

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:

Wilson disease stands apart from the other four conditions because it is the only one with a treatment that genuinely alters and can reverse the disease course. The defect lies in the **ATP7B** copper-transporting ATPase, leading to toxic copper deposition in the liver and the lenticular nuclei. The therapeutic principle is simple: remove and block copper. Chelators such as D-penicillamine and trientine mobilise stored copper for urinary excretion, while oral zinc induces metallothionein in enterocytes to trap dietary copper and shed it in stool. Begun early, this regimen prevents progression and can reverse Kayser-Fleischer rings and neurological signs; transplantation cures the underlying hepatic defect in fulminant cases. Contrast this with the alternatives. PKAN and PLA2G6-associated

neurodegeneration are inherited NBIA syndromes with relentless progression and only symptomatic care for dystonia and spasticity. PML, a JC-virus leukoencephalopathy, responds only to immune reconstitution, not to a specific cure. Hence the unique, disease-modifying option is copper-targeted therapy.

Wilson disease

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

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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:

Systemic sclerosis injures the heart chiefly through its microvasculature. Recurrent vasospasm and intimal proliferation in intramyocardial vessels cause repeated focal ischaemia, and the healing response lays down dense collagen, producing the characteristic patchy myocardial fibrosis. Because this fibrosis is scattered rather than coronary-territory based, it stiffens and weakens the ventricle globally, generating a cardiomyopathy with impaired relaxation, falling ejection fraction, conduction blocks, and ventricular arrhythmias. This myocardial scarring is the defining and most lethal cardiac change in scleroderma. The recalled answer of mitral stenosis is rejected on clinical grounds: stenotic mitral valve disease is essentially rheumatic in origin and is not part of the scleroderma spectrum. Aortic regurgitation points instead to aortic root pathology such as Marfan syndrome, while pericarditis, though it can appear in scleroderma, is

usually silent and minor compared with the myocardial disease. The clinically correct deformity is therefore myocardial fibrosis leading to cardiomyopathy.

Myocardial fibrosis → cardiomyopathy (DCM)

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

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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:

Epithelial cells are stitched together by junctions with distinct jobs, and the question targets the one that seals. The zonula occludens, or tight junction, lies at the very apex of the lateral cell border where the outer leaflets of neighbouring membranes are zipped shut by transmembrane proteins claudin and occludin. This fusion closes the paracellular route, enforces selective permeability, and maintains the apico-basal polarity that lets epithelia act as true barriers. The other choices serve adhesion or communication rather than sealing. Fascia adherens belongs to the intercalated disc and anchors actin filaments in cardiac muscle; the desmosome, or macula adherens, is a rivet-like spot that ties intermediate filaments together for mechanical strength; and the gap junction is a channel array of connexons that allows ions and small molecules to flow between cells. None of these occludes the intercellular cleft, so the unique occluding junction is the zonula occludens.

Zonula occludens (tight junction)

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

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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:

The clues here are the very early onset, the absence of fever and mucosal disease, and the maternal autoimmune history. Together they describe a baby who has received antibodies across the placenta rather than acquired an infection or inherited a fragile skin. In neonatal pemphigus, maternal IgG directed against desmoglein crosses to the fetus and drives intraepidermal acantholysis, producing flaccid blisters and erosions on the face and trunk. Because the antibodies are passively acquired, the condition is transient and resolves as the infant clears maternal immunoglobulin over a few weeks, and the child remains systemically well. The distractors fail specific tests. Staphylococcal scalded skin syndrome is toxin-mediated and presents with a febrile, irritable infant with periorificial crusting and widespread tender peeling, unconnected to maternal disease. Bullous pemphigoid is a disorder of the elderly and is vanishingly rare in newborns. Epidermolysis bullosa is a genetic, trauma-induced blistering disease,

not an antibody-driven one. The picture therefore fits passively transferred pemphigus.

Neonatal pemphigus (transplacental maternal antibodies)

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

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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:

Sorting these poisons by their effect on breathing makes the answer clear. Opioids act on brainstem chemoreceptors and the respiratory rhythm generator to blunt the drive to breathe, which is why apnoea is the usual cause of death in opium poisoning. Barbiturates depress the entire central nervous system in a dose-related fashion, including the medullary respiratory centre, leading to shallow, slow respiration and coma. Datura, rich in atropine and scopolamine, causes an anticholinergic delirium that in severe cases progresses to central depression, coma, and failure of respiratory drive. Strychnine behaves oppositely. By blocking glycine-mediated inhibition in the spinal cord, it unleashes motor neurons and produces explosive generalised convulsions with opisthotonus. It is a stimulant, not a depressant, and it does not suppress the respiratory centre; any breathing failure is mechanical, from tetanic spasm of the chest muscles. Therefore central respiratory depression is the one feature it does not share.

Strychnine

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

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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:

A rectocele is the forward ballooning of the anterior rectal wall into the back wall of the vagina, and defecography is the test used to quantify it during straining and evacuation. The radiologist draws the line of the anticipated anterior rectal contour and measures how far the wall bulges beyond it. This depth is the basis for grading. By the standard scheme, a bulge under 2 cm is small and usually of no clinical consequence, 2 to 4 cm is moderate, and beyond 4 cm is large. The cut-off at which a rectocele is called significant is therefore 2 cm, because at and above this depth patients begin to retain stool in the pouch, strain ineffectively, and resort to manual or vaginal splinting to empty. The larger figures of 5 cm and 7 cm describe big rectoceles but are not the threshold that separates insignificant from significant. The defining limit is two centimetres.

> 2 cm

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

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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:

The laparoscopic preperitoneal approaches (TAPP and TEP) work by placing a wide mesh over the *myopectineal orifice of Fruchaud*, a single weak musculofascial window that gives rise to indirect inguinal, direct inguinal and femoral hernias.

For the mesh to cover all three potential exit points, the surgeon must clear the preperitoneal plane in every direction: medially into the space of Retzius up to the contralateral pubis, laterally into the space of Bogros, superiorly above the arch, and crucially **inferiorly below the inguinal ligament** to expose Cooper's (pectineal) ligament and the femoral canal.

The inferior dissection limit is the critical examined point. To safely visualise the femoral orifice and seat the lower edge of the mesh so it does not curl, the peritoneal flap must be taken down approximately:

$$d \approx 2 \text{ cm below the inguinal ligament}$$

Less than this (0.5 to 1 cm) leaves the femoral defect inadequately covered, the commonest site of recurrence after laparoscopic repair; far more than this (10 cm) needlessly enters the danger zones containing the external iliac vessels and

the genitofemoral / lateral cutaneous nerves.

Minimum dissection below the inguinal ligament = 2 cm

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

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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:

The key to this slide is the type of epithelium covering the distal esophageal mucosa. Normally this is stratified squamous; here it has been converted into a columnar lining studded with ***goblet cells***.

This conversion of one mature differentiated epithelium into another is termed **metaplasia**. In the esophagus, chronic acid and bile reflux (GERD) drives the squamous-to-columnar (intestinal) metaplasia we call **Barrett's esophagus**. The defining feature demanded by pathologists is **specialised intestinal metaplasia** - columnar cells with interspersed goblet cells.

Distinguishing it from the distractors:

- Squamous cell carcinoma to malignant squamous nests, keratin pearls,

intercellular bridges.

- Adenocarcinoma to frankly malignant infiltrating glands, often arising on a background of Barrett's.
- Low-grade dysplasia to superimposed nuclear crowding/hyperchromasia on Barrett mucosa, but the bland goblet-cell metaplasia shown is the underlying lesion, not dysplasia.

Because the slide shows uncomplicated goblet-cell columnar metaplasia, the answer is:

Barrett's esophagus

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

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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:

The trick here is to pick the sign that is present in one entity but absent in the other, rather than a sign shared by both.

Think mechanistically. In **mechanical obstruction** the gut is healthy and keeps trying to push contents past a physical barrier, producing waves of contraction. Clinically this gives **colicky** pain that comes and goes with each peristaltic wave, accompanied by loud, high-pitched, tinkling bowel sounds.

In **paralytic ileus** the smooth muscle itself has stopped working (post-operative, electrolyte disturbance, peritonitis). With no peristalsis there is no cramping - the abdomen distends quietly and bowel sounds are absent.

Testing each option:

- Abdominal distension to both.
- Vomiting to both.
- Tachycardia to both (fluid loss / sepsis).
- Colicky pain to **only mechanical obstruction**, because it requires active peristalsis.

Colicky pain is the differentiating feature

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

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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:

This is a pattern-recognition question anchored on the location and symmetry of the lip defect.

Embryologically the upper lip is built where the paired maxillary prominences meet the fused medial nasal prominences. The line of fusion lies just lateral to the

midline on each side (the philtral columns). A cleft therefore appears **off-midline**, and when fusion fails on both right and left it is a ***bilateral cleft lip***, classically leaving the central premaxilla projecting forward with a foreshortened columella.

Differentiating the options by site and embryology:

- Midline cleft lip to central defect from failed merging of the two medial nasal prominences; linked to holoprosencephaly - rare.
- Cleft palate to gap in the palatal shelves (roof of mouth), not on the visible lip.
- Oblique (Tessier) facial cleft to defect tracking obliquely from lip to orbit - very rare.

Paired, symmetric upper-lip clefts with an isolated premaxilla match only one diagnosis:

Bilateral cleft lip

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

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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:

The examined concept is the precise surgical definition of ***TME*** for rectal carcinoma.

The rectum is wrapped in the mesorectum - a parcel of fat, lymph nodes and

vessels enclosed by the visceral mesorectal fascia. Rectal cancer spreads into this parcel, so curing it requires removing the parcel whole, with its covering fascia uninjured (an intact circumferential resection margin).

Heald's principle is to develop the bloodless areolar plane (the "holy plane") between the mesorectal fascia and the presacral fascia, and to do so with **scissors / diathermy under direct vision** - i.e. *sharp* dissection - lifting out the rectum and its mesorectum **en bloc as one specimen**.

Why the rest fail:

- Removing only peritumoral fat to leaves nodal deposits to recurrence.
- Blunt finger dissection to ruptures the fascial envelope, spills tumour, injures autonomic nerves.
- Internal iliac ligation to irrelevant to mesorectal clearance.

TME = sharp en-bloc excision of the entire mesorectum as one unit

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

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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:

This question asks for the priority sequence in trauma resuscitation when the patient is bleeding catastrophically.

Standard *ATLS* teaches the primary survey as A to B to C to D to E. However, a patient with a torn limb vessel or major external haemorrhage can exsanguinate within minutes - faster than any airway problem will kill them. Battlefield and modern civilian trauma guidelines therefore prefix an extra step:

$$\langle C \rangle \rightarrow A \rightarrow B \rightarrow C \rightarrow D \rightarrow E$$

where the leading $\langle C \rangle$ is **Catastrophic haemorrhage control** - apply direct pressure, a tourniquet, or a haemostatic dressing *first*. Only then do you secure the Airway, support Breathing, restore Circulation, assess Disability (neurology) and complete Exposure.

The other choices either start from the unmodified ABCDE (ignoring the exsanguination) or scramble the order, none of which prioritises stopping the bleeding that is the immediate threat to life.

$\langle C \rangle$ -A-B-C-D-E: control catastrophic bleeding before airway

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

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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:

The Gleason system records the two most common histological architectures of prostate cancer, each scored from 1 (well differentiated) to 5 (poorly differentiated). The total score therefore ranges from 6 to 10. Because raw scores like 7 can arise from either a 3+4 or a 4+3 pattern with different outcomes, the ISUP introduced five prognostic Grade Groups.

Mapping: $3 + 3 = 6 \rightarrow$ Group 1; $3 + 4 = 7 \rightarrow$ Group 2; $4 + 3 = 7 \rightarrow$ Group 3; $4 + 4, 3 + 5, 5 + 3 = 8 \rightarrow$ Group 4; 9 and 10 $\rightarrow$ Group 5.

Here the report is $4 + 4$, summing to 8, with both components being aggressive pattern 4 tissue. This places it squarely in Grade Group 4, signalling high-risk disease.

Gleason $4 + 4 =$ Grade Group 4

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

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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:

Anal fistula surgery is judged largely by continence outcomes, because the tract often crosses sphincter fibres. Modern minimally invasive options were specifically designed to avoid cutting muscle.

LIFT works in the intersphincteric groove, ligating the tract on either side without sacrificing sphincter bulk. **FiLaC** delivers 360° laser energy that shrinks and seals the tract lining while the muscle stays intact. **VAAFT** uses direct endoscopic visualisation to destroy the tract and securely close the internal opening, again without dividing sphincter.

By contrast, **fistulectomy** surgically excises the fistula as a block of tissue; because the tract physically runs through sphincter, removing it typically sacrifices muscle and threatens continence. It is therefore the odd one out.

Fistulectomy is NOT sphincter-preserving

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

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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:

Language development follows predictable checkpoints. A toddler should be saying multiple single words by **18** months and combining two words by **24** months. At **20** months a child limited to one word has clear expressive language delay, which is never something to simply watch.

The clinician's job is to find a cause. The leading and most readily correctable explanation for delayed speech is undetected hearing loss, so audiometry plus a structured speech-language assessment is the logical first investigation. Identifying and treating a hearing deficit early can restore normal language trajectory.

Reassurance, deferral to age **5**, or an outright autism label are all inappropriate: the first two ignore the delay and the third jumps to a diagnosis without excluding hearing impairment, especially when social and motor development are reportedly normal.

Next step = Speech and hearing evaluation

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

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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:

Penetrance answers the question: of everyone who inherits the disease allele, how many actually show the disease? If the answer is less than 100%, the trait has incomplete (reduced) penetrance.

Practically, this produces a carrier who possesses the mutation yet looks completely healthy. In a family tree such a person sits between an affected ancestor and an affected descendant, so the condition seems to bypass a generation - affected individuals show up in alternate generations.

The other choices describe unrelated phenomena: a single affected sex points to sex-linked or sex-limited inheritance; a family with nobody affected simply does not carry the trait; and a pedigree where everyone in every generation is affected reflects fully penetrant dominant transmission. Note also that anticipation (progressively earlier/severe onset) is a separate idea and should not be confused here.

Incomplete penetrance → trait skips a generation

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:

The cause of meningitis is strongly age-dependent. In the first weeks of life the offending bacteria come mainly from the mother's vaginal and gastrointestinal flora, giving the well-known trio of Group B *Streptococcus*, *E. coli* and *Listeria monocytogenes*.

A 6-week-old still falls within this early-infancy pattern. Of the four answer choices, only *Listeria* belongs to that maternal-flora group, making it the best response. *S. pneumoniae* and *H. influenzae* dominate later in infancy and childhood once passive maternal immunity declines, and *S. aureus* is tied to surgery, trauma, or indwelling devices rather than spontaneous infant meningitis.

This is also why guidelines pair ampicillin with a third-generation cephalosporin in this age group: the cephalosporin alone misses *Listeria*.

Listeria monocytogenes

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

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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:

This match-the-following is best solved by knowing the true level of the defect for each cause of amenorrhoea.

Asherman acts at the uterus itself - scarring and adhesions of the endometrial cavity, typically following a postpartum or post-abortion curettage, so menstruation stops despite normal ovarian and pituitary hormones. **Sheehan** is a pituitary problem: ischaemic necrosis of the gland after postpartum haemorrhage lowers FSH and LH (hypogonadotropic). **POF** is an ovarian problem where the gonads fail and FSH rises markedly (hypergonadotropic). **PCOD** features chronic anovulation with a high LH:FSH ratio and androgen excess.

Checking the options, only the first - Asherman paired with intrauterine adhesions from endometrial scarring - states the mechanism correctly. The remaining three deliberately swap mechanisms (PCOD is not low-LH, Sheehan is not ovarian, POF is not an outflow block).

Asherman = intrauterine adhesions

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:

Chorionicity drives twin complications, so anchor on the membranes. In an MCDA gestation there is one placenta shared by both fetuses but a two-layer dividing membrane creates two distinct amniotic compartments.

Shared-placenta problems still apply: unequal placental sharing produces selective FGR, the anastomoses can cause TTTS/TAPS, and monochorionic twins carry an excess of structural anomalies and deformities. So options offering sFGR and deformities are genuine MCDA risks.

The trap option is cord entanglement. Two cords can only knot around each other when both fetuses float in the *same* sac. That situation is monoamniotic (MCMA), where there is no separating membrane. The diamniotic membrane in MCDA keeps each cord in its own sac, so entanglement is essentially excluded.

First-trimester MTP is an elective intervention and not a disease complication, but the single best discriminator the examiner wants is the membrane-dependent event.

Cord entanglement — an MCMA, not MCDA, complication

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:

Work backward from the timeline. The swelling appears *immediately* after a forceps delivery, which points to trauma rather than infection or a developmental lesion.

Forceps blades can tear small vessels in the paravaginal connective tissue while the overlying mucosa stays intact. The escaping blood is trapped in a confined plane, so it forms a tense, exquisitely tender, fluctuant lump. Because the mass lies adjacent to the lower urinary tract, its pressure on the urethra explains the difficulty in passing urine.

Contrast the distractors: a urethral diverticulum is chronic with post-micturition dribbling; a cervical tear bleeds briskly from the canal rather than producing a vulval lump; an abscess needs days and brings fever and redness. None match an instant, painful, fluctuant post-forceps swelling.

Acute + painful + fluctuant + post-instrumental ⇒ a blood collection.

Vulvovaginal (puerperal) hematoma

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

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

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

Correct Answer: (D) Goodell sign

SOLUTION:

Pregnancy signs sit on a ladder of certainty. At the bottom are presumptive signs the patient notices; in the middle are probable signs the clinician elicits; at the top are positive signs that prove a fetus exists (heart tones, palpable fetal parts, ultrasound).

The probable group is dominated by softening and shape changes of the uterus and cervix found on bimanual examination. Goodell sign is one of these: the normally firm cervix becomes soft around the sixth week because of marked vascular engorgement and oedema. The examiner can feel it, but softening alone does not guarantee pregnancy, which is exactly why it is labelled probable rather than positive.

For completeness, Hegar (soft isthmus), Oslander (fornix pulsation) and Braxton Hicks (painless contractions) also belong to the probable tier — but the examiner has marked Goodell as the answer, and it fits the definition perfectly.

Cervical softening detected on exam ⇒ probable, not positive

Goodell sign

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

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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:

Think of progesterone as a respiratory stimulant. It sensitises the brainstem respiratory centre to carbon dioxide, so for any given PCO_2 the woman breathes more. The clinical correlate is the well-known dyspnoea/hyperventilation of normal pregnancy.

Because the extra ventilation comes chiefly from bigger breaths, tidal volume climbs by about a third while respiratory rate barely changes. That eliminates the option claiming a fall in tidal volume.

Greater alveolar ventilation washes out CO_2 , so arterial PCO_2 drops to the high-20s to low-30s mmHg, giving a chronic respiratory alkalosis that the kidney compensates by excreting bicarbonate. Therefore the options claiming a rise in PCO_2 are wrong. Oxygenation stays normal or improves marginally, so a fall in PO_2 is also incorrect.

$\uparrow$ progesterone $\Rightarrow$ $\uparrow$ respiratory drive $\Rightarrow$ $\uparrow V_T \Rightarrow$ $\downarrow PaCO_2$

Increased respiratory drive (hyperventilation)

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

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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:

A prior pre-eclampsia flags this pregnancy as high risk, so the question is really asking for the prophylaxis with the strongest evidence base.

That intervention is low-dose aspirin. Given early — by 12 to 16 weeks and ideally before 16 weeks — and continued to delivery, aspirin shifts the placental prostacyclin-to-thromboxane ratio at the time of trophoblast invasion and meaningfully lowers the rate of pre-eclampsia and its severe, early-onset forms. The stem's option of starting aspirin from 12 weeks captures this perfectly.

The remaining choices fall short. Pairing aspirin with LMWH adds no reliable pre-eclampsia benefit and is reserved for thrombophilia or recurrent pregnancy loss. Cutting salt has never been shown to prevent the disease. Calcium only helps where baseline dietary calcium is low and is at best adjunctive, not the best single answer for a high-risk woman.

High risk + start ≤ 16 wk $\Rightarrow$ low-dose aspirin

Aspirin from 12 weeks

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

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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:

The shape of the epidemic curve tells the story. A propagated epidemic is fuelled by one infected person passing the agent to the next, so cases arrive in successive waves; each wave sits about one incubation period after the previous one, giving a curve with several rising peaks rather than a single spike.

For that pattern to appear, the disease must spread directly between people. Measles fits exactly — an extremely transmissible droplet-spread virus that moves from case to case and generates the classic multi-peaked propagated curve.

The food-poisoning options behave differently. Staphylococcal and Salmonella food poisoning are point-source events: many people eat the same contaminated meal and fall ill together, producing one tight peak with no serial generations. Typhoid is likewise usually a water- or food-borne common-source outbreak, not the prototypical propagated epidemic.

Peaks $\approx$ 1 incubation period apart $\Rightarrow$ person-to-person spread

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:

The RMNCAH+A scorecard assigns colours by direction-of-benefit of each metric. Mortality measures (IMR, MMR) and fertility (TFR) are *inverse* indicators - lower is better - so a low recorded value sits in the GREEN zone. Coverage measures are *direct* indicators - higher is better.

Apply this rule:

IMR $< 20\% \Rightarrow$ good $\Rightarrow$ green

MMR $< 20\% \Rightarrow$ good $\Rightarrow$ green

children receiving healthcare $< 20\% \Rightarrow$ poor coverage $\Rightarrow$ red

Because only one in five children is reached, the service-delivery gap is severe and the district is flagged for corrective action.

Children receiving healthcare < 20% is the RED card indicator

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

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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:

In major-haemorrhage and massive-transfusion physiology, three self-amplifying derangements define the *lethal triad*: a fall in core temperature, failure of clotting, and a drop in blood pH. Symbolically:

Lethal triad = { hypothermia, coagulopathy, acidosis }

Citrate in stored blood binds ionised calcium, producing hypocalcemia (the usual "fourth" metabolic partner), and old stored cells release potassium, producing hyperkalemia. Of the four listed, coagulopathy and hypothermia are core triad members and hypocalcemia is the closely linked metabolic associate, leaving potassium excess as the outsider.

Hyperkalemia is NOT a classic lethal-triad component

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

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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:

Lamotrigine stabilises neuronal membranes by use-dependent blockade of voltage-gated Na^+ channels, damping repetitive firing and presynaptic glutamate release. This pharmacology makes it strong against partial (focal) seizures and secondarily generalised / primary generalised tonic-clonic seizures.

Cross-checking the options: myoclonic jerks may be *provoked* by lamotrigine, absence seizures respond better to ethosuximide and valproate, and atonic drops belong to Lennox-Gastaut management where lamotrigine is adjunctive only. The cleanest, board-favoured indication among the four is generalised tonic-clonic seizures.

Lamotrigine is particularly useful for GTCS

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

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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:

Reflex tachycardia from a calcium antagonist is a baroreflex response: vasodilation lowers pressure, baroreceptors fire less, and sympathetic outflow rises, releasing noradrenaline that speeds the heart. The release of noradrenaline at sympathetic terminals is gated by *N*-type voltage-gated calcium channels.

Cilnidipine is a dual *L/N*-type blocker. Its *L*-type action relaxes vascular smooth muscle to lower BP, while its *N*-type action at the sympathetic nerve ending suppresses the very noradrenaline release that would otherwise drive the reflex heart-rate rise:

***N*-type blockade $\Rightarrow$ $\downarrow$ noradrenaline release $\Rightarrow$ no reflex tachycardia**

T-type and R-type channels are not the sympathetic-terminal target responsible for this benefit.

Cilnidipine additionally blocks *N*-type Ca^{2+} channels

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:

Receptors split into two broad signalling classes. Ionotropic receptors are themselves ion channels - the agonist binds and the pore conducts ions within milliseconds. Metabotropic receptors couple to G-proteins and act via second messengers over seconds.

Sorting the choices:

5-HT₃ → cation channel (ionotropic)

Nicotinic → cation channel (ionotropic)

GABA-A → chloride channel (ionotropic)

H₂ → G_s-coupled GPCR ⇒ ↑ cAMP (metabotropic)

Since every histamine receptor subtype is G-protein-coupled, H₂ is the lone non-channel here.

H₂ receptor is NOT a ligand-gated ion channel

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

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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:

The Chapel Hill Consensus sorts vasculitis by the dominant calibre of vessel involved. Reading the options against that scheme:

Leukocytoclastic → small (postcapillary venules)

GPA → small (ANCA-associated)

MPA → small (ANCA-associated)

PAN → medium muscular arteries

Polyarteritis nodosa is the textbook necrotising arteritis of medium-sized muscular arteries, characteristically ANCA-negative and linked to hepatitis B, sparing the lungs. The other three sit in the small-vessel compartment.

PAN is the medium-vessel vasculitis

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

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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:

Liver fibrosis is fundamentally about excess collagen laid down by activated hepatic stellate cells, so the right answer is the stain that is specific for collagen.

Masson trichrome uses an acid dye sequence in which collagen retains aniline blue (or light green), giving fibrous septa a striking blue-green colour against red muscle and cytoplasm; this makes it the workhorse for grading hepatic fibrosis and confirming cirrhosis. The distractors target completely different targets: PAS marks glycogen and basement membranes and is used (with diastase) for alpha-1-antitrypsin globules, GMS is a silver method for fungi and Pneumocystis, and Gram stain separates bacteria into positive and negative groups. Since only one of these visualises collagen, the choice is unambiguous.

Masson trichrome

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

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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:

Geriatric falls are driven mostly by the systems that keep a person upright: the balance apparatus, the strength of the legs, and the integrity of the joints used for gait. Impaired balance from vestibular, proprioceptive or central causes destroys postural control; muscle weakness from sarcopenia removes the ability to recover

from a stumble; and rheumatoid arthritis produces painful, deformed, unstable joints with an antalgic gait. Each of these acts mechanically and directly to topple a patient. Hearing impairment, by contrast, contributes only indirectly – chiefly by reducing awareness of the surroundings and as a shared marker of inner-ear ageing – so among the four it is the option that is not a direct cause. Hence the odd-one-out is hearing.

Hearing impairment

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

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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:

The frailty syndrome describes an old person whose reserves have shrunk so far that a minor stressor causes disproportionate decline, and the engine of this process is loss of muscle. **Sarcopenia** – the progressive fall in skeletal muscle mass, strength and function – produces the grip weakness, slowed walking speed, fatigue and inactivity captured by the Fried phenotype, so it is the most direct determinant of frailty. The other choices are weaker: chronological age correlates with frailty but the two are conceptually separate, since fitness varies widely at any age; frailty is in fact commoner in women, ruling out male gender; and gait speed is merely one downstream marker rather than the cause. Therefore the underlying answer is muscle loss.

Sarcopenia

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

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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:

The key to nicotine replacement therapy is the pH-dependent buccal absorption of nicotine. Because the un-ionised form crosses the oral mucosa, the mouth must stay alkaline; acidic drinks such as coffee, cola and citrus juice lower the pH and trap nicotine in its ionised state, so the patient is instructed to avoid them for roughly fifteen minutes before and during chewing the gum or sucking the lozenge – which is exactly why the acidic-food statement is the right answer. Testing the other claims: an equivalent-strength lozenge dissolves fully and actually releases about a quarter more nicotine than gum, so gum being superior is false; varenicline's old boxed warning was for neuropsychiatric effects and was withdrawn after the EAGLES study, not a cardiovascular warning; and orally swallowed nicotine is largely destroyed by first-pass metabolism, so the gastrointestinal route is ineffective. Only the acid-avoidance instruction survives scrutiny.

Avoid acidic food ~ 15 min before oral NRT

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

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

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

Correct Answer: (C) Beta-adrenergic receptor

SOLUTION:

G-protein coupled receptors are sorted by the alpha-subunit they recruit, and the stimulatory class G_s is defined by its activation of adenylyl cyclase and a rise in **cAMP**. Among the options, the **beta**-adrenergic receptors (beta-1 in the heart, beta-2 in bronchial and vascular smooth muscle, beta-3 in fat) are the textbook G_s receptors. The remaining choices belong elsewhere: the **M2** muscarinic receptor and the **alpha₂**-adrenergic receptor both signal through G_i and lower cAMP, while the **M1** muscarinic receptor signals through G_q to drive the phospholipase-C/**IP₃**/calcium pathway. Therefore the one receptor that couples to G_s is the beta-adrenergic receptor.

β -adrenergic receptor (G_s)

Quick Tip: Which catecholamine receptor raises cAMP?

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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:

Each agent here is a targeted oncology drug defined by the kinase or receptor it blocks. Sunitinib is an oral multi-targeted inhibitor that hits VEGFR, PDGFR and **c-KIT**, which is why it works in renal cell carcinoma and in imatinib-resistant gastrointestinal stromal tumours. Nilotinib is a more potent second-generation inhibitor of the **BCR-ABL** fusion kinase and is used in chronic myeloid leukaemia. Trastuzumab is a humanised monoclonal antibody directed at the **HER2** receptor on breast and gastric cancers. Gefitinib is a small-molecule inhibitor of **EGFR** tyrosine kinase used in EGFR-mutant lung cancer. Pairing them gives Sunitinib with c-KIT, Nilotinib with BCR-ABL, Trastuzumab with HER2 and Gefitinib with EGFR, and only the first option keeps all four correct while the others swap targets.

Sunitinib-cKIT, Nilotinib-BCRABL, Trastuzumab-HER2, Gefitinib-EGFR

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

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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:

β -thalassemia major arises from grossly deficient β -globin chain production. Excess α -globin chains have no partner, precipitate, and form toxic inclusions inside developing erythroblasts.

Because these precursors are destroyed within the marrow before maturing, the marrow churns out cells but very few usable red cells survive - the hallmark known as *ineffective erythropoiesis*. This is the primary lesion and the correct choice.

The cascade that follows: the huge mass of dying erythroid precursors secretes *erythroferrone*, which switches off hepcidin. Low hepcidin keeps ferroportin open, so dietary iron pours in; combined with lifelong transfusions this produces systemic iron overload. Thus hepcidin is suppressed (not raised), making the "hepcidin increased" option false; the "suppressed hepcidin / iron overload" statement is true but is a downstream effect, not the defining mechanism. The destruction of red cells is mainly intramedullary and splenic (extravascular), not intravascular.

Ineffective (defective) erythropoiesis is the central mechanism

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:

Longevity pharmacology centres on a handful of nutrient-sensing nodes: mTOR, AMPK, sirtuins, and autophagy. Map each drug to its node.

Rapamycin is the prototypical **mTOR** inhibitor - correct. Metformin is an **AMPK** activator that mimics caloric restriction - correct. Resveratrol stimulates **SIRT1**, an **NAD⁺**-dependent deacetylase - correct.

The odd one out is spermidine. Spermidine is one of the most robust dietary inducers of *autophagy*; by inhibiting the acetyltransferase EP300 it relieves a brake on autophagy and thereby *up-regulates* it. The given pairing claims spermidine "decreases autophagy," which reverses its true action. Hence this is the incorrect match.

Spermidine **increases** autophagy ⇒ option 4 is wrong

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

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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:

Fenfluramine is a serotonergic antiseizure agent used in Dravet syndrome. The clinically decisive safety issue is rooted in its serotonin pharmacology.

Serotonergic stimulation of **5-HT_{2B}** receptors on cardiac valves drives fibrotic valvular thickening and can raise pulmonary vascular pressure. This is the same mechanism that caused the notorious fen-phen valvular heart disease in the 1990s. Consequently, regulatory labelling requires *cardiac surveillance*: an echocardiogram before starting, repeated during treatment, and after stopping, to screen for valvular regurgitation and pulmonary arterial hypertension.

The other options belong to different drugs - agranulocytosis to clozapine/carbamazepine, hepatic failure to valproate/felbamate, and renal failure is not the signature fenfluramine toxicity. Therefore cardiovascular (echo) monitoring is the answer.

Monitor cardiovascular status with echocardiography

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

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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:

Renin release is governed by the juxtaglomerular apparatus, which integrates three inputs. Work through what raises versus lowers renin.

The afferent arteriolar baroreceptor releases *more* renin when wall stretch falls, i.e., when renal perfusion pressure drops (hypovolemia, hypotension, renal artery stenosis). The macula densa releases *more* renin when it senses *low* **NaCl** delivery, and *less* when **Na⁺** delivery is high. Sympathetic β_1 activation also augments renin.

Testing the choices: high macula densa sodium $\Rightarrow$ inhibits renin (option 1 wrong); high perfusion pressure $\Rightarrow$ inhibits renin (option 2 wrong); expanded ECF volume $\Rightarrow$ inhibits renin (option 4 wrong). Only *reduced renal perfusion pressure* increases renin.

Reduced renal perfusion pressure stimulates renin

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

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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:

Approach any anemia by cell size first, then iron studies. Here the **MCV** is **68 fL**, well below the **80 fL** lower limit, placing this in the *microcytic* category.

Among microcytic anemias, the serum ferritin separates them cleanly. Ferritin reflects total body iron stores; a *low* ferritin means the iron tank is empty, which is specific for iron deficiency. In thalassemia and anemia of chronic disease ferritin is normal or elevated, so a low value rules them out.

The distractors fail on size or stores: megaloblastic anemia is macrocytic (high **MCV**), aplastic anemia gives pancytopenia with normal/high ferritin, and hemolytic anemia is normocytic with high reticulocyte count, **LDH** and bilirubin rather than a low ferritin. Everything converges on iron deficiency.

Low MCV + low ferritin ⇒ Iron deficiency anemia

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

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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:

Severe hypercalcemia of malignancy is a fluid problem before it is a bone problem. High calcium blunts the kidney's response to ADH, producing polyuria, and patients often vomit, so they arrive markedly *dehydrated*.

The first move is therefore vigorous *isotonic saline* resuscitation. Re-expanding the intravascular space restores glomerular filtration and enhances urinary calcium excretion, dropping the serum calcium within hours.

Only afterwards do the antiresorptives matter: a bisphosphonate such as zoledronic acid shuts down osteoclasts but its effect is delayed by **2-4** days, and calcitonin can bridge that gap. Loop diuretics are reserved for the volume-replete patient who develops fluid overload - giving them to a dehydrated patient worsens things. Searching for an exogenous calcium source is pointless when the driver is tumour PTHrP/osteolysis.

First step = IV isotonic saline (volume expansion)

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

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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:

The correct response is the **visual** system. Sensory transduction normally produces a depolarizing generator potential, but the retinal rod and cone are the textbook outlier. In the dark, a cGMP-gated cationic conductance sustains an inward dark current (Na^+/Ca^{2+}), holding the photoreceptor depolarized and tonically secreting glutamate. When a photon strikes, the cascade rhodopsin → transducin → phosphodiesterase degrades cGMP; the cation channels shut, the dark current collapses, and the membrane potential moves to a more negative value — i.e. it hyperpolarizes — cutting transmitter release. Contrast this with olfaction (cAMP-gated depolarization), gustation (depolarizing receptor potentials), and touch (mechanically gated depolarizing currents), all of which depolarize. The directionality is summarised by the dark-current logic: light ⇒ less cGMP ⇒ channels close ⇒ hyperpolarization.

Visual (photoreceptor) — hyperpolarizes to light

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

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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:

The single most important disturbance in refeeding syndrome is **hypophosphatemia**. The pathophysiology begins with reintroduced carbohydrate provoking endogenous insulin release. Insulin pushes phosphate, potassium and magnesium into cells and simultaneously ramps up phosphorylation reactions — ATP and 2,3-DPG generation — each of which sequesters inorganic phosphate. Because a starved patient already has depleted total-body phosphate stores, the serum level plunges, sometimes precipitously. Clinically this manifests as muscle weakness, rhabdomyolysis, diaphragmatic failure, cardiac arrhythmia, hemolysis and neurological signs. While potassium and magnesium also drop and require correction, phosphate is the marker that defines the syndrome and guides the cautious 'start low, go slow' refeeding protocol with prophylactic thiamine and electrolyte replacement. Calcium and sodium are not characteristic.

Hypophosphatemia

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

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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:

Two statements hold true for ESBL producers: **carbapenems are usable (indeed preferred)** and the **resistance is plasmid-borne**. Extended-spectrum beta-lactamases are mutant enzymes (TEM, SHV and the dominant CTX-M families) that cleave penicillins, the oxyimino third-generation cephalosporins and monobactams; they are classically inhibited by clavulanate on a test plate. Genetically, the determinants ride on transferable plasmids, which is why ESBL organisms spread between patients and species and frequently carry linked fluoroquinolone and aminoglycoside resistance. Therapeutically, a carbapenem such as meropenem is the stable backbone for serious disease. The two false claims: third-generation cephalosporins are substrates of the enzyme and must be avoided regardless of an in-vitro 'susceptible' flag, and piperacillin-tazobactam is unreliable in severe infection owing to the inoculum effect. Hence the true pair is carbapenem use and plasmid transmission.

Carbapenems usable + plasmid-transmitted

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

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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:

New neurons in the adult brain arise in the **dentate gyrus** of the hippocampus. Adult neurogenesis is restricted to two stem-cell niches: the subgranular zone of the hippocampal dentate gyrus, which adds granule neurons that support pattern

separation, learning and memory, and the subventricular zone lining the lateral ventricles, whose progeny migrate via the rostral migratory stream into the olfactory bulb. Of the four choices, only the dentate gyrus is one of these niches. The ventral spinal cord does not regenerate neurons; the substantia nigra's dopaminergic cells are not replaced, which is precisely why their progressive loss produces irreversible parkinsonism; and the noradrenergic locus ceruleus is likewise non-regenerative. Therefore the dentate gyrus is the unique correct site.

Dentate gyrus (hippocampus)

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

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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:

Impaired emotional prosody localises to the **cingulate gyrus**. Prosody denotes the musical contour of speech — pitch, rhythm, stress and tone — and its affective component lets us hear emotion in a voice. This affective layer is a limbic function, and the anterior cingulate gyrus is the central limbic hub that injects emotional and motivational drive into vocal output; damage here yields aprosodia, with speech that is monotonous and emotionally flat. The competing options serve different roles: the superior temporal gyrus underpins auditory comprehension (Wernicke's territory), the parietal lobe handles somatosensory integration, and the precentral gyrus is the primary motor strip for articulatory

movements. None of these is the generator of emotional tone. Thus the cingulate gyrus is the lesion site producing loss of emotional prosody.

Cingulate gyrus

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

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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:

The condition that does NOT cause high triglycerides is **abetalipoproteinemia**. Take each in turn. A fructose-rich diet floods the liver with substrate for de novo lipogenesis and accelerates VLDL secretion, elevating plasma TG. In diabetes mellitus, deficient or ineffective insulin lowers lipoprotein lipase activity and increases hepatic VLDL synthesis, a classic cause of hypertriglyceridemia. Nephrotic syndrome, through hypoalbuminemia, stimulates compensatory hepatic lipoprotein production, raising cholesterol and triglycerides together. Abetalipoproteinemia is the opposite scenario: a loss-of-function mutation in microsomal triglyceride transfer protein (MTP) prevents lipidation and assembly of apoB-containing particles, so chylomicrons, VLDL and LDL essentially vanish from plasma. The result is markedly low — not high — triglycerides and cholesterol, with fat malabsorption, acanthocytosis and fat-soluble vitamin deficiency. Hence it is the exception.

Abetalipoproteinemia (low TG)

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

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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:

The single best-known molecular signature of ultraviolet injury to DNA is the **pyrimidine dimer**.

When skin is exposed to UV-B, two adjacent pyrimidine bases on the same strand (classically thymine-thymine) absorb the photon and become covalently joined through a four-membered cyclobutane ring. The resulting *cyclobutane pyrimidine dimer* (and the related 6-4 photoproduct) kinks the helix and stalls DNA and RNA polymerases.

Cells correct this through nucleotide excision repair, in which a stretch of the damaged strand is excised and resynthesised. Loss of this machinery (as in xeroderma pigmentosum) leaves dimers in place, producing the hallmark C to T mutations of sun-induced skin cancer.

Contrast this with the distractors: cytosine deamination to uracil is a spontaneous hydrolytic mutation, p53 loss and cyclin elevation are downstream cell-cycle effects rather than the direct photochemical lesion.

UV light $\Rightarrow$ pyrimidine (thymine) dimers

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

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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:

Transaminases swap the amino group between an amino acid and an alpha-keto acid, and every one of them depends on a single B-vitamin cofactor: **Vitamin B6**.

In its active state, B6 (pyridoxine) is converted to *pyridoxal phosphate* (PLP). PLP sits in the enzyme active site and reacts with the incoming amino acid to form a Schiff-base aldimine; the amino group is then handed over via a pyridoxamine phosphate intermediate to the recipient keto acid. This ping-pong mechanism is the defining feature of aminotransferases such as AST and ALT.

The other vitamins serve different chemistries: thiamine (B1) as TPP drives decarboxylations, riboflavin (B2) as FAD and niacin (B3) as NAD/NADH handle redox transfers. None of these can perform amino-group transfer.

Transamination $\Rightarrow$ Pyridoxal phosphate (Vitamin B6)

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

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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:

The key constraints are two: the target is a **protein**, and it must be demonstrated *within a tissue section* (FFPE) without destroying the architecture. Only one assay satisfies both - **immunohistochemistry**.

IHC applies a primary antibody against the antigen of interest directly onto the deparaffinised slide; after antigen retrieval, a labelled secondary system and a chromogen (commonly DAB) produce a coloured deposit exactly where the protein resides. The pathologist can therefore see which cells and which subcellular compartment express the marker.

FISH is ruled out because it visualises DNA/RNA sequences, not proteins. Western blotting and flow cytometry can both quantify proteins, but each needs the tissue to be disrupted - western blot needs a lysate run on a gel, flow cytometry needs a single-cell suspension - so both forfeit the in-situ localisation the question demands.

FFPE protein localisation ⇒ Immunohistochemistry (IHC)

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

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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:

Bacterial genomics usually means one circular chromosome, but a handful of organisms break this rule. The textbook answer for a **two-chromosome genome** is ***Vibrio cholerae***.

V. cholerae packages its DNA into two circular replicons: a large chromosome (~3 Mb) holding the bulk of housekeeping and virulence genes, and a smaller second chromosome (~1 Mb) that evolved from an integrated megaplasmid but is now essential for viability. Both replicate in a coordinated fashion during cell division.

The remaining choices each have the conventional single circular chromosome: *E. coli* (with accessory plasmids), the slow-growing GC-rich *M. tuberculosis*, and the minimalist wall-less *Mycoplasma genitalium*.

Two circular chromosomes ⇒ *Vibrio cholerae*

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

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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:

This vignette is deliberately built to look like pulmonary tuberculosis - chronic constitutional symptoms, cavitary lesions, caseating granulomas - yet the standout details point to **histoplasmosis**.

Two features steer the diagnosis. First, the patient is from *Assam*, part of the North-East Indian belt where *Histoplasma capsulatum* is endemic. Second, the biopsy is rich in *histiocytes*; this dimorphic fungus survives and multiplies as tiny yeasts within macrophages, so a histiocyte-laden, necrotising granuloma is its signature. Special stains (GMS, PAS) reveal the intracellular yeast forms.

The competitors fall away on histology: sarcoid granulomas are characteristically non-caseating; aspergillosis shows septate branching hyphae filling a cavity rather than intracellular yeast; and granulomatosis with polyangiitis produces necrotising vasculitis with geographic necrosis and a sinonasal/renal flavour.

Assam + TB-like caseating histiocytic granuloma ⇒ 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:

The trick here is to spot the one azole that **cannot** treat mucormycosis - that is **voriconazole**.

Mucorales are intrinsically resistant to voriconazole. Clinically this is more than a footnote: widespread voriconazole prophylaxis in neutropenic and transplant patients is a well-documented cause of *breakthrough* mucormycosis, because it suppresses *Aspergillus* while leaving the Mucorales niche open.

Effective options follow a clear hierarchy. Liposomal amphotericin B is the backbone of therapy, given alongside surgical debridement and reversal of the host risk factor. For step-down or salvage, the broad-spectrum azoles posaconazole and isavuconazole both retain anti-Mucorales activity (isavuconazole carries a specific FDA approval).

No anti-Mucorales activity ⇒ Voriconazole

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:

The trabecular meshwork (TM) sits in the angle of the anterior chamber and is the main conventional outflow channel for aqueous humour. Anatomically it is organised into three concentric layers:

1. **Uveal meshwork** - the most internal layer facing the anterior chamber, with wide intertrabecular spaces.
2. **Corneoscleral meshwork** - the bulky middle layer with progressively smaller pores.
3. **Juxtacanalicular (cribriform) meshwork** - the thin outermost layer abutting the endothelium of Schlemm canal, and the site of maximal outflow resistance (relevant in primary open-angle glaucoma).

The **canal of Schlemm** is the circumferential endothelium-lined channel that drains filtered aqueous into the episcleral collector channels and aqueous veins; it lies *beyond* the meshwork and is therefore not counted as a meshwork component.

Hence the meshwork is made of the uveal, corneoscleral and juxtacanalicular parts only.

Uveal + Corneoscleral + Juxtacanalicular meshwork

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

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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:

Dark adaptation is the process by which the eye becomes progressively more sensitive in low light. Several events drive it:

- **Pupil dilates (mydriasis):** sympathetic tone and reduced light input cause the dilator pupillae to widen the pupil, increasing the photon flux reaching the retina - an early effect.
- **Visual pigment regeneration:** in the visual (retinoid) cycle, vitamin A / retinol is converted to 11-cis retinal, which combines with opsin to rebuild *rhodopsin*. Stored rhodopsin makes the rods exquisitely sensitive.
- **Rise in retinal sensitivity:** with abundant rhodopsin and a shift to scotopic (rod) vision, the threshold for light detection falls dramatically.

The statement that rhodopsin/opsin breaks down to *photopsin* is wrong for the dark phase: pigment **bleaching** (rhodopsin → opsin + all-trans retinal) is a property of bright light, and photopsin denotes cone pigments, not a rod-bleaching product. So that option is false.

Dark room $\Rightarrow$ mydriasis + rhodopsin regeneration + $\uparrow$ sensitivity

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

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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:

This is a polytrauma scenario: chest trauma, transient haemodynamic instability and bilateral femoral plus tibial fractures. The orthopaedic strategy hinges on the patient's physiological status.

Damage-control orthopaedics (DCO) - for an *unstable* patient - means rapid temporary stabilisation (usually external fixation of all long bones) to limit blood loss and the inflammatory 'second hit'.

Early total care / definitive fixation - for a *stable* patient - means proceeding to the best long-term construct.

The stem explicitly states the patient is *haemodynamically stabilized*, so we move to definitive fixation:

- **Femoral shaft:** closed intramedullary nailing is the established gold standard, allowing early weight-bearing and reliable union.
- **Tibia:** given the systemic insult and uncertain soft tissues, external fixation provides safe, low-burden stabilisation.

Plating the femur or external-fixing it as definitive care is suboptimal; choosing whatever is fastest is a DCO mindset for the unstable patient; ex-fixing every bone is only temporising.

IM nailing of both femurs + external fixation of both tibiae

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

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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:

The decision in a multiply-injured patient with chest trauma and four long-bone fractures depends on physiological status. The stem states resuscitation has succeeded and the patient is *haemodynamically stable*, so we are choosing

definitive rather than temporary fixation.

Guiding principles:

1. For an unstable patient, perform damage-control external fixation of all long bones - quick, low-stress, life-saving.
2. For a stabilized patient, proceed to the best long-term implant per bone.

Applying principle 2 here:

- **Femur:** closed intramedullary nail - the recognised gold-standard definitive treatment of diaphyseal femoral fractures, enabling early rehabilitation.
- **Tibia:** external fixation provides a safe, low-burden definitive option given the systemic and soft-tissue insult already sustained.

Reversing the constructs (option 2), choosing the fastest technique (option 3, a DCO concept for the unstable), or externally fixing everything definitively (option 4) are all inferior here.

Nail the femurs; external fixation of the tibiae

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

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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:

The diagnostic anchor here is the pairing of a **one-sided** bow-leg with a **past injury** to that limb.

Angular deformities of the lower limb can be:

- *Systemic/metabolic* (e.g., rickets) - these are virtually always **bilateral and symmetric**, since the underlying mineralisation defect affects both legs equally.
- *Local/structural* (e.g., post-fracture malunion, physeal injury, focal bone lesions) - these can be **unilateral**.

Because this deformity is unilateral and tied to earlier trauma, a fracture that united in an angulated (varus) position - i.e., **malunion** - is the best answer. Rickets is excluded by the unilaterality; non-ossifying fibroma is typically an incidental cortical lesion; osteofibrous dysplasia is a rare paediatric tibial lesion producing anterolateral bowing, not a trauma-related varus.

Malunion of a previous fracture

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

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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:

The clinical picture - sudden posterior-ankle pain on push-off, loss of plantarflexion power, and an MRI showing a gap in the Achilles tendon - defines an **Achilles tendon rupture**.

The confirmatory bedside manoeuvre is the **Thompson (Simmonds) calf-squeeze test**. With the patient prone and feet over the edge of the couch, the examiner compresses the gastrocnemius-soleus. In an intact tendon the foot plantarflexes; when the tendon is torn, the foot stays still - recorded as a *positive* test, indicating discontinuity of the musculotendinous unit.

The other choices do not fit:

- *McMurray test* is for knee meniscal pathology.
- *Steroid injection* is a management item and is actually risky near the Achilles.
- *Plaster cast* is a treatment, not a diagnostic sign.

Positive Thompson (Simmonds) test

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

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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:

The vignette gives two anchors: severe immunodeficiency (HIV, CD4 < 40/mm³) and a ring-enhancing intracranial mass. Both point to an opportunistic process rather than a tumour.

In AIDS, the differential for ring-enhancing lesions is led by **toxoplasmosis**, which reactivates when CD4 falls below ~100. On imaging it gives multiple peripherally enhancing nodules with central necrosis and vasogenic oedema, favouring the basal ganglia and corticomedullary junction. The standard approach is empirical sulfadiazine + pyrimethamine; radiological improvement at 2 weeks confirms it.

The competing entities behave differently: primary CNS lymphoma is usually solitary, periventricular, shows restricted diffusion and avidly takes up thallium/FDG; a tuberculoma is a reasonable but less typical lead here; and PML produces non-enhancing demyelinating white-matter lesions and therefore cannot explain ring enhancement.

Hence the single best answer is toxoplasmosis.

Toxoplasma encephalitis

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

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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:

Right iliac fossa pain for two days with a confirmatory ultrasound is a textbook presentation of acute appendicitis. On colour Doppler the inflamed appendix appears as a blind-ending, non-compressible tubular structure measuring more than 6 mm, with a hyperaemic wall and possibly surrounding free fluid.

Because the sonographic diagnosis is already secure, the management question reduces to definitive treatment. The treatment of choice for uncomplicated acute appendicitis is operative removal — appendicectomy, preferably laparoscopic.

The alternatives do not fit: a CECT would only add value if the USG were inconclusive or the appendix non-visualised; image-guided drainage applies to a mature appendicular abscess rather than to acute inflammation; and antibiotic-only therapy is not the standard examination answer when the patient can undergo surgery.

Therefore the next step is to operate.

Surgery — appendicectomy

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

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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 $\text{GFR} < 50 \text{ mL/min/1.73m}^2$
- (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 $\text{GFR} < 50 \text{ mL/min/1.73m}^2$

SOLUTION:

The question hinges on the ACR risk stratification of gadolinium-based contrast for nephrogenic systemic fibrosis (NSF). Linear agents (Group I) carry the greatest risk, macrocyclic agents (Group II) carry a negligible risk, and Group III agents have insufficient data.

The defining feature of **Group II** agents is that their association with NSF is so weak that the ACR does not require routine eGFR measurement before use and permits administration even in patients with impaired kidneys. So the scenario that best demonstrates safe Group II use is the patient whose renal function is known to be reduced.

Among the options, the patient with $\text{GFR} < 50 \text{ mL/min}$ directly tests this principle — a Group II agent remains safe despite the low GFR. The two "no RFT" choices are about whether screening is needed (a separate issue), and the child with normal renal function does not showcase the renal-impairment advantage of Group II agents.

Hence the correct response is the low-GFR patient.

GFR < 50 — Group II GBCA is safe

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:

Upper respiratory tract infection in a 12-year-old is, in the absence of complications, a bedside clinical diagnosis driven by history and examination. The illness is typically viral and resolves on its own, so the value of any imaging is essentially nil.

The guiding principle is appropriateness plus radiation protection in the paediatric age group (ALARA). Imaging is justified only when the picture suggests lower respiratory tract disease — pneumonia, respiratory distress, persistent fever — none of which is stated here. Consequently, the correct next step is to perform no radiological investigation.

Looking at the distractors: a chest radiograph would be the first imaging chosen *if* pneumonia were suspected; HRCT belongs to interstitial or chronic airway disease workups; and CECT brings contrast plus the highest radiation dose, which is wholly unnecessary for a simple cold.

Therefore no imaging is required.

No radiological investigation

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 $\text{GFR} < 50 \text{ mL/min/1.73m}^2$
- (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 $\text{GFR} < 50 \text{ mL/min/1.73m}^2$

SOLUTION:

This is a contrast-safety question built on the NSF risk classification of gadolinium agents. Linear chelates (Group I) are the riskiest, macrocyclic chelates (Group II) are essentially risk-free for NSF, and Group III carries insufficient evidence.

The teaching point about **Group II** agents is their freedom from significant NSF risk: they can be administered to patients with poor renal function and do not strictly require an eGFR check beforehand. The vignette therefore looks for the patient in whom renal function is the limiting concern.

The option describing $\text{GFR} < 50 \text{ mL/min}$ is exactly that patient, and a Group II agent is safe for them. The "no RFT" choices address screening policy rather than safety in renal disease, while the normal-RFT patient could take any agent and so does not isolate the Group II advantage.

The best answer is the patient with low GFR.

GFR < 50 — Group II GBCA is safe

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:

An uncomplicated upper respiratory tract infection in a 12-year-old is managed on clinical grounds; it is a viral, self-resolving condition for which imaging offers nothing.

Two principles converge here — investigation appropriateness and radiation protection in children (ALARA). Radiology is reserved for situations that raise concern for lower respiratory tract disease such as pneumonia or respiratory distress, none of which is present. The correct next step is therefore to obtain no radiological investigation.

The remaining choices fail on appropriateness: a chest radiograph is the entry-level study *only* when pneumonia is suspected; HRCT is for chronic interstitial or airway disease and carries a high dose; and CECT layers contrast on top of an even larger radiation burden — all unjustified for a simple cold.

So no imaging is indicated.

No radiological investigation

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:

The clue is that imaging has reached its limit. In a 70-year-old with prior pulmonary TB on ATT, a newly found vertebral lesion on MRI raises two competing possibilities: tuberculous spondylitis versus a malignant lesion such as metastasis or myeloma. These cannot be reliably separated by appearance alone.

The principle in oncology and infectious-disease workup is the same: when a focal destructive lesion's nature is uncertain, obtain tissue. A CT-guided or open biopsy provides histopathology plus material for AFB culture and sensitivity, simultaneously confirming or excluding TB and screening for cancer in one step.

By contrast, a Mantoux merely reflects past exposure (uninformative here), electrophoresis is a non-specific myeloma screen, and a CT chest evaluates the wrong region. None gives a definitive vertebral diagnosis.

Biopsy – tissue diagnosis is the definitive next investigation

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:

Read this as a sign-to-disease mapping exercise built around epidermal cohesion. Where keratinocytes lose adhesion (acantholysis, as in pemphigus vulgaris), tangential pressure peels the skin and Nikolsky is positive. Where the split is below the epidermis (bullous pemphigoid, a hemidesmosomal/sub-epidermal disease), the overlying epidermis stays cohesive and Nikolsky is negative.

SJS/TEN produces full-thickness epidermal death; pressure over already inflamed skin shears it off, which is described as a pseudo-Nikolsky (Asboe-Hansen) phenomenon rather than the true acantholytic sign.

The single most disease-specific clue, however, is morphologic: pemphigus vegetans - an intertriginous, vegetating variant of pemphigus - produces a tongue with deep furrows and ridges resembling brain convolutions, i.e., the cerebriform tongue. That is the highlighted match.

Cerebriform tongue ⇒ Pemphigus vegetans

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

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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:

The deciding feature is location plus colour: a deeply pigmented lesion on the heel/sole points to acral lentiginous melanoma, the melanoma variant that disproportionately affects palms, soles and nail beds in pigmented skin. Such lesions are clinically deceptive and frequently diagnosed late, so the workup must aim straight at a tissue diagnosis.

For any pigmented lesion suspected of melanoma, the gold standard is histopathological examination of an excisional biopsy. This not only confirms malignancy but yields the Breslow depth, the single most important prognostic determinant guiding margins and sentinel-node decisions.

The bedside stains offered are mismatched to the problem: a KOH preparation hunts fungal hyphae, a Gram stain hunts bacteria, and a Tzanck smear hunts acantholytic or viral giant cells - none can grade or confirm a melanocytic tumour.

Biopsy (histopathology) 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:

Anchor on two words: painful and vesicles. Among genital ulcer diseases, the lesion that is vesicular before it ulcerates and is painful is herpes genitalis, driven by herpes simplex virus (predominantly HSV-2). A primary episode also brings fever, malaise and tender groin nodes, matching the systemic picture given.

Contrast this with the other classic genital infections. Chancroid (Haemophilus ducreyi) is painful but presents as ragged ulcers, not vesicles. HPV gives painless cauliflower-like warts. Syphilis (Treponema pallidum) gives a single painless, firm chancre with painless lymphadenopathy. Only HSV unites grouped vesicles, pain, and constitutional symptoms.

Herpes simplex virus (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:

The key pharmacologic principle is penetration. Tinea capitis is a dermatophytosis that lodges inside the hair follicle and shaft, a compartment topical creams cannot reach in effective concentration. Therefore cure demands a drug delivered systemically.

Oral antifungals - griseofulvin (the traditional first choice in children), terbinafine, or itraconazole - accumulate in keratin and the follicle, clearing the organism. Adjunctive antifungal shampoo reduces shedding but is never used alone.

The distractors are traps: antibiotics target bacteria (useful only for a secondarily infected kerion), methotrexate immunosuppresses and would aggravate infection, and topical steroids drive tinea incognito by masking inflammation while the fungus spreads.

Oral antifungals

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

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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:

Think about what makes VDRL and RPR clinically useful. These reaginic tests measure antibody to a cardiolipin antigen, and crucially they are reported as titres obtained through serial dilution. Being quantitative is their defining strength, and from that single property flows their main clinical use: serial titres can be compared over time, so a four-fold fall confirms cure and a four-fold rise signals relapse or re-infection. Hence both quantitation and treatment monitoring are genuine benefits.

The other two choices are distractors. A single standard dilution is not how these tests work - they are titrated across multiple dilutions. Lifelong positivity is the hallmark of treponemal-specific tests (TPHA, FTA-ABS), whereas non-treponemal titres wane and may sero-revert after adequate therapy, which is exactly why they are useful for follow-up rather than a drawback.

Quantitative $\Rightarrow$ monitoring response to treatment

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:

This is a psychiatric emergency. The man satisfies several validated suicide risk markers - he is male, divorced (no spouse, poor social support), an alcohol user, and clinically depressed, and he is now expressing passive suicidal ideation.

When a depressed patient with stacked risk factors openly questions the point of living, the clinician must treat this as imminent self-harm risk. The single most important first action is to remove the patient from a setting where they could harm themselves and place them under observation, i.e. admit them.

Pharmacotherapy with antidepressants and structured psychotherapy such as CBT form the backbone of long-term recovery, but both have a delayed onset and cannot protect the patient over the next hours. Telling him this is a "normal part of life" ignores a treatable, potentially fatal illness. Therefore, after ensuring immediate safety through admission, definitive treatment is layered on.

Hospitalize the patient

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:

Classical conditioning is associative learning described by Ivan Pavlov. A biologically potent unconditioned stimulus (UCS, e.g. food) reflexively evokes an unconditioned response (UCR, e.g. salivation). When a neutral stimulus (e.g. a bell) is paired with the UCS many times, the neutral stimulus is transformed into a conditioned stimulus (CS).

The defining, observable outcome of successful conditioning is that this conditioned stimulus, presented alone, now triggers a learned reaction - the conditioned response (CR). So the phrase that captures the learned phenomenon is "conditioned stimulus producing a conditioned response."

The other choices are distractors: a UCS evoking a UCR is just the pre-existing reflex; describing the link as CS-CR or UCS-UCR misrepresents the mechanism, since the underlying association formed during acquisition is between the CS and the UCS.

CS → CR (conditioned stimulus produces conditioned response)

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:

Tinea capitis is a dermatophytosis affecting the hair-bearing scalp, with the fungus colonising the hair shaft itself. Clinically you see scaling, patchy hair loss, black dots from broken hairs, and at times a tender boggy kerion.

Because the dermatophyte resides within the follicle and hair shaft, drugs applied only to the skin surface cannot achieve fungicidal concentration at the site of infection. Effective cure therefore requires a drug that reaches the hair via systemic circulation.

Oral antifungals are the cornerstone - griseofulvin is the classic agent, and oral terbinafine, itraconazole or fluconazole are also used. Medicated antifungal shampoos serve only to limit contagious spore shedding.

Antibiotics target bacteria and are irrelevant unless there is bacterial superinfection; methotrexate is an immunosuppressant that would aggravate the infection; topical corticosteroids blunt local immunity and may mask or worsen the lesion (tinea incognito).

Oral antifungals

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:

This is a classic toxicology matching item. The signature clue most strongly tied to a poison is the bitter-almond smell of cyanide, which is the intended single best (circled) response.

Cyanide blocks cytochrome c oxidase, producing histotoxic hypoxia; cells cannot use oxygen, so venous blood stays bright red and the breath/tissues carry a bitter-almond odour.

For completeness, the remaining associations are also genuine: carbon monoxide forms carboxyhaemoglobin causing cherry-red lividity; phosphorus (along with arsenic and thallium) gives a garlic-like odour and luminous, "smoking" stools; organophosphates inhibit acetylcholinesterase, flooding the body with acetylcholine and causing copious secretions (lacrimation, salivation, bronchorrhoea) summarised by SLUDGE/DUMBELS.

Among these, the cyanide-almond link is the most distinctive and exam-tested pairing.

Cyanide → bitter almond smell

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:

Forensic medicine groups mechanical injuries into abrasions, contusions, lacerations, sharp-force (incised and stab) wounds and firearm/blast injuries. The descriptor in the stem - blunt force with no break in skin continuity - points to a contusion.

A contusion, or bruise, forms when blunt impact ruptures small subcutaneous and deeper vessels, allowing blood to seep into surrounding tissue while the skin surface stays intact. Its colour change over days helps in estimating age of injury.

The distractors all involve a breach of the skin: a stab is a sharp, penetrating wound; a laceration is a blunt tear that splits the skin with ragged margins and tissue bridging; a bomb blast causes mixed open injuries with marked tissue disruption.

Contusion

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

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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:

A 16-year-old sexual-assault survivor who is 16 weeks pregnant raises overlapping duties under the MTP Act and the POCSO Act. The task is to find the step that does NOT belong.

Under the amended Medical Termination of Pregnancy Act, the number of doctors whose opinion is required depends on gestational age: up to 12 weeks, one registered medical practitioner suffices; beyond 12 and up to 20 weeks, the opinion of two registered medical practitioners is needed. Since this patient is at 16 weeks, two opinions are compulsory - therefore terminating on a single doctor's opinion is wrong and is the answer.

The remaining actions are all legitimate. Sexual assault of a minor is a mandatorily reportable crime under POCSO, so police notification is required. Products of conception must be preserved for DNA analysis to support prosecution. Because the patient is a minor, guardian/parental consent is obtained for the procedure.

MTP on one doctor's opinion is NOT included (needs two beyond 12 weeks)

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:

Organophosphate compounds covalently phosphorylate acetylcholinesterase, abolishing acetylcholine hydrolysis and flooding cholinergic synapses.

Management has two prongs: *symptomatic* control with atropine (a muscarinic antagonist that dries secretions and corrects bradycardia) and *causal* reversal of the enzyme block.

Enzyme reactivation is achieved only by an **oxime**. *Pralidoxime* carries a quaternary nitrogen that aligns it near the anionic site and a nucleophilic oxime group that attacks the phosphorus, regenerating free, functional AChE:



This particularly rescues nicotinic (muscle weakness, fasciculations) signs that atropine cannot touch. Efficacy falls once the phosphoryl-enzyme complex "ages."

Flumazenil (benzodiazepine antagonist) and naloxone (opioid antagonist) act on unrelated receptors and have no role here.

Pralidoxime

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:

Effective surgical prophylaxis depends on *timing* far more than on prolonging therapy. The bactericidal concentration must already be present in tissue when the scalpel breaks the skin and during entry into the gut.

Guidelines (CDC, SCIP, ASHP) converge on a single window: administer the agent so the infusion is complete **within 60 minutes before incision**, ideally at induction of anaesthesia. Standard cephalosporins are given in this **30-60** minute slot; agents needing slow infusion (vancomycin, ciprofloxacin) are started up to **120** minutes prior.

Doses given hours in advance (**4 h** or **6 h**) allow plasma levels to decay below the minimum inhibitory concentration before contamination, and a dose at closure misses the window entirely. A redose is added intra-operatively if surgery is prolonged (**> 3-4 h**) or blood loss is heavy.

Within 1 hour (30-60 min) before incision

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:

The vignette pairs two findings that, together, are almost diagnostic: jaundice that refuses to clear and a protruding umbilicus.

In **congenital hypothyroidism**, low thyroid hormone depresses uridine diphosphate glucuronyl transferase, so bilirubin conjugation lags and unconjugated jaundice lingers past the physiological window. The same hormone lack causes hypotonia and weak abdominal musculature, allowing an *umbilical hernia* to form. The full syndrome also includes a hoarse cry, macroglossia, constipation, a wide posterior fontanelle and feeding difficulty - remembered by the cretinism picture.

Diagnosis rests on a low T_4 with a high TSH ; newborn screening detects it before irreversible neurodevelopmental delay sets in, and levothyroxine is started promptly.

Down syndrome is a distractor (it shares hernia/hypothyroid risk but not this specific dyad), cerebral palsy produces no jaundice, and biliary atresia gives conjugated jaundice with acholic stools.

Congenital hypothyroidism

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:

The hallmark of **differential cyanosis** is a split colour: the head and right upper limb stay pink while the legs turn blue. Anatomically this can only happen when desaturated blood enters the aorta *beyond* the origin of the head-and-arm vessels.

A large **patent ductus arteriosus** that has developed pulmonary hypertension does exactly this. Once pulmonary pressure exceeds systemic (*Eisenmenger* reversal), venous blood in the pulmonary artery shunts through the duct into the descending aorta:

Pulmonary artery $\xrightarrow{\text{PDA, R} \rightarrow \text{L}}$ Descending aorta (post-subclavian)

Thus the lower body is perfused with deoxygenated blood (cyanosis, clubbing of toes) while pre-ductal vessels keep the upper body pink.

Osler-Weber-Rendu produces telangiectasias and AVMs, peripheral arterial disease produces ischaemic acrocyanosis, and uncomplicated transposition gives global cyanosis - none reproduce this gradient.

PDA with Eisenmenger (right-to-left) shunt

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

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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:

Shoulder dystocia is an obstetric emergency where the fetal anterior shoulder lodges behind the maternal pubic symphysis after the head delivers. Management proceeds in escalating steps (the **HELPER** mnemonic), and the very first physical action is non-invasive.

The **McRoberts manoeuvre** sharply flexes the mother's thighs against her abdomen. This rotates the pubic symphysis anteriorly and flattens the sacral promontory, enlarging the functional outlet and often releasing the shoulder. Adding *suprapubic pressure* (Rubin I) over the fetal anterior shoulder pushes it into the oblique diameter to complete the disimpaction.

If these fail, one escalates to internal rotational manoeuvres (Wood's corkscrew, Rubin II, delivery of the posterior arm). Only when everything else fails does one resort to the *Zavanelli* cephalic replacement or symphysiotomy.

McRoberts manoeuvre with suprapubic pressure

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

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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:

The **Pearl Index** quantifies how often a contraceptive fails in practice, allowing different methods to be ranked.

It is computed as accidental pregnancies normalised to *100 woman-years* of exposure:

$$PI = \frac{\text{accidental pregnancies} \times 1200}{\text{total months of use}}$$

where multiplying by **1200** scales the result to **100** women observed for **12** months each. So if **5** pregnancies occur across **100** women followed for one year, the index is **5**. The smaller the number, the more reliable the method (e.g., IUCDs and implants have very low indices, while barrier methods are higher).

Choices framing it merely "per woman year," as a ratio of pregnancies to losses, or per **1000** women misstate the accepted denominator.

Accidental pregnancies per 100 woman-years of exposure

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

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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:

The key phrase is the proportion of disease in the **entire population** that disappears once the risk factor is removed.

Recall the family of attributable measures:

$$AR_{exposed} = I_e - I_u$$

measures excess risk confined to the exposed.

$$PAR = I_{population} - I_u$$

and the population attributable fraction

$$PAF = \frac{I_{pop} - I_u}{I_{pop}}$$

gives the fraction of all cases in the community attributable to the exposure.

Since the question states that X% of *all* strokes in the population would be cured by abolishing hypertension, we are describing the community-level fraction, i.e. the population attributable risk.

Relative risk

$$RR = \frac{I_e}{I_u}$$

and the odds ratio only describe how strongly exposure and disease are linked; they cannot express a removable population burden. Attributable risk addresses only the exposed subgroup, so it is also incorrect.

Population attributable risk

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

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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:

When several trials are combined, their effect sizes rarely line up perfectly. The degree to which they disagree is the **heterogeneity** of the meta-analysis.

It is captured by:

$$I^2 = \frac{Q - df}{Q} \times 100\%$$

where

$$Q$$

is Cochran's statistic and

$$df$$

the degrees of freedom. An

$$I^2$$

near 0% means the studies essentially agree; values approaching 75% indicate substantial between-study variation, prompting use of a random-effects model.

Therefore heterogeneity is fundamentally a measure of *variation between the included studies*.

It does NOT measure:

- Publication bias - that is judged by funnel-plot asymmetry / Egger's test.
- Confounding - a within-study internal-validity problem.
- Single-study precision - reflected by each study's confidence interval and weight.

Evaluates variation between the included studies

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

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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:

Ticagrelor belongs to the antiplatelet group that targets the ADP-driven

P2Y₁₂

receptor on platelets.

The discriminating point among the options is the **nature of binding**:

• Thienopyridines - clopidogrel, prasugrel, ticlopidine - are prodrugs that bind

P2Y₁₂

irreversibly for the platelet's lifespan.

• Ticagrelor is *not* a prodrug; it is directly active and binds the receptor *reversibly* at an allosteric site.

Consequences of reversible, short-acting binding:

1. It must be dosed *twice daily*, not once.
2. Platelet function recovers faster after stopping the drug.

Regarding bleeding, major bleeding rates were broadly comparable to clopidogrel in PLATO, so 'more cerebral bleed' is not the defining true statement. Therefore the single clearly correct option is its reversible P2Y₁₂ inhibition.

Reversible P2Y₁₂ inhibitor

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

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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:

The graph plots two quantal (cumulative) curves: a left-shifted *efficacy* curve and a right-shifted *toxicity* curve. The 50% points on each define the two doses we need.

Therapeutic index is the ratio of the toxic dose to the effective dose:

$$TI = \frac{TD_{50}}{ED_{50}}$$

Plugging in the figure values

$$TD_{50} = 400 \text{ mg}, \quad ED_{50} = 100 \text{ mg}$$

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

Checking the traps:

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$$\frac{100}{400} = 0.25$$

is the reciprocal - this would wrongly suggest the drug is unsafe.

•

$$400 - 100 = 300$$

(or 500) is a difference, not the required ratio.

• A TI of 1 would mean no separation between benefit and harm.

A higher TI denotes a wider margin of safety, so the drug here has a fourfold safety margin.

$$TI = 4$$

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:

The plot is a classic renal-physiology graph of relative filterability versus effective molecular radius for three differently charged species.

Underlying principle - the glomerular filtration membrane is lined with fixed **negative charges** (heparan sulfate and sialic-acid-rich glycoproteins). By simple electrostatics:

- Like charges repel so anions are held back.
- Opposite charges attract so cations pass most easily.

Hence, holding size constant, the ranking is:

$$\text{filterability}_{\text{cation}} > \text{filterability}_{\text{neutral}} > \text{filterability}_{\text{anion}}$$

On the figure, curve A sits highest (cation), B is intermediate (neutral) and C is lowest and labelled 'anions'. The molecule filtered most readily on the basis of charge is therefore curve A, the cation.

This same charge barrier explains why anionic albumin is largely retained in health, and why its loss (as in minimal change disease) produces selective proteinuria.

A (cation)

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

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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:

The therapeutic angle here is **oral rehydration therapy**, the mainstay of managing secretory (e.g. cholera) diarrhea.

Mechanism of the disease vs the cure:

• Cholera toxin locks open the apical

CFTR

chloride channel, so the gut pours out Cl, Na and water - CFTR drives the illness, it is not the treatment.

• The brush-border **sodium-glucose cotransporter (SGLT1)** stays fully active during the infection. It moves one glucose with two Na ions into the enterocyte, and water follows by osmosis.

Oral rehydration solution deliberately contains *both* glucose and sodium so that SGLT1 can couple their uptake and pull water back across the mucosa, correcting dehydration. This glucose-sodium coupling is precisely why ORS is effective.

The other choices fail: the Na/K-ATPase is a basolateral support pump (not the exploited luminal route), and aquaporin-2 is a renal ADH-dependent water channel unrelated to the gut.

SGLT (sodium-glucose cotransporter)

Quick Tip: Why does ORS contain both salt AND glucose? The cotransporter that links

them is the answer.

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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:

Reversible covalent modification controls the direction of glycogen metabolism. Protein kinase A adds a phosphate group; protein phosphatase-1 removes it. The rule for the storage pathway is simple: the enzyme that *builds* glycogen is turned on when the phosphate is removed.

Glycogen synthase_(dephospho) $\xrightarrow{\text{active}}$ glycogen synthesis

This matches the fed state, where insulin activates phosphatases. Glycogen phosphorylase behaves oppositely - it needs the phosphate (phosphorylated form) to break glycogen down, which is the fasting/glucagon response. For the bifunctional liver enzyme, dephosphorylation activates the PFK-2 (kinase) activity to drive glycolysis, while phosphorylation favours the bisphosphatase. Thus among the classic storage enzymes, the one switched ON by dephosphorylation is glycogen synthase.

Glycogen synthase

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:

The question gives three overlapping descriptors - diphtheria toxin, AB exotoxin, ADP-ribosylation - which are really three facets of the same answer. The molecular event being asked about is inactivation of elongation factor 2.

Diphtheria toxin is a two-component (A-B) exotoxin. The B fragment binds and delivers the A fragment into the cytosol, where the A fragment transfers an ADP-ribose group from NAD⁺ onto diphthamide, a modified histidine residue found only on EF-2:



Once ADP-ribosylated, EF-2 can no longer drive ribosomal translocation, so translation stops and the cell dies. Cholera and *E. coli* LT toxins also ADP-ribosylate, but their target is G α , not EF-2, producing secretory diarrhoea rather than cytotoxicity. Hence the EF-2 inhibitor is the diphtheria toxin.

Diphtheria toxin (ADP-ribosylates EF-2)

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:

Work through the clues. Gram-positive cocci plus catalase positivity points to staphylococci, and in a joint infection that means *Staphylococcus aureus*. The single decisive clue is the antibiotic susceptibility: cefoxitin is the laboratory marker for methicillin resistance. A cefoxitin-sensitive strain is MSSA - it still possesses normal penicillin-binding proteins and is not MRSA.

Catalase(+) GPC $\Rightarrow$ *S. aureus*; Cefoxitin(S) $\Rightarrow$ MSSA

For MSSA the optimal therapy is a β -lactamase-stable anti-staphylococcal penicillin: cloxacillin (or flucloxacillin, nafcillin, oxacillin). These bind PBPs more avidly and clear MSSA faster than glycopeptides. Plain penicillin G fails because staphylococcal penicillinase hydrolyses it, while vancomycin and linezolid should be held back for genuinely resistant (cefoxitin-resistant) MRSA to limit toxicity and resistance.

Cloxacillin

Quick Tip: Cefoxitin sensitive = MSSA, so use a penicillinase-resistant penicillin, not vancomycin.

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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:

The combination of uncontrolled diabetes and a black, dead-tissue eschar on the nose or palate is pathognomonic for mucormycosis (zygomycosis), an aggressive angioinvasive fungal infection. The responsible fungi belong to the Mucorales, and the single most common genus is *Rhizopus*.

Why this fungus and this host: in diabetic ketoacidosis the low pH plus high glucose, together with iron liberated from carrier proteins, create an ideal growth environment, and *Rhizopus* expresses ketone reductase to exploit it. The hyphae invade blood vessels, thrombose them, and the resulting infarction produces the black eschar.

DKA + black eschar ⇒ Mucorales (broad aseptate, 90° branching)

Aspergillus (septate, acute-angle hyphae) is a neutropenic-host pathogen, *Candida* produces budding yeast with pseudohyphae and mucosal disease, and *Cryptococcus* causes meningitis - none fit the diabetic eschar scenario.

***Rhizopus* (mucormycosis)**

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:

Accommodation is the eye's ability to add focusing power for near vision, and that ability is maximal in young children because the lens is highly elastic. Its magnitude is estimated using Hofstetter's average-amplitude rule:

$$A_{avg} = 18.5 - 0.30 \times \text{age (years)}$$

Substituting age = 7:

$$A = 18.5 - 0.30(7) = 18.5 - 2.1 = 16.4 \text{ D}$$

This calculation lands the value in the mid-teens of dioptres, and the matching option offered is +14 D. The other choices correspond to older ages - about +10 D in adolescence and +5 D near the late thirties - reflecting the steady decline that culminates in presbyopia. So for a 7-year-old, the expected accommodative power is approximately +14 D.

+14 D

Quick Tip: Hofstetter: amplitude = $18.5 - 0.3 \times \text{age}$; young children have the highest value.

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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:

Nerve injuries are ranked by Seddon from least to most severe: neuropraxia < axonotmesis < neurotmesis. The question asks for the essence of the mildest grade.

Neuropraxia means the nerve simply stops conducting across a segment - a physiological block, usually due to focal demyelination or compression - while the axon itself and every surrounding connective-tissue layer stay structurally intact. Because no axon is cut, distal Wallerian degeneration does not occur, and once the demyelinated segment remyelinates, conduction returns fully, often within days to a few weeks.

Neuropraxia: conduction blocked, axon intact, no Wallerian degeneration

By contrast, "endoneurium cut" reflects axonotmesis/neurotmesis where the axon is interrupted, and "sheath cut" reflects neurotmesis with complete division requiring surgical repair. Therefore the correct description of neuropraxia is a reversible physiological conduction block.

Physiological (reversible) conduction block, axon intact

Quick Tip: Mildest Seddon grade - conduction blocked but axon and sheath stay intact.

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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:

Nerve injuries are graded by Seddon into three types of increasing severity. The question asks for the meaning of the first and mildest type, *neurapraxia*.

In neurapraxia the insult is essentially a focal *demyelination* of a nerve segment, most often from compression or stretch (e.g., "Saturday night palsy"). The crucial point is that the underlying *axon* stays anatomically continuous and all connective-tissue layers are undamaged. What is lost is the ability to conduct an impulse across that segment - hence a purely *physiological* block.

Two consequences follow. First, since the axon is not severed, *Wallerian* degeneration does not take place distally. Second, recovery is the rule and is rapid, occurring over days to a few weeks as the segment remyelinate; nerve conduction studies show conduction block but preserved distal responses.

Contrast this with the distractors: cutting the axon while sparing the endoneurial tube is *axonotmesis*, and division of the axon together with its sheaths is

neurotmesis. Both of these provoke Wallerian degeneration, unlike neurapraxia.

Neurapraxia = physiological conduction block, axon intact

Quick Tip: Mildest Seddon grade - axon intact, only conduction temporarily blocked.

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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:

This vignette is a textbook description of *infantile scabies*. The three pillars pointing to the answer are distribution, itch and contact history.

Distribution: unlike adults, infants with scabies show lesions on the *palms, soles, umbilicus*, axillae and even the face and scalp. The presence of vesicles on the palms and soles is almost a giveaway, because these acral sites are involved far more often in babies than in older patients.

Symptom: the eruption is fiercely pruritic and classically worsens at *night*, reflecting the activity of the mite *Sarcoptes scabiei* within its burrows.

Epidemiology: scabies spreads by close skin contact, so the fact that other household members have the same itchy rash strongly supports the diagnosis.

Ruling out alternatives - atopic dermatitis in this age group sits on the cheeks and extensor limbs and spares the umbilicus; psoriasis presents as sharply marginated silvery plaques rather than itchy acral vesicles; seborrhoeic dermatitis is a non-itchy greasy scaling of the scalp and flexures. None reproduce the palm-sole-umbilicus pattern with contagion.

Diagnosis = Scabies

Quick Tip: Palms, soles, umbilicus + nocturnal itch + affected family contacts.

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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:

The clinical concern here is a pigmented lesion on a weight-bearing acral site - the heel - which raises the spectre of **acral lentiginous melanoma**, the melanoma variant disproportionately seen on palms, soles and nail beds in pigmented skin.

The fundamental rule for any lesion suspected of being melanoma is that diagnosis is **histological**. You cannot confirm or exclude melanoma by a smear or a lamp; you need tissue. The preferred procedure is an **excisional** biopsy taken through the full thickness of skin so the pathologist can report depth of invasion (**Breslow** thickness), ulceration and mitotic rate - the very parameters that determine prognosis and the width of subsequent wide local excision.

The competing options test whether the candidate confuses investigations: a **KOH** preparation is for dermatophytes, a Gram stain for bacteria, a **Tzanck** smear for herpetic/varicella vesicles or acantholytic pemphigus cells, and Wood's lamp for fluorescent infections or pigment localisation. None can establish a tissue diagnosis of malignancy.

Investigation of choice = Biopsy (histopathology)

Quick Tip: Pigmented lesion suspicious of melanoma - only tissue (biopsy) confirms it.

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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:

This is a classic forensic toxicology distinction between **body packing** and **body stuffing**.

A body packer is a deliberate drug courier - often crossing international borders - who swallows or inserts many carefully manufactured, double- or triple-wrapped packets (frequently condoms or latex) each holding a large, sometimes lethal, quantity of narcotic. The scenario given (an international traveller, multiple professionally sealed condom packets of drugs) is the textbook picture of this premeditated smuggling, hence the answer.

By contrast, a body *stuffer* acts on impulse: when arrest is imminent, the person hastily gulps small amounts of loosely or unwrapped drug simply to hide evidence. Because the wrapping is poor, stuffers are at high risk of early packet rupture and overdose despite smaller drug loads.

Masking is a different concept altogether - concealing the presence of a drug from detection (adulterating samples, using cutting agents), not swallowing it. "Body pushing" is not a valid medico-legal term and serves only as a distractor.

Term = Body packing

Quick Tip: Pre-planned smuggling, many well-sealed packets - not the panic act of stuffing.

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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:

The vignette turns on the *timing* and *pattern* of developmental regression. After an initially normal start, an infant - classically a girl - begins to lose skills very early in life and develops a distinctive movement disorder of the hands. That constellation defines **Rett syndrome**.

Rett results from a mutation in the X-linked **MECP2** gene, which is why it manifests almost only in females (affected males usually do not survive). Around 6 to 18 months the child shows arrest then regression: head circumference growth slows producing acquired **microcephaly**, purposeful hand use is lost and replaced by midline **hand-wringing** or hand-washing stereotypies, and spoken language and gait deteriorate.

The examiner's marginal note '> 2 yrs / Heller' is itself the clue to the trap. **Childhood disintegrative disorder** (Heller syndrome) also features regression, but only after a prolonged period of clearly normal development - at least two and usually more than three years. Since this child regresses much earlier, Heller is excluded.

Autism lacks the acquired microcephaly and hand stereotypies, and ADHD involves no loss of milestones at all.

Diagnosis = Rett syndrome

Quick Tip: Girl, early regression, shrinking head, hand-wringing - think MECP2.

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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:

The drug under scrutiny is *lamotrigine*, and the task is to match it with its most characteristic toxicity rather than confuse it with the side-effect profile of other antiepileptics.

Lamotrigine's defining hazard is dermatological. A skin *rash* develops in a noticeable fraction of patients and can escalate to the severe end of the spectrum - *Stevens-Johnson* syndrome and toxic epidermal necrolysis. Two situations sharply increase this danger: starting at too high a dose or escalating too quickly, and combining the drug with *valproate*, which slows lamotrigine clearance and raises its blood level. For these reasons the drug is introduced with deliberately slow titration. Separately, dose-dependent central effects such as *dizziness*, diplopia and ataxia are frequently reported, which matches the alternative half of the option.

The distractors each belong to a different agent: dose-independent serious *hepatotoxicity* is a valproate/felbamate problem, *gingival* overgrowth flags phenytoin, and permanent visual-field loss is the trademark of *vigabatrin*. Assigning any of these to lamotrigine would be an error.

Lamotrigine → rash / SJS-TEN (and dizziness)

Quick Tip: Slow-titrated AED feared for skin rash and SJS, especially with valproate.

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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:

The answer hinges on remembering the plasmid division of labour in *Bacillus anthracis*. Two plasmids together confer virulence:

***pXO1* → anthrax toxin** (protective antigen + lethal factor + edema factor)

***pXO2* → poly-D-glutamate capsule** (via the *capBCADE* operon)

The capsule of anthrax is distinctive: instead of a polysaccharide, it is a polypeptide of γ -linked D-glutamic acid. Being a poor immunogen and resisting opsonophagocytosis, it lets the bacillus evade host defences. Because the question asks where the capsule comes from, the relevant plasmid is the capsule plasmid.

A useful clinical anchor: the Sterne vaccine strain is deliberately *pXO1*⁺, *pXO2*⁻, so it makes toxin (for immunity) but no capsule (so it stays attenuated).

Conversely a *pXO1*⁻, *pXO2*⁺ strain is encapsulated but non-toxigenic and also avirulent.

Capsule of *B. anthracis* = *pXO2*

Quick Tip: Two plasmids: one for toxin, one for the protein capsule - capsule is on *pXO2*.

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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:

To pick the “novel” antidepressant, rank the candidates by era and mechanism.

Vilazodone ⇒ SPARI (2011) - serotonin partial agonist + reuptake inhibitor; it both blocks the serotonin transporter and partially agonises the **5-HT_{1A}** autoreceptor, which is thought to give a faster onset and fewer sexual side effects than classic SSRIs.

Trazodone ⇒ SARI, an older serotonin antagonist/reuptake inhibitor used at low dose mainly as a hypnotic.

Imipramine, Amitriptyline ⇒ TCAs, the oldest generation, acting on serotonin and noradrenaline reuptake with anticholinergic and cardiac liabilities.

Among these, only vilazodone represents a recently developed molecule with a distinctive dual serotonergic mechanism, so it is the answer.

Novel antidepressant = Vilazodone

Quick Tip: Think SPARI: SSRI plus 5-HT_{1A} partial agonism - the newest of the four.

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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:

This is a classic pattern-recognition stem in reproductive endocrinology. Two discriminating features carry the diagnosis: breast development and sexual hair.

Because the breasts are well formed, oestrogen is acting on the body. Because the pubic and axillary hair are sparse, androgen action is failing - sexual hair is the most androgen-sensitive secondary sexual characteristic.

Oestrogenised body + androgen-unresponsive hair ⇒ Complete AIS

The mechanism: the patient is genetically **46,XY** with intra-abdominal testes. The testes secrete anti-Müllerian hormone, which involutes the Müllerian structures, explaining the *absent uterus*. The testes also make testosterone, but a defective androgen receptor means target tissues ignore it; peripheral aromatisation of that testosterone to oestradiol then drives breast growth. The result is a phenotypically female individual with a short blind vagina, no uterus, and minimal sexual hair.

Contrast with MRKH (**46,XX**): there the uterus is also absent, but androgen receptors work, so pubic and axillary hair are entirely normal - which is not the case here.

Diagnosis = Complete Androgen Insensitivity Syndrome

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:

The combined eye and skin images are a giveaway for neurofibromatosis type 1. On the slit lamp, the small dome-shaped iris hamartomas are Lisch nodules; together with the cutaneous photograph they place this in the NF1 spectrum.

NF1 is diagnosed when at least two clinical criteria coexist:

≥ 6 **café-au-lait macules**, neurofibromas, axillary/inguinal freckling, Lisch nodules, optic glioma, a bony dysplasia, or an affected first-degree relative.

Among the skin lesions, the café-au-lait macule appears earliest - frequently from birth - making it the most characteristic initial marker. A practical numeric threshold to remember:

CALM ≥ 6 lesions, ≥ 5 mm (prepubertal) or ≥ 15 mm (postpubertal).

The distractors belong to other phakomatoses: ash-leaf macules and the shagreen patch are tuberous sclerosis findings, while a port-wine stain suggests Sturge-Weber syndrome. None of these is the defining early lesion of NF1.

Earliest hallmark of NF1 = Café-au-lait macules

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:

A tattoo is permanent precisely because of *where* the ink ends up. The needle pierces past the surface and lays pigment into the dermis.

Reasoning by skin biology:

Epidermis → sheds every ~ 4 weeks ⇒ cannot hold pigment

Dermis → stable collagen + resident macrophages ⇒ traps pigment indefinitely

Once in the dermis, ink particles too large to be cleared are captured by macrophages and fixed within the fibrous matrix, so the design stays visible for life and may even resist early putrefaction - which is why forensic experts rely on tattoos for identifying decomposed or unknown bodies. Some pigment also drains to draining lymph nodes, occasionally pigmenting them.

Layers deeper than the dermis (subcutaneous fat, deep fascia) are below the needle's reach, and the superficial epidermis is far too transient to retain ink.

Tattoo pigment resides 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:

The classic emergency in opioid overdose is failing respiration with pinpoint pupils and depressed consciousness. The drug that reverses this is naloxone.

Mechanism:

Naloxone $\rightarrow$ μ -opioid receptor (competitive antagonist)

By displacing the opioid agonist from μ receptors, naloxone restores the respiratory drive within minutes when given intravenously, intramuscularly or intranasally. A key caution is its short half-life relative to many opioids, so the patient can re-sedate (“renarcotisation”) and may need repeat boluses or a continuous infusion, especially with methadone or extended-release formulations.

Antidote pairings to keep straight:

Benzodiazepines $\rightarrow$ Flumazenil

Organophosphates $\rightarrow$ Atropine (+ pralidoxime)

Paracetamol $\rightarrow$ N-acetylcysteine

Only naloxone targets the opioid receptor, so it is the correct choice for opioid

poisoning.

Opioid antidote = Naloxone

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:

The AAST colon scale runs by how much of the bowel wall is breached. Think of it as a ladder: partial-thickness or contusion sits at *I*, a laceration under half the circumference at *II*, a laceration over half the circumference at *III*, a clean transection at *IV*, and a transection with loss of tissue/blood supply at *V*.

Here the wound crosses more than 50% of the circumference yet the colon is not fully divided, so it stops at the over-half-circumference rung.

laceration > 50% circumference, no transection ⇒ Grade III

Grade III

Quick Tip: Less than 50% circumference is Grade II; more than 50% (no transection) bumps it up one grade.

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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:

The Glasgow Coma Scale sums three components, each scored at the patient's best response. Mapping each finding:

Eyes open only when hurt $\Rightarrow E = 2$.

Only grunts/groans, no real words $\Rightarrow V = 2$.

Reaches toward the painful stimulus $\Rightarrow M = 5$.

Summing:

$$GCS = E + V + M = 2 + 2 + 5 = 9$$

A GCS of 9 corresponds to moderate head injury.

GCS = 9

Quick Tip: E2 + V2 + M5 - just add the three best responses.

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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:

Atrial fibrillation throws clots; when one travels to the superior mesenteric artery, the small bowel is suddenly starved of blood. The classic clue is agonising pain with a deceptively soft, benign-looking abdomen.

Starved tissue switches to anaerobic glycolysis, dumping lactate into the blood, and as cells die they spill lactate dehydrogenase. So the lab signature is a rising serum lactate (driving a high-anion-gap metabolic acidosis) together with elevated LDH.

Amylase is too non-specific to clinch it, and neither calcium nor bilirubin track this disease.

AF embolus + pain out of proportion + ↑ lactate/LDH ⇒ acute mesenteric ischemia

Raised serum lactate / LDH

Quick Tip: Dying gut means anaerobic metabolism - look at the lactate.

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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:

Kawasaki disease is a medium-vessel vasculitis diagnosed by prolonged fever plus a checklist: red cracked lips and strawberry tongue, a polymorphous rash, swollen then peeling hands and feet, neck node enlargement, and red eyes.

The crucial eye detail is that the redness is a ***bulbar conjunctival injection without discharge*** - it spares the limbus and produces no pus. Therefore a ***purulent*** (discharging) conjunctivitis breaks the pattern and is the finding that does not belong; it should make you think of a bacterial or other infective cause instead.

The remaining choices - non-exudative bilateral conjunctival injection, mucosal changes, and cervical adenopathy - are all bona fide criteria.

Purulent conjunctivitis is NOT a feature of Kawasaki disease

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:

When a newborn's sugar dips, the first question is always: is the baby symptomatic? That single answer steers the treatment.

A baby who is jittery, floppy, fitting, or refusing feeds is symptomatic and needs glucose into the vein straight away - a small **2 mL/kg** bolus of **10%** dextrose, then a maintenance infusion.

This particular term baby reads **36 mg/dL** yet looks completely well and is breastfeeding happily. Being **asymptomatic** is exactly what permits the gentler path: keep breastfeeding, then recheck the glucose, escalating to IV dextrose only if the level stays low or symptoms appear.

Asymptomatic $\Rightarrow$ breastfeed and recheck rather than 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:

The HPV vaccine programme aims at girls **9-14** years old, ideally before any sexual exposure. WHO's SAGE and several national programmes have moved to a **single-dose** schedule for this band, and the vaccine reliably triggers strong, high-titre immune responses.

The statement that fails scrutiny is the promise of **complete, 100%** protection. The available vaccines neutralise the dominant oncogenic strains - types 16 and 18 cause roughly **70%** of cervical cancers, with extra coverage in the nonavalent product - but they cannot block every high-risk HPV type. So the protection is substantial yet partial, which is precisely why women must keep up with cervical screening.

"100% complete protection" is the INCORRECT statement

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:

Begin by asking a single question: what is being counted on each axis? Both the average alcohol consumption and the cirrhosis death rate are computed for an *entire nation*, and there are 10 nations giving 10 dots on the scatter plot.

Whenever the observation is a population summary rather than a person, the design is correlational at the group level. The four classic analytic designs differ by their unit of analysis. Cohort, case-control and cross-sectional studies all collect exposure and disease status on *named individuals*. Only the ecological design uses aggregated, population-level figures and correlates them across groups. Mechanism of inference: by lining up 10 countries, the investigator looks for a trend (more drinking → more cirrhosis). This generates a hypothesis cheaply from routine data, but the conclusion cannot be transferred to individuals — doing so risks the ecological fallacy. Confounding by country-level factors (diet, healthcare access, reporting) is also uncontrolled. Therefore the matched description is the group-correlation design. To be sure, contrast it with the alternatives one by one: a cohort would enrol drinkers and non-drinkers as people and watch who develops cirrhosis; a case-control would take cirrhosis patients and disease-free people and ask each how much they drank; a cross-sectional survey would measure drinking and liver status in the same individuals at one moment. Every one of these counts persons. Only the present design counts countries, so it stands apart as ecological.

Ecological (correlational) 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:

The odds ratio summarises a case-control study by comparing the odds of being exposed in the diseased group with the odds of being exposed in the non-diseased group. First build the odds of exposure in each arm. Among the 80 cases, 60 are exposed and 20 are not, so the exposure odds in cases are $60/20 = 3$. Among the 120 controls, 40 are exposed and 80 are not, so the exposure odds in controls are $40/80 = 0.5$. The odds ratio is the ratio of these two odds:

$$OR = \frac{60/20}{40/80} = \frac{3}{0.5} = 6$$

This is algebraically identical to the diagonal cross-product ad/bc , which confirms the value. Because the result exceeds 1, the exposure is associated with markedly higher odds of disease — roughly a sixfold increase. Values such as 1.5 or 0.17 would come from dividing the odds the wrong way round or pairing the wrong cells.

$$OR = 6$$

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:

Positive predictive value answers a clinically practical question: *given a positive test, how likely is real disease?* It is therefore computed down the column of everyone the test labelled positive. The positive column here contains two kinds of people: the genuinely diseased who tested positive (true positives, $TP = 90$) and the disease-free who were wrongly flagged (false positives, $FP = 50$). The denominator is every positive result, $TP + FP = 140$. Applying the definition:

$$PPV = \frac{TP}{TP + FP} = \frac{90}{140} \approx 0.643$$

Expressed as a percentage this is about **64.3%**. In words, roughly two-thirds of positive results are correct and one-third are false alarms. Note that PPV depends on disease prevalence, unlike sensitivity and specificity; in a low-prevalence population the same test would yield a lower PPV because false positives would dominate.

$$PPV \approx 64.3\%$$

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:

This item tests a vocabulary distinction that examiners love. Three related terms describe vaccine performance, and each is tied to a specific setting. Efficacy is measured inside a randomized controlled trial, where participants are selected, doses are guaranteed, and the cold chain is perfect. That protected, idealized estimate here is the higher figure, **90%**. Effectiveness is measured when the same vaccine is rolled out through the routine immunization programme, exposed to logistic losses, incomplete coverage, and a heterogeneous population. Because conditions are imperfect, the field value is lower — the stated **85%**. A third related term, protective efficacy, is essentially the relative reduction in disease attributable to the vaccine and is usually quoted from the trial alongside efficacy; it should not be confused with the field effectiveness asked for here. So the question maps cleanly: the real-life community figure is the effectiveness. The drop from 90% to 85% reflects programme realities — incomplete cold chain, dropouts who miss doses, and a more varied population than the trial enrolled. The 5% gap or the 87.5% average are distractors with no epidemiological meaning, and 90% is specifically the trial-based efficacy.

Effectiveness = 85%

Quick Tip: Effectiveness = real-world/field; efficacy = ideal trial.

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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:

The pair efficacy/effectiveness is separated entirely by the conditions of measurement, so anchor each number to its setting. A randomized controlled trial is the gold-standard, tightly controlled environment: eligible volunteers, supervised dosing, intact cold chain. The protection observed there is the efficacy, and in this question that controlled figure is **90%**. When the vaccine then enters everyday programme use, real-world friction — partial coverage, missed boosters, storage lapses, a sicker and broader population — erodes some benefit. That field value, **85%**, is the effectiveness, and it is expected to sit below the efficacy. Hence the trial-based, idealized number is the efficacy. A useful memory hook: efficacy answers "*can it work?*" (best-case, in a trial), while effectiveness answers "*does it work?*" (everyday programme). Because the everyday answer is dragged down by missed doses, storage lapses and a broader population, effectiveness almost always trails efficacy, which is why 85% is below 90%. The 5% difference and the 87.5% mean are nonsense distractors with no biological meaning, and 85% specifically names the real-world effectiveness rather than efficacy.

Efficacy = 90%

Quick Tip: Efficacy = ideal RCT value (the higher one, 90%).

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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:

The key idea is *extravascular compression*: coronary vessels run inside the myocardium, so whatever pressure the surrounding chamber generates during systole squeezes them and limits flow. Think about the left side first. The left coronary artery feeds the left ventricle, which generates aortic-level systolic pressure around **120 mmHg**. That intense squeeze almost stops left coronary inflow during systole, so the left coronary bed fills chiefly when the ventricle relaxes — a strongly diastolic pattern. Now the right side. The right coronary artery mostly supplies the right ventricle, a low-pressure chamber peaking near **25 mmHg** in systole. With so little compressive force, the artery is not meaningfully throttled, so it keeps receiving blood during systole and continues through diastole. The result is a biphasic tracing with flow in both phases, which is exactly what the "**d + s**" note describes. There is a clinical corollary worth remembering. Because the left ventricle depends so heavily on diastolic filling of its coronaries, anything that shortens diastole — such as a fast heart rate (tachycardia) — preferentially reduces left coronary perfusion and predisposes to subendocardial ischaemia, since the subendocardium is squeezed hardest of all. The right coronary bed, sustained partly during systole, is comparatively less hostage to diastolic time. This is also why aortic diastolic pressure is the main driving force for left coronary flow. Thus the contrast is: right coronary = both phases (biphasic, low RV pressure); left coronary = mainly diastole (high LV pressure). The options claiming a single phase for both, or the reversed assignment, contradict this physiology.

RCA: biphasic (systole + diastole); LCA: predominantly diastolic

Quick Tip: Low RV pressure lets the RCA flow in systole too — biphasic; LCA is mainly diastolic.

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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:

Read the ECG topographically. The triad of *II*, *III* and *aVF* defines the *inferior* territory of the left ventricle. The artery feeding this territory is the one that gives off the posterior descending branch.

Because the human coronary circulation is right-dominant in the large majority of individuals, the posterior descending artery is a branch of the right coronary artery. Hence inferior wall ischaemia maps to the RCA. The same vessel typically supplies the SA and AV nodes, which is why inferior MIs are classically associated with sinus bradycardia and second-degree AV block.

Contrast this with anterior changes (*V*₁-*V*₄, LAD), lateral changes (*I*, *aVL*, *V*₅-*V*₆, LCx) and the global ST depression with aVR elevation seen in left-main disease. None of these fit an isolated inferior pattern.

Inferior wall MI (II, III, aVF) → Right Coronary Artery

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:

Trace the biochemistry. Sustained inflammation drives hepatic synthesis of the acute-phase protein *serum amyloid A* (**SAA**), a high-density-lipoprotein-associated apolipoprotein. When inflammation is chronic and unresolved, **SAA** is cleaved and its insoluble fragments aggregate into beta-pleated fibrils - this deposited material is termed **AA** amyloid, and the resulting disease is reactive (secondary) systemic amyloidosis.

Rheumatoid arthritis is the prototypical Western cause; worldwide, chronic infections such as tuberculosis and osteomyelitis contribute heavily. The kidney is the dominant target organ, explaining the heavy proteinuria.

The alternatives map to different precursors: light-chain **AL** to plasma-cell dyscrasias, **A β ₂**-microglobulin to chronic dialysis, and transthyretin **ATTR** to senile/hereditary cardiac amyloidosis.

Chronic inflammation → SAA → AA amyloid

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:

Interpret the graph through its defining signature: the time taken for any concentration to halve is identical everywhere on the curve. That invariance of $t_{1/2}$ is only possible when elimination rate scales linearly with drug present.

Set up the rate law. If $\frac{dC}{dt} = -kC$, integration gives $C = C_0 e^{-kt}$, an exponential decay. The half-life is $t_{1/2} = \frac{0.693}{k}$, a quantity that contains no C_0 term - hence it is dose-independent. A fixed *fraction* (not amount) leaves per unit time. This is exactly what most drugs at therapeutic concentrations obey, because their metabolising enzymes and transporters are far from saturated.

Were the enzymes saturated, a constant *amount* would be cleared each unit time (zero-order), the plot would be a straight descending line and $t_{1/2}$ would lengthen as concentration rose - the behaviour of ethanol and high-dose phenytoin. The curve described does the opposite.

Constant $t_{1/2}$, $C = C_0 e^{-kt} \Rightarrow$ First-order kinetics

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:

The question targets the radiographic definition of *bulky* mediastinal disease used in Ann Arbor staging of Hodgkin lymphoma, flagged by the letter **X**.

Mechanically, the radiologist measures the greatest transverse width of the mediastinal mass and compares it to the maximum internal transverse diameter of the thorax on a standing posteroanterior chest film, conventionally at the **T5-T6** level. When the ratio exceeds $\frac{1}{3}$, the mass qualifies as bulky and the stage gains the **X** suffix. An alternative size-based criterion labels any single nodal mass of ≥ 10 cm as bulky.

Bulk matters because it predicts higher relapse risk and frequently mandates the addition of radiotherapy to chemotherapy. The other listed fractions ($\frac{1}{2}$, $\frac{1}{4}$, $\frac{2}{3}$) are distractors with no basis in the staging rules.

Bulky mediastinal mass = width $> \frac{1}{3}$ intrathoracic diameter

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:

Diagnose first, then localise the lesion. A nephritic child - smoky/cola urine, **RBC** casts, oedema and hypertension - presenting roughly two weeks after pharyngitis (or six weeks after skin infection) with a nephritogenic strain of group A streptococcus has post-streptococcal glomerulonephritis.

The pathology is immune-complex mediated. Antigen-antibody complexes lodge on the *outer*, subepithelial aspect of the glomerular basement membrane, producing discrete dome-shaped electron-dense deposits the classic literature calls **humps**. Immunofluorescence corroborates this with coarse granular **IgG** and **C3** in a starry-sky distribution; serum complement (**C3**) is transiently low.

Each distractor belongs elsewhere: tram-tracking with subendothelial deposits is membranoproliferative GN, universal foot-process effacement without deposits is minimal change disease, and a smooth linear GBM pattern is anti-GBM disease.

PSGN ⇒ subepithelial electron-dense 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:

Begin with antibody class and target. Anti-Ro (**SS-A**) and anti-La (**SS-B**) are maternal **IgG** autoantibodies, classically of Sjögren syndrome and lupus. Their **IgG** nature lets them traverse the placenta into the fetal circulation.

Once in the fetus they bind antigens expressed in fetal tissues, including the cardiac conduction system. The result is neonatal lupus erythematosus - a passively acquired, self-limited skin eruption that fades as the borrowed maternal antibody is catabolised, accompanied by the feared and frequently irreversible complication of congenital complete heart block from inflammatory injury and fibrosis of the atrioventricular node. Affected infants may also show transient cytopenias and transaminitis.

The other options arise from entirely different maternal antibodies: anti-**AChR** (neonatal myasthenia), anti-**Rh(D)**/blood-group (haemolytic disease of the newborn) and stimulating anti-**TSH**-receptor antibodies (neonatal Graves).

Maternal anti-Ro/anti-La ⇒ Neonatal lupus ± congenital heart block

Quick Tip: SS-A/SS-B antibodies crossing the placenta damage the fetal conduction system.

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:

The microscopic appearance of a myocardial infarct changes predictably with time, and the question asks for the 24-72 hour (1-3 day) picture.

Early on (first day, 4-24 h) the myocardium shows ongoing coagulative necrosis, wavy fibres at the margins, and contraction-band necrosis. By the **second to third day**, the dead muscle provokes a vigorous acute inflammatory reaction, so the slide is dominated by sheets of **neutrophils** infiltrating the necrotic zone. Their enzymes begin to break down the dead tissue.

After this, the inflammatory cast changes: **macrophages** move in around days 3-7 to phagocytose necrotic debris, granulation tissue with neovascularisation appears in the second week, and a firm **collagenous scar** is laid down over weeks 2-8. Therefore the macrophage, granulation, and scar options correspond to later intervals and are excluded.

Matching the 1-3 day window to its hallmark cell:

Neutrophilic (PMN) infiltration of necrotic myocardium

Quick Tip: Acute inflammation dominates the first few days - think which white cell arrives first.

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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:

The brain task described - lining up what the eyes see with where the hand is - is a classic example of multisensory integration, and that job belongs to the **parietal lobe**.

Each lobe has a dominant role: the occipital lobe is purely visual (it cannot move a hand), the temporal lobe handles sound, memory and recognising objects, and the frontal lobe issues the final motor output and plans behaviour. None of these by itself fuses vision with body-position sense.

The parietal lobe, in contrast, carries the primary somatosensory cortex on its postcentral gyrus and, just behind it, the posterior parietal association cortex. This region merges visual and proprioceptive signals to direct reaching and grasping, build a spatial map, and maintain awareness of the body in space. Damage produces hemispatial neglect, apraxia, and impaired visually-guided movement - confirming its role.

Hand--eye coordination is a parietal lobe function

Quick Tip: Which lobe houses somatosensory cortex and posterior visuospatial association areas?

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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:

The inguinal ligament spans from the **ASIS** to the **pubic tubercle** and marks the boundary between the abdominal wall above and the thigh below. The question is essentially asking which flat abdominal muscle gives rise to it.

Of the three flat muscles of the anterolateral abdominal wall, the **external oblique** is the most superficial. Its aponeurosis, on reaching the lower margin, folds backward on itself like a hem; this in-rolled lower border IS the inguinal ligament. Hence it is a direct modification of the external oblique aponeurosis.

The internal oblique and transversus abdominis instead arch over the spermatic cord and unite medially as the conjoint tendon, while the internal oblique also supplies the cremaster muscle. The rectus abdominis is a vertical strap muscle that takes no part in forming this ligament. Several secondary ligaments - lacunar, pectineal and reflected inguinal - are likewise extensions of the same external oblique aponeurosis.

Inguinal ligament = modified lower border of external oblique aponeurosis

Quick Tip: The most superficial of the three flat abdominal muscles rolls under to form this ligament.

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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:

Foreign-body aspiration favours the right side, and the reason lies entirely in comparative bronchial anatomy.

The right main bronchus is the **wider**, **shorter** and more **vertical** of the two; it descends almost as a straight continuation of the trachea. Gravity and the airflow path therefore deliver inhaled objects preferentially down this near-vertical pipe. The left main bronchus is narrower, longer and more horizontal because it must arch over the heart and aortic arch, so it catches debris less often.

On the parenchymal side, the right lung is divided by oblique and horizontal fissures into **three lobes** (superior, middle, inferior), while the left has only two lobes with the lingula representing the middle-lobe equivalent. In an upright, conscious patient an aspirated object typically ends up in the posterobasal segments of the right lower lobe.

Putting the bronchial features together:

Wider + shorter + more vertical bronchus; right lung = 3 lobes

Quick Tip: Which bronchus most nearly continues the straight line of the trachea?

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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:

A positive Benedict's test needs a sugar that still has a free anomeric (reducing) carbon able to donate electrons and reduce Cu^{2+} to red Cu_2O . So the question reduces to: which option has NO free anomeric carbon?

Glucose is a monosaccharide aldose with an open-chain aldehyde - clearly reducing. Maltose joins two glucose units by an α -1,4 bond and lactose joins galactose to glucose by a β -1,4 bond; in both, one anomeric carbon stays free, so both reduce Benedict's reagent.

Sucrose is unique here: its glucose and fructose are united by an α , β -1,2 glycosidic bond that engages BOTH anomeric carbons simultaneously. With no free reducing group left, sucrose cannot reduce cupric ion and the test stays blue (negative). It only turns positive after acid hydrolysis ("invert sugar") frees the monosaccharides.

Sucrose = non-reducing sugar, Benedict negative

Quick Tip: Which disaccharide locks BOTH of its anomeric carbons in the glycosidic bond?

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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:

Pretomanid is the "Pa" in the modern short oral regimens BPaL and BPaLM for highly resistant tuberculosis, and identifying its target separates it from its regimen partners.

It is a **nitroimidazo-oxazine prodrug** that must first be reduced intracellularly by the F420-dependent nitroreductase **Ddn**. This bioactivation explains its two complementary killing modes. In dividing aerobic bacilli it **blocks mycolic acid biosynthesis**, crippling assembly of the waxy mycobacterial cell wall. In dormant, non-replicating bacilli under anaerobic conditions, the activated drug liberates **reactive nitrogen intermediates such as nitric oxide** that disrupt respiration and sterilise persistent organisms.

The decoys correspond to its companion drugs and standard agents: rifampicin hits RNA polymerase (*rpoB*), bedaquiline (**B**) blocks mycobacterial ATP synthase,

and fluoroquinolones (*L* = linezolid is a ribosomal agent, while moxifloxacin) target DNA gyrase. None of these is pretomanid's action.

Pretomanid: inhibits mycolic acid synthesis + releases reactive nitrogen species

Quick Tip: A nitroimidazole prodrug activated by Ddn that blocks cell-wall (mycolic acid) synthesis.

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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:

Necator americanus, the New World hookworm, is a soil-transmitted helminth whose infective form is the **filariform (third-stage, L3) larva** that develops in warm moist soil from eggs shed in stool. The single accepted portal of entry is **active percutaneous penetration** — the larva drills through unbroken skin, typically of the foot in people walking barefoot.

Once in, the larva follows the heart-lung migration: skin venule → right heart → pulmonary capillaries → alveoli → bronchi/trachea → swallowed → small intestine, where it matures into a blood-sucking adult and produces chronic iron-deficiency anaemia.

Contrast: *Ancylostoma duodenale* can additionally be acquired orally, but *Necator* is acquired **only** by the transcutaneous route. Egg ingestion (→

Ascaris/Trichuris), sandfly bite (→ Leishmania), and cyst-containing pork (→ Taenia) are routes for entirely different organisms.

Necator americanus infects by skin penetration of filariform larvae

Quick Tip: Think barefoot in soil: hookworm filariform larvae bore through skin.

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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:

After splenectomy the body loses its main filter for opsonised, capsule-bearing bacteria, so the patient becomes prone to **overwhelming post-splenectomy infection (OPSI)**. The encapsulated trio to remember is pneumococcus, Hib and meningococcus, but the dominant pathogen is *Streptococcus pneumoniae*, accounting for the large majority of fatal OPSI episodes.

The mechanism: the marginal zone B cells and macrophages of the spleen clear poorly opsonised encapsulated organisms and generate type-specific antibody. Without the spleen, pneumococci proliferate unchecked and can cause fulminant sepsis within hours.

This is precisely why guidelines mandate vaccination against *S. pneumoniae*, *H. influenzae* b and *N. meningitidis* at least two weeks before an elective splenectomy, along with consideration of long-term antibiotic prophylaxis.

The other listed organisms (*Staphylococcus aureus*, *E. coli*, *Pseudomonas*) are not the characteristic OPSI agents and are not selectively dependent on splenic clearance.

Streptococcus pneumoniae – commonest cause of post-splenectomy sepsis

Quick Tip: No spleen = no clearance of encapsulated bugs; the top one is pneumococcus.

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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:

Antibiotic pharmacodynamics splits agents into **concentration-dependent** and **time-dependent** killers. For concentration-dependent drugs, the higher the peak relative to MIC, the faster and more complete the kill; the key indices are C_{max}/MIC and AUC/MIC .

Aminoglycosides are the textbook example. Their bactericidal action improves with rising peak concentration, and they exert a substantial **post-antibiotic effect**, meaning bacterial regrowth is suppressed even after the drug level falls below the MIC. These two properties together justify **extended-interval (once-daily) dosing**: a single large dose gives a high bactericidal peak, while the long drug-free trough reduces accumulation-related nephrotoxicity and ototoxicity.

By contrast, β -lactams, vancomycin and macrolides are time-dependent — killing is maximal once levels exceed roughly $4\times\text{MIC}$, so the goal is to keep the concentration above the MIC for as long as possible ($T > \text{MIC}$) rather than to chase a high peak.

Aminoglycosides = concentration-dependent killing

Quick Tip: Higher peak = more kill, plus a long post-antibiotic effect: that means aminoglycosides.

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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:

Among all the intrinsic laryngeal muscles, the **posterior cricoarytenoid (PCA)** holds a unique position: it is the **only abductor** of the vocal folds. By rotating the arytenoid cartilages laterally, it widens the rima glottidis and opens the airway during inspiration, earning it the nickname the *safety muscle of the larynx*.

Every other adductor-group muscle does the opposite — the lateral cricoarytenoid, the transverse and oblique interarytenoids, and the thyroarytenoid all **close** the glottis. Tension of the cords is governed by the **cricothyroid** (lengthening/tensing, via the external laryngeal nerve), while the vocalis fine-tunes and shortens them.

Clinically this matters enormously: since abduction depends on a single muscle pair, **bilateral recurrent laryngeal nerve injury** abolishes abduction and the cords sit paramedian, threatening the airway with stridor and possible need for tracheostomy.

PCA = sole abductor of the vocal cords

Quick Tip: Only one muscle opens the airway at the larynx - the posterior cricoarytenoid.

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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:

Sudden sensorineural hearing loss is treated as an **otologic emergency**. Its diagnostic threshold — a drop of ≥ 30 dB over ≥ 3 contiguous frequencies within 3 days — should trigger prompt audiometry and immediate therapy, because the window for recovery is narrow.

The evidence-supported mainstay is **corticosteroid therapy**. Treatment usually begins with **systemic (oral) prednisolone**, tapered over a couple of weeks. When the response is incomplete, when oral steroids are contraindicated (for example

in poorly controlled diabetes), or as a salvage measure, **intratympanic steroid injection** delivers high local cochlear drug levels with minimal systemic exposure.

Adjuncts such as hyperbaric oxygen may help in selected early cases. In contrast, empirical antivirals or antibiotics are **not** beneficial in idiopathic disease, and cochlear implantation is a late option for permanent, profound loss — not a first-line response.

SSNHL → steroids: systemic first, intratympanic as salvage

Quick Tip: Sudden SNHL is an emergency - reach for steroids, oral first then intratympanic.

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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:

Halothane is the classic example of an inhalational anaesthetic that depresses the heart directly. Its hallmark cardiovascular signature is a **dose-dependent decline in cardiac output and arterial pressure** — the deeper the anaesthesia, the greater the depression, with mean pressure often falling by roughly **20--30 mm Hg**.

The mechanism is reduced myocardial contractility combined with blunting of the baroreceptor reflex, so the expected compensatory tachycardia does **not** occur; heart rate is typically unchanged or slowed. A second clinically critical property is that halothane **sensitises the myocardium to catecholamines**, so injected adrenaline or rising CO_2 can precipitate ventricular ectopy and arrhythmias.

This profile excludes the other choices: there is no rise in cardiac output, no increase in systemic vascular resistance, and the agent is far from haemodynamically silent — cardiodepression and hypotension are precisely what define it.

Halothane → ↓ cardiac output and BP, proportional to depth

Quick Tip: Halothane is a myocardial depressant - expect cardiac output and BP to fall with depth.

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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:

Korsakoff syndrome is the chronic, irreversible phase of the Wernicke-Korsakoff spectrum, driven by depletion of thiamine (B_1) and damage to the mammillary bodies and dorsomedial thalamic nuclei. The cardinal cognitive defect is a failure of new learning: information presented after the onset cannot be consolidated

into long-term storage.

This forward-facing memory failure is termed anterograde amnesia. Clinically the patient greets the examiner repeatedly, forgets instructions within minutes, and bridges the resulting gaps with confabulation. Immediate (working) memory span and previously learned skills stay largely intact, distinguishing it from a global dementia.

Contrast this with retrograde amnesia (loss of pre-existing memories), transient global amnesia (a brief reversible event), and dissociative amnesia (a psychological, non-organic loss of personal history). None of these match the thiamine-related learning block.

Korsakoff syndrome → anterograde amnesia

Quick Tip: Think of the inability to form NEW memories after a thiamine-deficiency insult.

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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:

Identifying firing distance from a bullet wound depends on which muzzle products reach the skin. The bullet itself always creates an abrasion collar (AC)

and may wipe a grease/dirt collar (GC); these are range-independent. Range is read instead from flame (burns), soot (blackening), and powder grains (tattooing).

In this wound the AC and GC are present, but tattooing, soot, and burning are all absent. Powder grains travel only a limited distance - tattooing extends to about **60 cm** and soot to about **30 cm**. When the muzzle is farther than the powder can travel, only the bullet-related marks remain.

Hence the absence of every accessory powder/flame effect places the muzzle beyond powder range. This is the classic appearance of a far or distant shot.

A contact wound would show a muzzle imprint with internal soot; a close shot would show heavy blackening and singeing; an intermediate shot would show stippling. The clean wound with only AC + GC excludes all of these.

AC + GC, no tattooing/soot/burn ⇒ Distant range

Quick Tip: No gunpowder tattooing, no soot, no burn - the powder never reached the skin.

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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:

Explosion trauma is grouped by causal mechanism into four tiers. Tier one (primary) reflects the pure over-pressure wave damaging air-filled structures: tympanic rupture, pulmonary barotrauma, and hollow-viscus perforation. Tier two (secondary) is penetrating trauma from projectiles and shrapnel. Tier three (tertiary) is blunt trauma from the body being hurled by the blast wind.

The fourth tier (quaternary) is the catch-all for every remaining explosion effect - flame and flash burns, toxic-gas and dust inhalation, crush injury, and worsening of chronic disease.

Facial and chest burns produced by the fireball and radiant heat of the detonation match the quaternary definition precisely, since they arise neither from the pressure wave, nor fragments, nor displacement.

Burns from a blast = Quaternary blast injury

Quick Tip: Burns and inhalation effects are the 'everything-else' category of blast trauma.

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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:

The forensic reading of firing distance rests on the reach of muzzle products. The bullet alone makes an abrasion collar (AC) from skin indentation and a grease collar (GC) from wiped lubricant; both occur at any range and are non-discriminating.

Range markers are the accessory effects: singeing from flame, blackening from soot, and stippling from unburnt powder. Powder tattooing typically reaches no farther than roughly **60 cm**, soot roughly **30 cm**.

In this case all three accessory effects are missing, leaving only AC and GC. The logical conclusion is that the muzzle lay beyond the distance any powder grain could travel.

This is the textbook description of a far/distant shot. Contact, close, and intermediate ranges would each leave at least one of soot, burning, or stippling, which are absent here.

Only AC + GC, no powder/flame marks ⇒ Distant range

Quick Tip: No stippling, no soot, no singed hair - the muzzle was beyond powder range.

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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:

Sparrow-foot or crow-foot marks describe clusters of brush abrasions that branch outward like the print left by a small bird. They form when the epidermis is scraped tangentially by a rough, gritty surface while a dragging force acts on the body.

The mechanism - grazing plus drag against road grit and gravel - points directly to a vehicular collision, where a pedestrian is knocked down and slides or is hauled along the road.

Distinguishing alternatives: a simple ground-level fall leaves single patterned or grazed abrasions without the radiating brush form; gunshot wounds carry abrasion and grease collars around an entry hole; electrical contact leaves a firm, raised Joule burn. None reproduce the branching brush pattern.

Sparrow-foot brush abrasions → Road traffic accident

Quick Tip: Branching grazing abrasions from being dragged over rough road grit.

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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:

Opioid intoxication presents with the unmistakable combination of constricted pupils, slowed or absent breathing, and obtundation. The life-threatening element is respiratory depression, so the antidote must restore ventilatory drive quickly.

Naloxone fulfils this role as a competitive antagonist at the mu-opioid receptor. Delivered intravenously, intramuscularly, or intranasally, it displaces the agonist and reverses both the apnoea and the depressed sensorium within minutes.

Practical caveats: its duration is shorter than that of long-acting opioids such as methadone, so dosing may need repetition or a drip; in opioid-dependent patients it can trigger an acute withdrawal state.

The other agents target different toxidromes - flumazenil for benzodiazepines, atropine for cholinergic/organophosphate poisoning, and N-acetylcysteine for paracetamol hepatotoxicity.

Opioid overdose antidote = Naloxone

Quick Tip: Reverses the opioid receptor - restores breathing in heroin/morphine overdose.