteroids (systemic, with intratympanic steroids as salvage)

Solution:

Step 1: Define the condition. Sudden sensorineural hearing loss (SSNHL) is an otologic emergency defined as a loss of ≥ 30 dB across at least three contiguous frequencies occurring within 72 hours (3 days). Most cases are idiopathic, with proposed viral, vascular and inflammatory mechanisms.

Step 2: Identify the first-line treatment. The mainstay is **corticosteroids**. **Systemic (oral) steroids** such as prednisolone are started as early as possible, since outcomes are best when therapy begins within the first 2 weeks.

Step 3: Salvage / adjunctive therapy. If there is incomplete recovery or contraindications to systemic steroids (e.g., diabetes), **intratympanic steroid injection** is used - either as primary therapy or as salvage after failed oral steroids. Hyperbaric oxygen may be considered as an adjunct in selected cases.

Step 4: Why the others are wrong. Routine antivirals and antibiotics have not shown benefit in idiopathic SSNHL. Cochlear implantation is reserved for permanent profound bilateral loss, not initial management.

Key fact: First-line treatment of SSNHL is **corticosteroids (systemic first, intratympanic as salvage)**.

Quick Tip: Sudden SNHL is an emergency - reach for steroids, oral first then intratympanic.



138. Which is the most characteristic cardiovascular effect of the inhalational anaesthetic **halothane**?

- (A) Dose-dependent fall in cardiac output and blood pressure (often a 20-30 mm Hg drop) from myocardial depression
- (B) Increase in cardiac output with reflex tachycardia
- (C) Rise in systemic vascular resistance with hypertension
- (D) No measurable effect on cardiac output or blood pressure

Correct Answer: (A) Dose-dependent fall in cardiac output and blood pressure (often a 20-30 mm Hg drop) from myocardial depression

Solution:

Step 1: Recall the pharmacology of halothane. Halothane is a halogenated inhalational anaesthetic that is a potent **direct myocardial depressant**.

Step 2: Cardiovascular effects. It produces a **dose-dependent reduction in myocardial contractility and cardiac output**, leading to a fall in mean arterial pressure (commonly on the order of a **20-30 mm Hg drop**) that is roughly **proportional to the depth of anaesthesia**. Unlike many agents it does **not** cause a compensatory tachycardia - heart rate tends to stay the same or fall (it blunts the baroreceptor reflex).

Step 3: Arrhythmogenic potential. Halothane also **sensitises the myocardium to catecholamines**, predisposing to ventricular arrhythmias, especially with exogenous adrenaline or hypercarbia.

Step 4: Why the others are wrong. Halothane decreases (not increases) cardiac output, does not raise SVR/cause hypertension, and certainly is not haemodynamically neutral - its hypotensive, cardiodepressant effect is one of its defining features.

Key fact: Halothane causes a **dose-dependent fall in cardiac output and blood pressure (a 20-30 mm Hg drop)** through direct myocardial depression.

Quick Tip: Halothane is a myocardial depressant - expect cardiac output and BP to fall with depth.



139. A chronic alcoholic with thiamine deficiency develops a memory disturbance in which he is unable to form new memories, though older memories are relatively preserved. This Korsakoff syndrome is characterised by which type of memory deficit?

- (A) Anterograde amnesia
- (B) Retrograde amnesia
- (C) Transient global amnesia
- (D) Dissociative amnesia

Correct Answer: (A) Anterograde amnesia

Solution:

Step 1: Korsakoff syndrome is a chronic amnestic disorder caused by thiamine (vitamin B1) deficiency, classically seen in chronic alcoholics and often following an episode of Wernicke encephalopathy. The lesion involves the mammillary bodies and the dorsomedial nucleus of the thalamus.

Step 2: The hallmark feature is **anterograde amnesia** - the inability to form and

lay down new memories after the onset of the illness. The patient cannot retain new information for more than a few minutes.

Step 3: Associated features include confabulation (filling memory gaps with fabricated accounts), variable retrograde amnesia, and relatively preserved immediate recall and procedural memory.

Step 4: Why the others are wrong - pure retrograde amnesia (loss of past memories) is not the defining feature of Korsakoff; transient global amnesia is a self-limited episodic disorder; dissociative amnesia is a psychogenic, stress-related loss of autobiographical memory without an organic thiamine basis.

Key fact: Korsakoff syndrome to thiamine deficiency to predominant **anterograde amnesia** with confabulation.

Quick Tip: Think of the inability to form NEW memories after a thiamine-deficiency insult.

140. A firearm wound shows an entry with an abraded collar and a grease (dirt) collar, but there is NO tattooing (no gunpowder stippling), no soot blackening, and no burning around the wound. What is the range of fire?

- (A) Distant range
- (B) Contact range
- (C) Close range
- (D) Near (intermediate) range

Correct Answer: (A) Distant range

Solution:

Step 1: The range of a gunshot wound is judged by the marks produced by the products that leave the muzzle - flame (burning), smoke/soot (blackening), and unburnt/partially burnt powder grains (tattooing or stippling) - in addition to the

bullet itself.

Step 2: Here the wound shows only an **abrasion collar** (AC, produced by the bullet indenting and grazing skin on entry) and a **grease/dirt collar** (GC, wiped off dirt and lubricant from the bullet). Both are produced by the bullet at ANY range and carry no range information by themselves.

Step 3: The absence of tattooing, soot, and burning is the decisive finding. Powder tattooing is seen up to roughly 60 cm (about 2 feet) and soot up to roughly 30 cm; beyond the maximum travel of powder grains, none of these accessory marks reach the skin.

Step 4: Therefore the shot was fired beyond the range at which gunpowder can reach the target - a **distant (far) range** wound, showing only AC and GC.

Step 5: Why the others are wrong - contact wounds show muzzle imprint, soot, and burning; close range shows dense blackening and burning; near/intermediate range shows powder tattooing. None of these features are present here.

Key fact: Only abrasion + grease collar with NO tattooing/soot/burning = **distant range** firearm wound.

Quick Tip: No gunpowder tattooing, no soot, no burn - the powder never reached the skin.

141. A victim of a bomb blast sustains burns to the chest and face caused by the fireball and the heat of the explosion (not by the blast wave, flying fragments, or being thrown against a structure). This burn injury is best classified as which category of blast injury?

- (A) Quaternary blast injury
- (B) Primary blast injury
- (C) Secondary blast injury
- (D) Tertiary blast injury

Correct Answer: (A) Quaternary blast injury

Solution:

Step 1: Blast injuries are classified into four categories based on the mechanism by which the explosion harms the victim.

Step 2: Primary injury is due to the blast (over-pressure) wave acting on gas-containing organs - ear drum rupture, blast lung, and bowel perforation.

Secondary injury is due to flying fragments and debris causing penetrating and lacerating wounds. **Tertiary** injury is due to the body being thrown against a hard surface, causing blunt trauma and fractures.

Step 3: Quaternary injury includes all other explosion-related effects not covered above - burns (flash/flame), inhalation of toxic gases and dust, crush injury, and exacerbation of pre-existing conditions (e.g., asthma, angina).

Step 4: Burns to the chest and face from the heat/fireball of the explosion fall squarely under the **quaternary** category.

Step 5: Why the others are wrong - the pressure wave (primary), fragments (secondary), and displacement of the body (tertiary) did not produce these burns; the thermal effect of the blast did.

Key fact: Blast-related **burns** = **quaternary** blast injury.

Quick Tip: Burns and inhalation effects are the 'everything-else' category of blast trauma.

142. An entry firearm wound demonstrates an abrasion collar and a grease collar but completely lacks gunpowder tattooing (stippling), soot, and singeing of hair. The most appropriate interpretation of the firing distance is:

- (A) Distant range
- (B) Contact (tight) range
- (C) Close range
- (D) Intermediate (near) range

Correct Answer: (A) Distant range

Solution:

Step 1: Firing distance is determined by which muzzle by-products - flame (singeing/burns), soot (blackening), and powder grains (tattooing) - reach the skin, alongside the bullet's own marks.

Step 2: The abrasion collar (bullet indenting skin) and grease collar (lubricant/dirt wiped off the bullet) are produced at every distance and give NO information about range.

Step 3: Here, tattooing, soot, and singeing are all absent. Powder tattooing is generally seen only within about 60 cm and soot within about 30 cm. Their complete absence means the powder grains and smoke did not reach the target.

Step 4: A wound showing only AC and GC with none of the gunpowder/flame effects is therefore a **distant range** gunshot wound, fired beyond the maximum travel of powder.

Step 5: Why the others are wrong - contact wounds show muzzle imprint with cavity soot and burning; close range shows heavy soot and singeing; intermediate range shows powder tattooing. None are seen here.

Key fact: Absence of tattooing/soot/singeing with only AC + GC to **distant range**.

Quick Tip: No stippling, no soot, no singed hair - the muzzle was beyond powder range.



143. A characteristic pattern of multiple radiating, brush-like abrasions resembling a bird's foot ("sparrow-foot" or crow-foot marks) is found on the skin of a deceased. This injury pattern is most characteristic of:

- (A) Motor vehicle (road traffic) accident
- (B) Fall from height onto a flat surface
- (C) Firearm injury
- (D) Electrocution

Correct Answer: (A) Motor vehicle (road traffic) accident

Solution:

Step 1: "Sparrow-foot" (also called crow-foot) marks are a pattern of multiple grouped, branching/radiating abrasions resembling the imprint of a bird's foot.

Step 2: They are produced when the skin is grazed by gravel, road grit, and irregular surfaces, with tangential dragging force. This combination of grazing plus drag is classically seen when a body is dragged or skids across a rough road surface.

Step 3: Hence sparrow-foot marks are typical of **motor vehicle / road traffic accidents**, where the victim is struck, run over, or dragged along the road.

Step 4: Why the others are wrong - a fall onto a flat surface produces simple abrasions or lacerations without the branching brush pattern; firearm injuries produce entry/exit wounds with collars; electrocution produces Joule (electric) burns with blistering, not brush abrasions.

Key fact: Sparrow-foot / brush abrasions to road traffic (vehicular) accident.

Quick Tip: Branching grazing abrasions from being dragged over rough road grit.

144. A patient is brought to the emergency department with pin-point pupils, depressed respiration, and a reduced level of consciousness following suspected opioid (e.g., morphine/heroin) overdose. Which drug is the specific antidote?

- (A) Naloxone
- (B) Flumazenil
- (C) Atropine
- (D) N-acetylcysteine

Correct Answer: (A) Naloxone

Solution:

Step 1: The clinical triad of pin-point (miotic) pupils, respiratory depression, and CNS depression is the classic opioid toxidrome.

Step 2: The specific antidote is **naloxone**, a pure, competitive opioid (mu-receptor) antagonist. Given IV/IM/intranasal, it rapidly reverses respiratory depression and sedation.

Step 3: Because naloxone has a shorter half-life than many opioids, repeated doses or an infusion may be needed to prevent re-narcotisation; it can precipitate acute withdrawal in dependent patients.

Step 4: Why the others are wrong - flumazenil reverses benzodiazepines (GABA-A); atropine is the antidote for organophosphate/cholinergic (muscarinic) poisoning and anticholinesterase effects; N-acetylcysteine is the antidote for paracetamol (acetaminophen) overdose.

Key fact: Opioid overdose antidote = **naloxone** (opioid receptor antagonist).

Quick Tip: Reverses the opioid receptor - restores breathing in heroin/morphine overdose.